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# Abstracts of the 58th annual meeting of the Swiss Society of Nephrology

Bern (Switzerland), December 3–4, 2026



# 58TH ANNUAL MEETING OF THE SWISS SOCIETY OF NEPHROLOGY (SGN-SSN)

## BERN, DECEMBER 3–4, 2026

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## LONG ORAL PRESENTATIONS

### LO-01

### An integrated single-nucleus and spatial atlas resolves compartment-specific endothelial states and glomerular niche remodeling in human kidney disease

Dr. Anna Rinaldi[1], Prof. Marie Frimat[2], Dr. Viviane Gnemmi[3], Dr. Sarah Bruneau[4], Dr. Muriel Auberson[5], Dr. Sarah Ducastel[6], Dr. Lea Lutz[7], Ms. Cristina Torcasio[7], Dr. Nathalie Merkel[1], Dr. Ivan Berest[8], Dr. Moglie Lequintrec[9], Dr. Simon Ville[4], Prof. Pietro Cippà[10], Prof. Fadi Fakhouri[11]

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Renal endothelial cells (ECs) are essential regulators of filtration, vascular tone, and tissue homeostasis, displaying marked structural and phenotypic heterogeneity across glomerular, arteriolar, and peritubular capillary compartments. Defining endothelial transcriptional states within their anatomical context is therefore critical for understanding the cellular mechanisms underlying human kidney disease. Here, we integrated single-nucleus RNA sequencing (snRNA-seq; 10x Genomics) for unbiased gene-expression profiling with high-resolution in situ spatial transcriptomics (Xenium; custom 473-gene panel) to map microvascular phenotypes in human kidney tissue.

We applied this multimodal strategy to thrombotic microangiopathy (TMA), a severe kidney disorder driven by microvascular endothelial injury. The snRNA-seq atlas comprised samples from 16 patients, while the spatial transcriptomics dataset included 14 additional samples spanning complement-mediated atypical haemolytic uraemic syndrome (aHUS), scleroderma renal crisis (SRC), anti-VEGF therapy, and gemcitabine toxicity.

snRNA-seq resolved the major renal cell lineages and partitioned the EC compartment into distinct glomerular, peritubular capillary, and arteriolar subpopulations. Projection of snRNA-seq annotations onto spatial coordinates showed strong concordance with tissue architecture, as independently assessed by a renal pathologist, with the highest fidelity in structurally demarcated glomeruli and arteries/arterioles. Spatial transcriptomics further validated snRNA-seq identified EC injury programs, confirming their localization to vascular compartments and consistent directional dysregulation across TMA samples. Focusing on the glomerular niche, we investigated local cell–cell communication. Analysis of canonical podocyte-to-glomerular EC signaling through the VEGFA–VEGFR2 axis revealed distinct aetiology-specific changes. Cofactor and ligand-receptor analyses of VEGF signaling further supported these divergent patterns of pathway activity across clinical groups.

Together, this integrated framework resolves renal microvascular heterogeneity and compartment-specific signaling disruption, enabling prioritization of candidate pathways and genes for diagnostic and therapeutic interventions.

### LO-02

### Anti-Nephrin Antibody Positive Minimal Change Disease as a Manifestation of Graft-versus-Host Disease Following Stem Cell Transplantation – A Minimal Change?

Dr. Mateja Kraljevic[1], Dr. Jakob Brune[1], Dr. Anne Stelmes[2], Prof. Jörg Halter[2], Dr. Helmut Hopfer[3], Dr. Anne Mühlig[4], Dr. Felicitas Hengel[4], Prof. Nicola Martin Tomas[4], Prof. Tobias Huber[4], Dr. Caroline Wehmeier[1]

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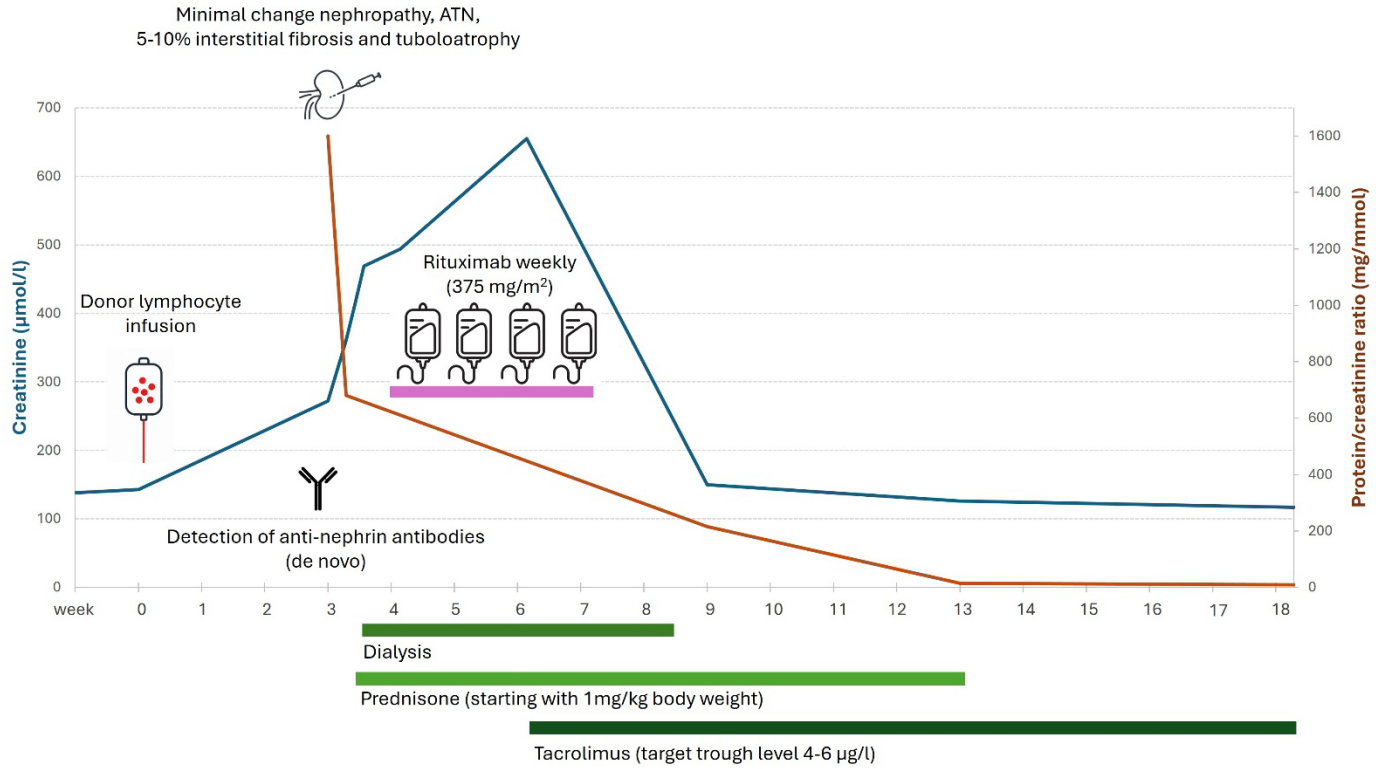
**Background:** Nephrotic syndrome is a rare complication after allogeneic hematopoietic stem cell transplantation (allo-HSCT), suspected to represent a manifestation of graft-versus-host disease (GvHD). While membranous nephropathy accounts for the majority of post-transplant nephrotic syndromes, minimal change disease (MCD) is a less common described histologic diagnosis. In contrast to membranous nephropathy, previous literature suggested a mainly T-cell–mediated process leading to podocyte injury in these cases. However, anti-nephrin antibodies have emerged over the past two years as an important pathogenic mechanism and biomarker in MCD. Whether anti-nephrin antibodies play also a role in MCD as a manifestation of GvHD has so far not been investigated.

**Case Description:** A 72 year old patient with acute myeloid leukemia underwent allo-HSCT from an 11/12 HLA-matched unrelated donor. Five months post-transplant the patient received donor lymphocyte infusion due to persistent measurable disease. Two weeks later, the patient developed nephrotic syndrome with acute anuric kidney failure requiring intermittent hemodialysis. Kidney biopsy revealed MCD suspicious of GvHD despite lacking any other organ involvement. Treatment with high-dose corticosteroids was initiated. Given the severe clinical course and the suspected GvHD, therapy with rituximab (375 mg/m$^2$ weekly for 4 weeks) and subsequently tacrolimus (trough level 5-6 µg/l) was added. After one month of therapy kidney function slowly recovered and the nephrotic syndrome showed complete remission. Interestingly, anti-nephrin antibodies determined at the time of kidney biopsy were positive while these antibodies could not be detected in a serum sample taken one week before allo-HSCT.

**Conclusion:** This case demonstrates that anti-nephrin antibodies can also be detected in minimal change disease occurring after allo-HSCT, which suggests that antibody-mediated podocyte injury may contribute to GvHD-associated MCD. Therefore, B-cell depleting therapy with rituximab is likely the most promising treatment strategy. Mechanisms that trigger anti-nephrin antibody production in the setting of allo-HSCT remain to be elucidated.

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## Figure




## LO-03

### Bicarbonate supplementation reshapes the renal and systemic immune environment in a rat model of chronic kidney disease

Ms. Hannah Auwerx[1], Ms. Rahel Daetwyler[2], Dr. Nima Yassini[2], Dr. Francesco Prisco[3], Dr. Monika Hilbe[3], Dr. Laurine Lang[1], Dr. Zsuzsa Radvanyi[1], Mr. Ashley Fernandes[1], Dr. Soline Bourgeois[1], Prof. Nicole Joller[2], Prof. Carsten Wagner[1]

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**Background.** Metabolic acidosis (MA) is a hallmark complication of late-stage chronic kidney disease (CKD). Sodium bicarbonate is used to correct MA in advanced CKD, however, the underlying mechanisms remain incompletely understood. The immune-modifying properties of local pH are well established in oncology and inflammatory diseases, yet the immune changes underlying renal MA and its correction with bicarbonate have not been addressed. We hypothesize that CKD-related MA and its correction with bicarbonate remodels local and systemic immunity.

**Methods.** We developed high-dimensional flow cytometry panels for rats and used them to perform the first characterization of the renal and systemic immunity in Cy/+ rats, a model of CKD with spontaneous MA. Cy/+ rats received bicarbonate supplementation or vehicle and renal, splenic and blood immune populations were analyzed. Healthy rats received pure alkali or acid loads to assess the effect of acid-base status in the absence of CKD. Finally, human PBMCs were cultured in under different pH conditions to evaluate the translational relevance.

**Results.** Bicarbonate supplementation triggered a kidney-specific adaptive immune response and resulted in local T-cell activation and proliferation in Cy/+ rats. On a systemic level, bicarbonate dampened CKD-driven granulocytosis. These effects were largely disease-dependent, and pure acid-base challenges in healthy rats were insufficient to induce similar immune alterations. Accordingly, alkalinization modified activation and effector functions in human peripheral blood mononuclear cells (PBMCs), only after prior T-cell activation.

**Conclusion.** This work provides a resource for immune profiling in rat models and bridges a gap in rat immunology. Moreover, we offer the first immune characterization of the widely used Cy/+ rat model and uncovered a previously underappreciated role of CKD-related MA and its treatment in shaping the local and systemic immunity.

## LO-04

### Biopsy-proven renal fibrosis is associated with left ventricular mass on cardio-renal MRI in chronic kidney disease

Dr. Aurélie Huber[1]

*1 Geneva University Hospitals*

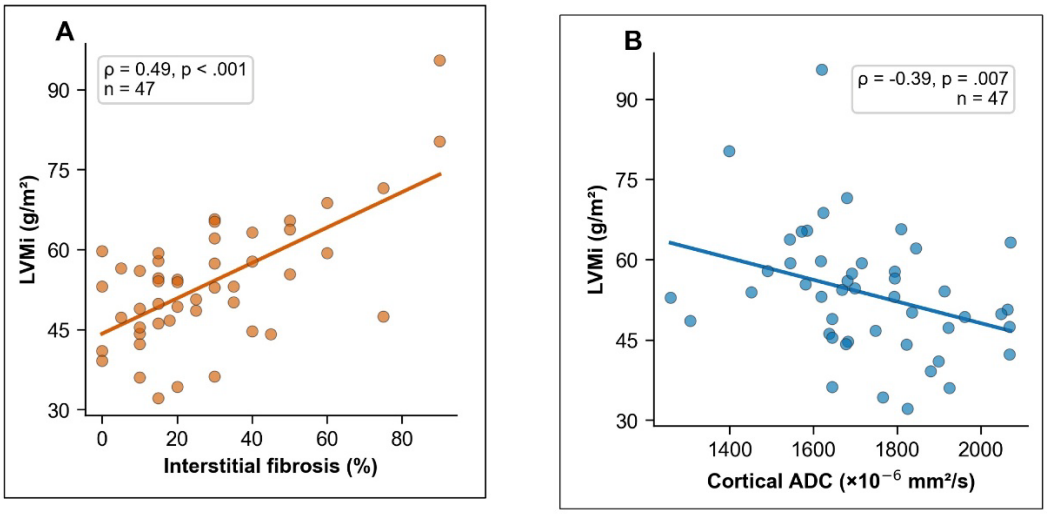
**Background**: Left ventricular remodelling is prevalent in chronic kidney disease (CKD) and independently predicts cardiovascular mortality. Prior studies have linked renal functional markers to cardiac structural changes, but the direct relationship between histological renal fibrosis and cardiac MRI parameters remains uncharacterized. We aim to investigate the association between biopsy-proven renal fibrosis and cardiac MRI parameters in CKD patients, and to assess whether renal apparent diffusion coefficients (ADC) reflect these associations.


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**Methods**: This prospective single-centre cohort included CKD patients with native kidneys who underwent same-week cardiorenal MRI and renal biopsy. Renal fibrosis was quantified as a percentage of the affected cortex area. Five cardiac parameters were selected a priori: LVMi, LVEDVi, GLS, GCS, and native myocardial T1. Associations with renal fibrosis were assessed using Spearman correlations, and multivariable linear regression adjusted for age and estimated glomerular filtration rate (eGFR). The association of these five cardiac parameters with renal ADC (cortical, medullary, and corticomedullary difference) was examined.

**Results**: Forty-seven patients were included (median age, 49 years; 55% male). LVMi correlated with renal fibrosis (ρ = 0.49; p <0.001). GLS (ρ = 0.33; p = 0.03) and GCS (ρ = 0.30; p = 0.04) showed consistent trends that did not survive statistical correction. Renal fibrosis remained independently associated with LVMi after adjustment for age and eGFR (β = 0.33; p <0.001). Adding LVEDVi attenuated the fibrosis coefficient by 22% (β = 0.26; p <0.001). Renal cortical ADC correlated with LVMi (ρ = −0.39; p = 0.007), GLS (ρ = −0.36; p = 0.01), and myocardial T1 (ρ = −0.36; p = 0.01), similar to the fibrosis association.

**Conclusion**: In CKD patients, biopsy-proven renal fibrosis is independently associated with LV mass through both volume-dependent and volume-independent pathways. Renal cortical ADC reflects these cardiorenal associations, supporting its role as a non-invasive fibrosis surrogate.




**Legend**: Association of left ventricular mass index (LVMi, g/m$^2$) with renal interstitial fibrosis rate (%) on the left panel (A), and with cortical renal ADC (x10$^{-6}$ mm$^2$/s) on the right panel (B) using Spearman correlation. Each point represents one patient.
ADC = apparent diffusion coefficient, LVMi = left ventricular mass index.

## LO-05

### Clinical and histological responses to iptacopan in IgA-mediated glomerular disease: a real-world cohort with paired kidney biopsies

Dr. Stephanie Damm[1], Dr. Sucheta Sridhar[2], Prof. Britta George[1], Dr. Carolin Eul[2], Dr. Thomas Welte[3], Dr. Birgit Maria Helmchen[4], Dr. Ariana Gaspert[1]

*1. Universitätsspital Zürich, 2. Universität Zürich, 3. Division of Nephrology, University Hospital Zürich, Zürich, Switzerland, 4. Division of Pathology, University Hospital Zürich, Zürich, Switzerland*

**Background** Iptacopan inhibits alternative complement pathway activation, but evidence linking kidney function trajectories with histological responses in IgA-mediated glomerular disease remains limited.

**Methods:** We evaluated 6 patients with IgA nephropathy, IgA vasculitis nephritis or IgA-dominant glomerulonephritis receiving iptacopan (200 mg twice daily). At initiation, all received maximally tolerated renin–angiotensin–aldosterone system blockade and an SGLT2 inhibitor. Clinical outcomes were assessed over 12 months. Individual eGFR slopes were calculated by linear regression before and after initiation. Paired biopsies underwent Oxford MEST-C and semiquantitative immunofluorescence assessment. Pretreatment biopsies preceded therapy by a median of 11 months; repeat biopsies followed a median of 9 months after initiation. Five-year risks of 50% eGFR decline or kidney failure were explored using the race-inclusive International IgAN Prediction Tool at both biopsies.

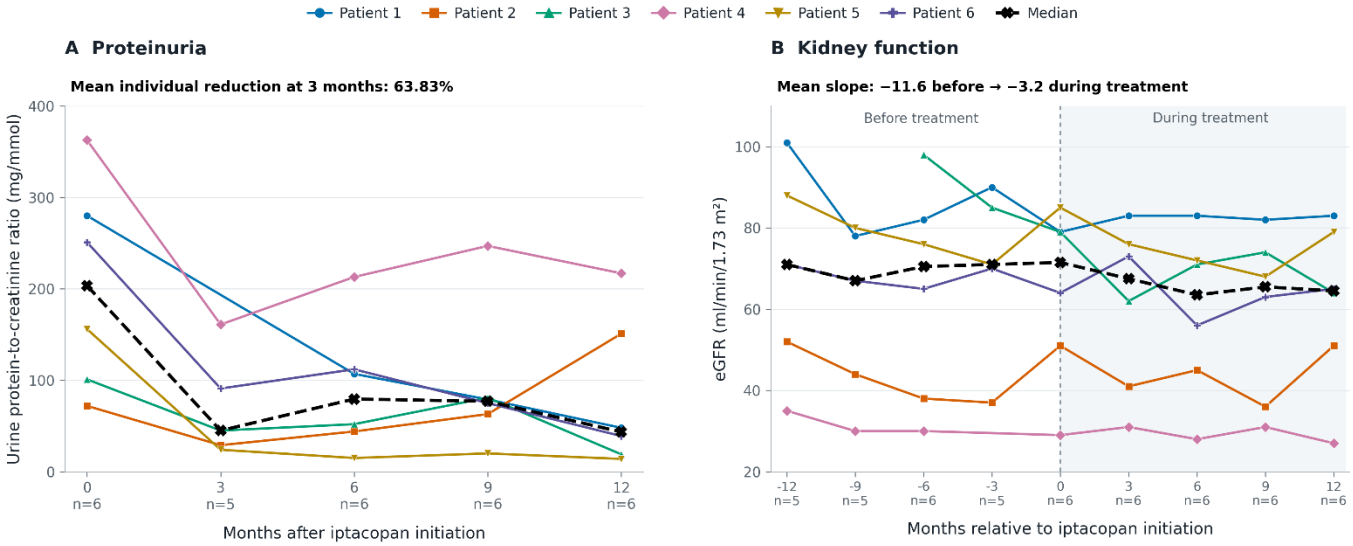
**Results:** Mean annual eGFR slope improved from −11.6 to −3.2 ml/min/1.73 m²/year. Mean eGFR was 64.5 at baseline and 61.5 ml/min/1.73 m² at 12 months. Mean urine protein-to-creatinine ratio decreased from 203.8 to 81.3 mg/mmol (60% reduction). Glomerular microhematuria declined from 5/6 patients at baseline to 3/6 at 3 months and 0/6 at 6 and 9 months; 1/6 had microhematuria at 12 months. Endocapillary hypercellularity resolved in all 3 initially affected patients, and crescents resolved in 5/6. Tubular atrophy/interstitial fibrosis scores remained unchanged. Glomerular C3 intensity decreased in all patients, becoming undetectable in 2/6, whereas IgA deposits persisted in all. Exploratory recalculation of the International IgAN Prediction Tool using repeat-biopsy data yielded lower estimated 5-year risks in all 6 patients. Median predicted risk decreased from 7.65% to 4.15%, with a median within-patient relative reduction of 43.9%.

**Conclusions:** In this small observational cohort, iptacopan was associated with attenuation of eGFR decline, reduced proteinuria, resolution of glomerular microhematuria at 6–9 months, and regression of active histological lesions and C3 deposits despite persistent IgA deposition.


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## Clinical course during iptacopan treatment

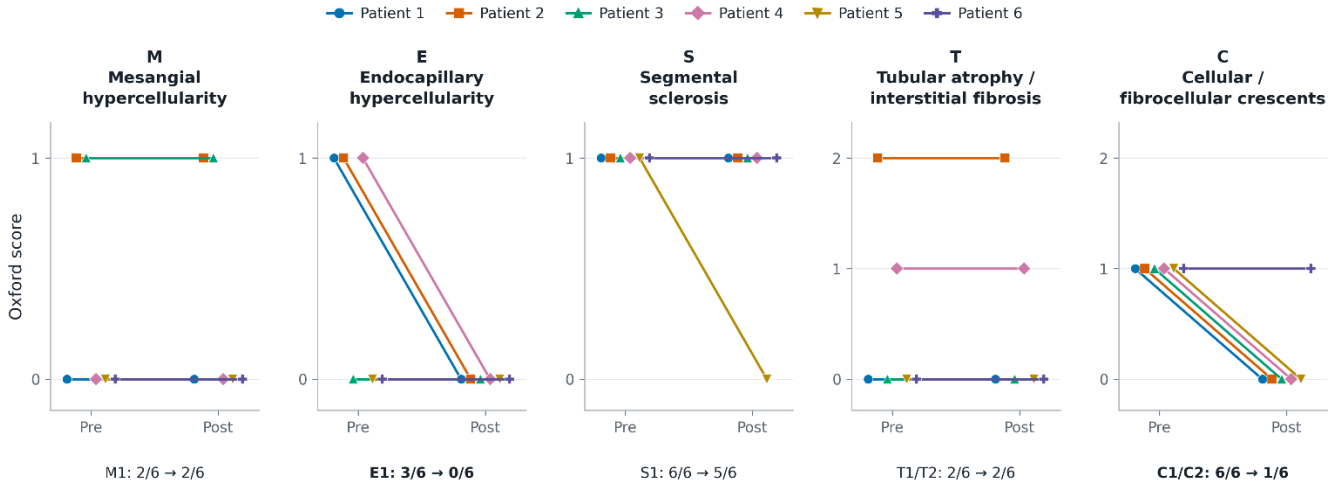
Six patients with paired kidney biopsies and 12-month clinical follow-up




Lines connect observed measurements across missing visits; no values are imputed. Black dashed lines show medians. n = available measurements.
Slopes are mean individual linear-regression slopes (ml/min/1.73 m²/year); month 0 is included in both periods.

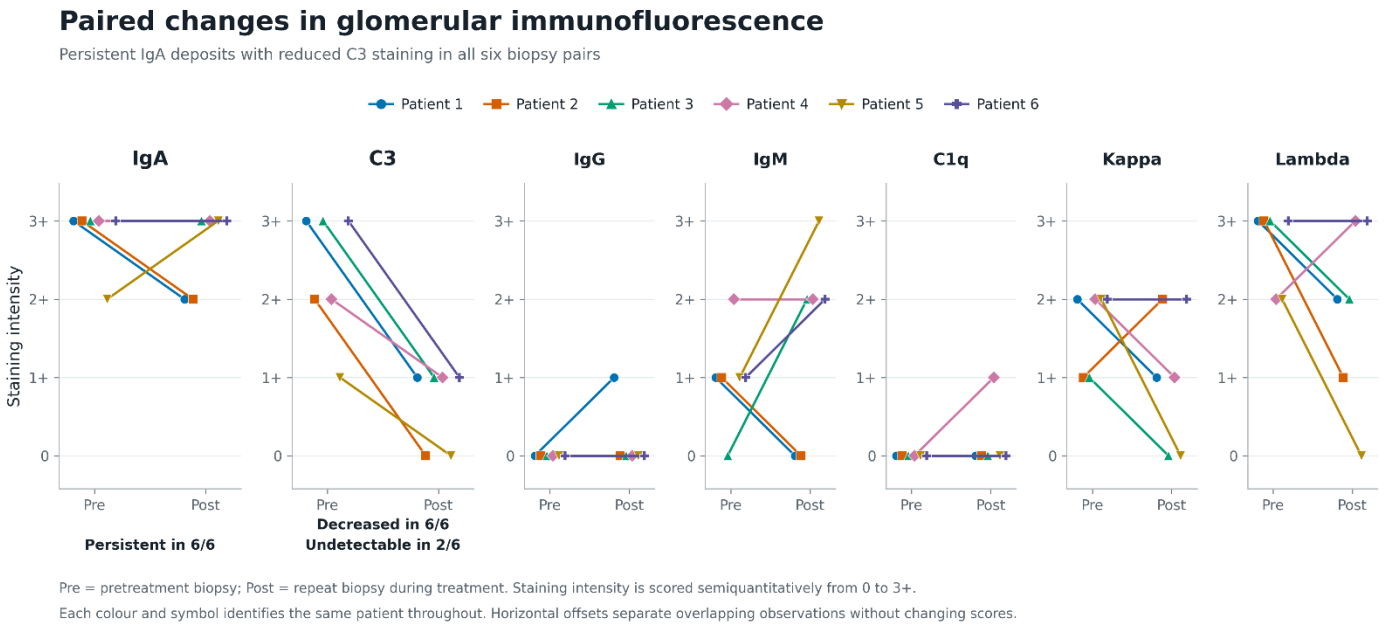
## Paired changes in Oxford MEST-C scores

Six biopsy pairs before and during iptacopan treatment




Pre = pretreatment biopsy; Post = repeat biopsy during treatment. Each colour and symbol identifies the same patient throughout.
Horizontal offsets separate overlapping observations; score values are unchanged. Absence of a lesion on repeat biopsy may reflect sampling.


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## LO-06

### Discovery of anti-Formin-like Protein 1 autoantibodies in lupus nephritis patients and their clinical significance

Dr. Filippo Donati[1], Prof. Marco Prunotto[1], Prof. Leonardo Scapozza[1], Prof. Maurizio Bruschi[2], Dr. Gian Marco Ghiggeri[3]

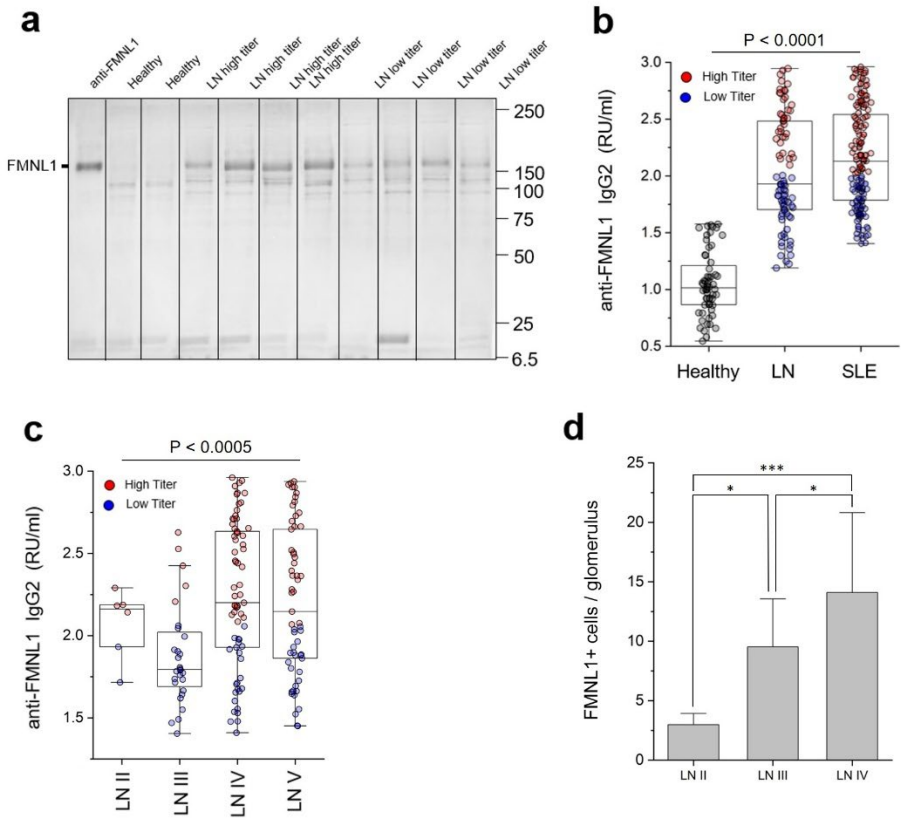
*1. University of Geneva, 2. Università di Genova, 3. IRCCS Istituto Giannina Gaslini*

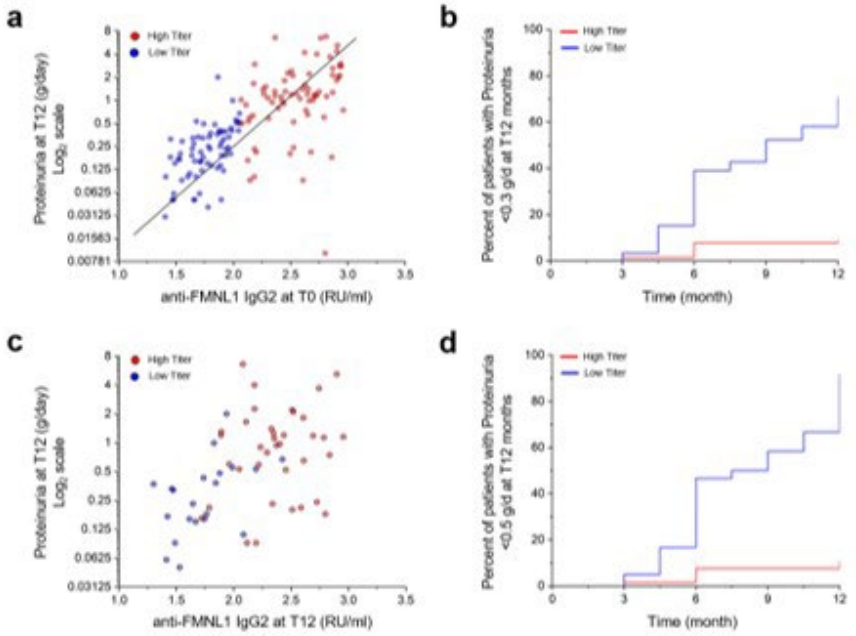
Systemic lupus erythematosus (SLE) and lupus nephritis (LN) are prototypical disorders characterized by the presence of pathogenetic autoantibodies (AutoAbs) directed against several cell components in the kidney. The identification and characterization of AutoAbs in SLE and LN and their role in autoimmunity remain an active field of research.

Screening the interaction of SLE and LN sera with peptides of a customized array identified 133 reactive proteins, with Formin-like Protein 1 (FMNL1) showing the highest probability score. FMNL1 is highly expressed in macrophages derived from circulating monocytes. In renal parenchyma, FMNL1 co-localizes with the macrophage marker CD68. In FMNL1-/- knockout mice, loss of FMNL1 in two experimental models of renal damage was associated with increased urinary protein excretion and worsening renal function. Circulating anti-FMNL1 IgG2 levels, measured by a specific ELISA, were high in a large proportion of LN and SLE patients of the Zeus cohort, correlated linearly with proteinuria at 12 months, and represented a unique and very strong predictor of proteinuria at 1 year. Glomerular FMNL1 expression, circulating anti-FMNL1 IgG2 levels, and macrophage infiltration positively correlated with proliferative states (class III-IV glomerulonephritis). In a second cohort of LN patients randomized to receive Belimumab (CALIBRATE), high circulating anti-FMNL1 IgG2 were again associated with poor outcomes and a poor response to the drug.

The peptide array approach, coupled with a novel workflow for data analysis, allowed the identification of FMNL1 as a novel target antigen of AutoAbs in SLE and LN. AutoAbs directed against FMNL1 might contribute to disease progression by impairing macrophage-mediated tissue repair, and assessment of their circulating levels might serve as a novel biomarker of renal outcome and response to specific therapies in LN patients. Further work is needed to clarify whether depletion of these circulating AutoAbs could also represent a therapeutic option in SLE and LN patients.

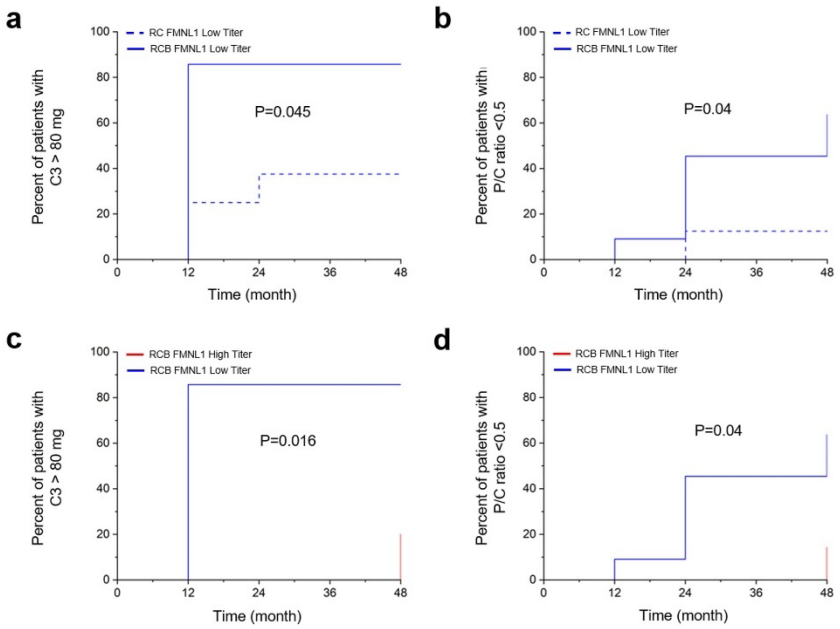

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## LO-07

### ERMP1 safeguards metabolic integrity and limits acute ischemic injury in the kidney

Mr. Basel Yousef[1], Dr. Thibaut Charlemagne[1], Mr. Raphaël Yvon[1], Dr. Thomas Verissimo[1], Prof. Sophie de Seigneux[2], Prof. Eric Feraille[1], Dr. Monika Gjorgjieva[1]

*1. University of Geneva, Faculty of Medicine, PHYM department, 2. Hôpitaux Universitaires Genève*

**Introduction:** Renal tubular epithelial cells have exceptionally high energy demands required to sustain electrolyte transport and tubular reabsorption, relying predominantly on mitochondrial oxidative phosphorylation. This high oxidative dependence renders the kidney particularly vulnerable to ischemic injury and metabolic stress. We previously identified ER metallopeptidase 1 (ERMP1) as a regulator of hepatic metabolic homeostasis and showed that its expression increases in renal pathology. However, its role in renal metabolism and ischemic injury remains unknown.

**Methods:** Renal tubular cell-specific *Ermp1* knockout (K.Ermp1KO) mice were generated using Pax8-Cre recombination of a "floxed" *Ermp1* allele. Bulk RNA sequencing and proteomic analysis were performed in kidneys from young K.Ermp1KO mice. Renal function, including glomerular filtration rate (GFR) and urine output was assessed in young and aged animals. Proteomic analysis was further performed on kidneys from aged mice. Finally, unilateral renal ischemia–reperfusion injury (IRI) was induced, and kidneys were analysed at 3 and 7 days post-injury.

**Results:** K.Ermp1KO mice developed persistent renal hypotrophy from an early age. Young mice displayed increased lipid trafficking and turnover and altered tubular transport pathways associated with enhanced mitochondrial respiration and glycolysis, despite preserved renal structure and function. With age, K.Ermp1KO mice developed a progressive decline in GFR, and impaired urine concentrating capacity. Proteomic analysis in older K.Ermp1KO mice confirmed metabolic remodelling characterized by enhanced mitochondrial oxidation, oxidative stress signatures, depletion of renal lipid pools, and marked alterations in tubular transport proteins, including increased $Na^+/K^+$-ATPase abundance. Following renal ischemia, K.Ermp1KO mice exhibited impaired recovery marked by sustained tubular injury, apoptosis, and inflammation.

**Conclusion:** Our study identifies ERMP1 as a key player in renal metabolic flexibility, linking oxidative metabolism, lipid remodeling, and ion transport to tubular resilience during aging and ischemic injury.

## LO-08

### Hemodialysis Anticoagulation with Apixaban Following Heparin-Induced Thrombocytopenia: A Case Report

Ms. Noemi Galliker[1], Dr. Robert Escher[1], Dr. Ivo Bergmann[1]

*1. Spital Emmental*

**Background:** Anticoagulation during hemodialysis poses a particular challenge in patients with a history of antibody-positive heparin-induced thrombocytopenia (HIT). Although argatroban is an established alternative to heparin, its use for dialysis anticoagulation may be limited by reimbursement and practical considerations. Regional citrate anticoagulation is another option but is not routinely used in all centers. Direct oral anticoagulants (DOACs), such as apixaban, may represent an alternative, but evidence regarding their use for hemodialysis anticoagulation, particularly in patients with HIT, remains limited.

**Case presentation:** During initial inpatient treatment, the patient received argatroban. Phenprocoumon was subsequently initiated for outpatient anticoagulation but discontinued because of acute liver injury. Once systemic anticoagulation was no longer required, argatroban was considered for dialysis-related anticoagulation; however, insurance coverage was denied. As citrate anticoagulation was not routinely used at our center, apixaban 5 mg was administered 2 hours before hemodialysis. Several episodes of filter occlusion occurred under this regimen. Serial anti-factor Xa activity measurements showed values below the fifth percentile of those observed in a reference population with preserved renal function. The apixaban dose was therefore increased to 7.5 mg. Thereafter, anti-factor Xa activity was within the intended range, and no further filter occlusions occurred. No bleeding complications have been observed to date.


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**Conclusion:** This case suggests that apixaban may provide effective anticoagulation during hemodialysis in selected patients with HIT when conventional strategies are not feasible. Anti-factor Xa activity-guided dose adjustment may be required. Further data are needed to establish the safety and efficacy of this approach, particularly with higher-than-standard DOAC doses.

## LO-09

### Impact Of Blood Transfusions After Renal Transplantation On Clinical Outcomes And Broadness Of Sensitization

Ms. Stella Gerber[1], Prof. Stefan Schaub[2], Dr. Matthias Diebold[2], Dr. Gideon Hoenger[3], Dr. Caroline Wehmeier[2]

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**Background:** Whether erythrocyte transfusion (ET) after kidney transplantation triggers the formation of de novo donor-specific HLA-antibodies (HLA-DSA) and rejection remains debated. If this effect of ET was true, we would expect a general broadening of HLA sensitization, reflected by an increase in calculated panel reactive antibodies (cPRA), and the formation of de novo HLA-antibody specificities beyond HLA-DSA.

**Methods:** This retrospective study in 519 kidney transplant recipients investigated the impact of ET within the first-year post-transplant on the occurrence of allograft rejection, the incidence of de novo HLA-DSA, the evolution of cPRA, and emerging de novo HLA-antibody specificities beyond HLA-DSA. ET was assessed both as a binary variable (with/without ET) and in quantity-based groups (without ET, 1-2 ET, 3-5 ET, >5 ET).

**Results:** There was no difference between patients with/without ET in the one-year incidence of clinical rejection (19.7 vs. 15.3%, p = 0.43), clinical/subclinical rejection (30.0% vs. 28.6%, p = 0.86), TCMR (15.6% vs 19.4%; p = 0.30) and AMR (15.6% vs 10.1%; p = 0.19). Quantity-based analysis also showed no significant difference, except for patients with >5 ET having a higher incidence of AMR (p = 0.03). This association was no longer significant after multivariable adjustment in a time-variant Cox regression model (HR 1.10 (95%-CI 0.52-1.59); p = 0.74). De novo HLA-DSA incidence did not differ between patients with/without ET (3.5% vs 3.1%, p = 0.80), nor across quantity-based groups (p = 0.17). Likewise, cPRA change did not differ between patients with/without ET or across quantity-based groups. No preferential sensitization against highly prevalent HLA-antigens class I was observed (e.g. HLA-A1 [0.4%], HLA-A2 [0.8%], HLA-B51 [0.8%], HLA-Cw7 [1.3%]).

**Conclusions:** ET after kidney transplantation under tacrolimus/mycophenolate/prednisone-based immuno-suppression was not associated with allograft rejection, de novo HLA-DSA formation, and broadening of HLA sensitization in the first-year posttransplant. These findings argue against the clinical need for HLA-matched/selected ET.

## LO-10

### Lessons learnt from a patient whose dialysis treatment could be stopped due to complement therapy

Dr. Jan Brügger[1], Dr. Bernhard Hess[2], Prof. Andrea Vicelli[3], Dr. Ariana Gaspert[4], Dr. Birgit Helmchen[4], Dr. Harald Seeger[5], Prof. Thomas Müller[6]

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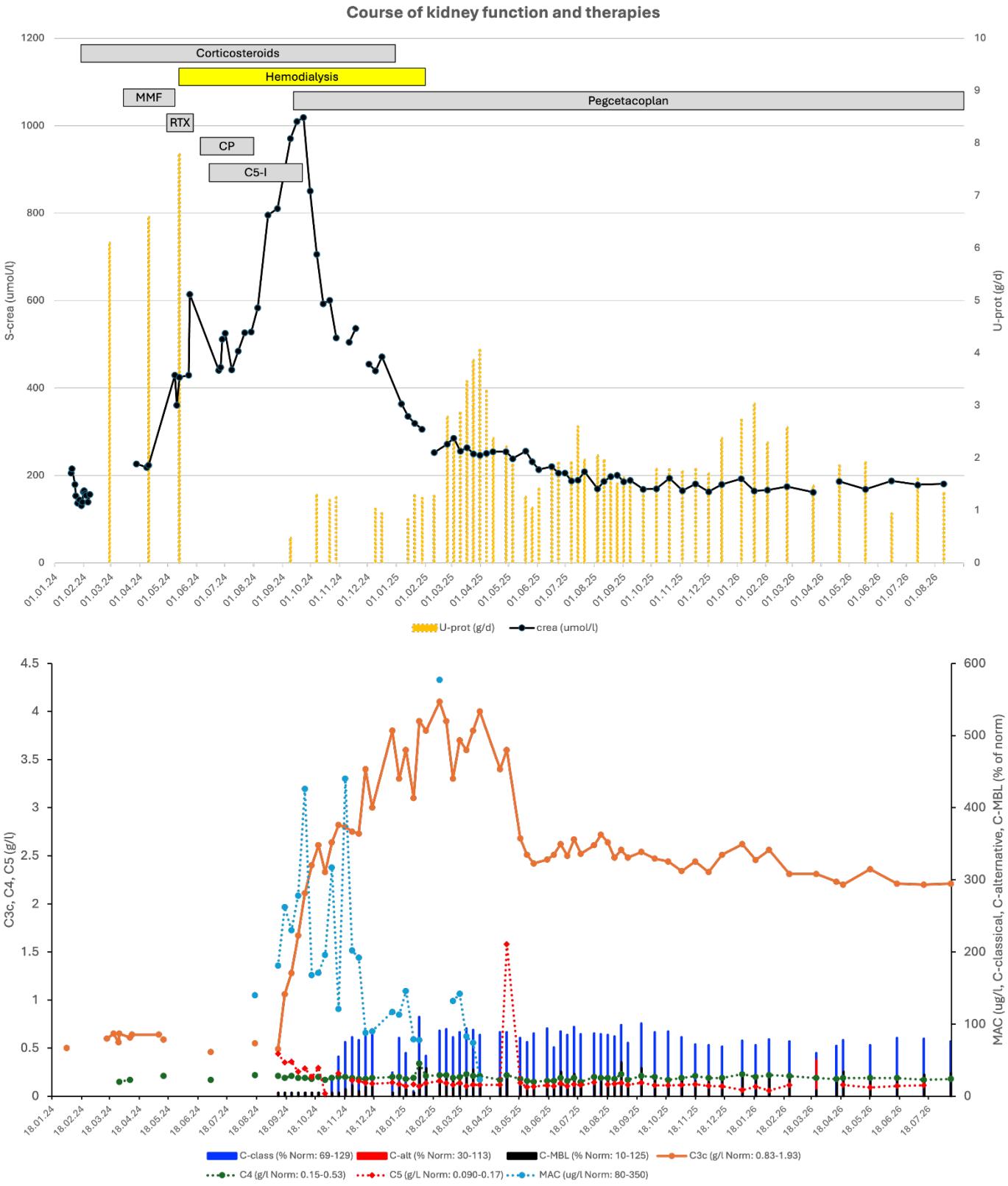
**Background:** There is still a paucity of data on indication and treatment with C3 complement inhibitor therapies in membrane-proliferative glomerulonephritis (IC-MPGN & C3G), particularly regarding long-term courses of complement dynamics. At the last SGN conference we presented the early results of a patient with C3G who had been successfully treated with pegcetacoplan (PEG) and could be weaned off chronic dialysis treatment. Treatment and course of this case offers unique insights.

**Methods:** Clinical, functional and complement markers (circulating C3, C4, C5, MBL, classical and alternative pathway as well as membrane attack complex (MAC) were prospectively monitored in 2 weeks intervals.

**Results:** The period of observation is 32 months. PEG was started in Sep 2024 and is currently given twice weekly. Clinically the patient remains fatigued but otherwise leads a normal life and experienced no significant side-effects of the treatment. Creatinine levels showed an exponential improvement under PEG treatment, mirroring the course of increase before PEG. Proteinuria more than halved from maximum levels of 5-8 g/d to now 1-2 g/d. About 6 months after initiating PEG overall kidney function has stabilised (Fig. 1). The complement levels showed low S-C3 levels before (around 0.6 g/l) and increased values after initiation of C3-inhibition (around 3.5-4 g/l). In the long-run they stabilised but remained elevated (around 2.5 g/l). The pre-PEG levels of the MAC decreased due to the treatment. C4, C5, pathways of classical and lectin activation remained largely unaffected by PEG (Fig. 2).

**Conclusion:** A trial with C3-inhibition therapy in C3G/IC-MPGN seems to be justified even if kidney failure and dialysis treatment have been ongoing for several months. This approach is supported by the presented case, the treatment response in GFR and proteinuria, the typical patterns of complement levels and overall the major impact on the patient's life quality.

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## LO-11

### Mapping tubular injury and repair states across single-cell transcriptomics and imaging mass cytometry

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**Background:** Kidney repair is a complex biological process involving epithelial, immune, and stromal cells whose interactions shape recovery or scarring after injury. Single-cell transcriptomics has substantially advanced the characterization of tubule cell states, yet tissue dissociation eliminates spatial relationships, cell-cell interactions, and protein-level functional readouts. To overcome these limitations, we established imaging mass cytometry (IMC) as an antibody-based platform for spatial protein profiling. We integrated IMC and single cell RNAseq to study kidney repair in a mouse model of ischemia-reperfusion injury.

**Methods:** We analyzed mouse scRNA-seq datasets covering acute injury and recovery. In parallel, we applied our kidney IMC panel (24 markers of tubular structure, identity, injury, vasculature, immune cells) to mouse kidneys at baseline and days 2, 13, and 28 post-AKI. IMC images were segmented into single cells, enabling quantification of protein-defined populations and their spatial neighborhoods. Cross-modal integration anchored IMC-defined protein states to transcriptomic clusters and supported the study of tubular injury trajectories and associated microenvironments.

**Results:** We identified distinct tubular cell states, including LRP2-high mature proximal tubule cells and SOX9-positive injury-associated tubule cells lacking canonical segment markers. Spatial analysis revealed microenvironmental niches in which SOX9-positive tubules were juxtaposed to CD45-positive immune infiltrates and ACTA2-positive stroma cells, highlighting coordinated epithelial–immune–stromal interactions during injury and repair. scRNA-seq provided complementary transcriptomic states and marker candidates, enabling cross-modal annotation and demonstrating how unbiased scRNA-seq discovery can identify candidate targets and guide future targeted spatial protein panels.

**Conclusions:** scRNA-seq and IMC capture complementary dimensions of kidney injury and repair. This multimodel framework provides a platform to study the biological processes underlying tubule injury and repair and supports the discovery of biomarkers and potentially targetable pathways.

## LO-12

### Mechanical prosthetic valve endocarditis caused by Mycoplasma pneumoniae in a immunocompromised patient with systemic lupus erythematosus

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**Background:** Culture-negative endocarditis accounts for approximately 7% of infective endocarditis cases and poses a major diagnostic challenge, particularly when caused by fastidious organisms such as *Mycoplasma pneumoniae* (MP). MP endocarditis is extremely rare, with only very few cases reported. Diagnosis is complicated by unreliable blood cultures and serology, especially in immunocompromised patients. In patients with systemic lupus erythematosus (SLE), non-infectious Libman-Sacks endocarditis represents an important differential diagnosis requiring different management.

**Case presentation:** We report a case of a 23-year-old woman with a history of *Staphylococcus aureus* prosthetic mitral valve endocarditis and SLE, maintained on rituximab, who presented with embolic stroke despite therapeutic anticoagulation and new vegetations on her mechanical mitral valve. With repeated negative blood cultures, and clinically quiescent SLE, the differential diagnosis remained uncertain between infective and Libman-Sacks endocarditis. Absent lupus serological activity and antiphospholipid markers argued against an autoimmune aetiology. Following valve replacement, histopathology showed non-specific inflammatory changes, and conventional valve cultures were negative. Eubacterial 16S rRNA PCR performed on two independent valve tissue samples identified MP, while PCR of peripheral blood remained negative. Targeted therapy with doxycycline led to full clinical and echocardiographic recovery.

**Discussion:** This is one of only six reported MP endocarditis cases, the first affecting a mechanical prosthetic valve, and the second on a patient treated with rituximab. It highlights the limitations of conventional microbiological and serological diagnostics in B-cell-depleted patients and underscores the diagnostic value of tissue-based molecular techniques, particularly eubacterial 16S rRNA PCR, when standard methods fail.

**Conclusion:** MP endocarditis should be considered in culture-negative endocarditis, especially in immunosuppressed or B-cell-depleted patients, where serology may be falsely reassuring. Tissue-based molecular diagnostics are essential for accurate identification and can critically guide management, distinguishing infective from autoimmune valve disease.

## LO-13

### Molecular Consequences of Conversion from Calcineurin Inhibitor to Belatacept in Kidney Allografts

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**Background:** Chronic calcineurin inhibitor (CNI) nephrotoxicity is recognized by late histologic scarring, whereas its active, potentially reversible biology remains largely hidden. Belatacept conversion removes CNI exposure within the same kidney. As a dual intervention, changes may be belatacept-attributable or associated with CNI withdrawal.

**Methods:** We compared 10 belatacept-converted kidney transplant recipients with 10 matched CNI-treated controls, each contributing paired biopsies, yielding 40 biopsies with histology, Molecular Microscope Diagnostic System (MMDx) assessment, and raw bulk transcriptomes normalized in one batch. Conversion biopsies were obtained ≤12 months before conversion (on CNI) and ≥6 months after conversion (off CNI); controls had two biopsies over a comparable interval and were matched 1:1 using 11 covariates, including MMDx scores at base-line. The primary analysis compared longitudinal change between cohorts (difference-in-differences), complemented by Hallmark gene set enrichment analysis (GSEA), paired pre/post testing, eGFR slopes, and sensitivity analyses.

**Results:** Banff lesions remained unchanged, whereas MMDx rejection (Δ -0.14 vs. +0.05; $p = 0.045$), TCMR ($p = 0.024$), AMR ($p = 0.045$), and global disturbance scores (ANCOVA $p = 0.048$) declined after conversion. Genome-wide, 39 probesets differed at nominal $p<0.001$ (34 lower, 5 higher). The top-

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ranked transcript, *PDK1*, fell after conversion while rising under continued CNI, whereas *SPP1* (osteopontin) rose consistently (3 of 5 increased probesets); the paired analysis showed de-repression of calcineurin-NFAT targets (*RCAN1*, *TRPM6*, *KL*). 25 of 50 Hallmark pathways changed at FDR <0.05: interferon-gamma, interferon-alpha, allograft-rejection, and inflammatory programs decreased, whereas oxidative phosphorylation and fatty-acid metabolism increased. eGFR slope was +4.8 ml/min/1.73m$^2$/year after conversion versus -8.8 in controls (p = 0.045).

**Conclusions:** Declining rejection- and inflammation-related programs are most plausibly belatacept-attributable, since withdrawal of immunosuppression alone should increase allo-immune activity, whereas de-repression of calcineurin-NFAT targets and recovery of tubular metabolic programs indicate an active, partially reversible, CNI-associated injury component invisible to histology. Matched controls worsened under continued CNI. These exploratory attributions require prospective validation.

## LO-14

### Patient-reported experiences of hand massage for CKD-associated pruritus during hemodialysis.

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**Background:** Chronic kidney disease-associated pruritus (CKD-aP) is linked to depression, sleep disturbances and reduced quality of life. In our recent study in French-speaking part of Switzerland, one in four patients receiving hemodialysis reported CKD-aP. Hand massage is a personcentered, non-pharmacological complementary intervention that nurses can integrate into routine care. Understanding how patients experience this intervention may reveal aspects of care not captured by standardized outcome measures.

**Objective:** To explore how people receiving hemodialysis experience nurse-delivered hand massage for CKD-aP, including its perceived meaning and benefits beyond symptom relief.

**Methods:** This qualitative study is part of an ongoing, sequential, multicenter mixed-methods study. Phase 1 is a randomized controlled trial with a targeted sample of 54 adult participants with CKD-aP, comparing hand massage to an attention-control conversation without physical touch. Trial outcomes include itch severity, quality of life and sleep. Phase 2 involves semi-structured interviews with an expected sample of 12 participants from the massage group. Interviews are recorded, transcribed and thematically analyzed.

**Results:** To date, 19 participants are enrolled in the trial and six have completed the qualitative interviews (Table 1). Preliminary analysis of the interviews identified four effects of hand massages: (1) A moment of well-being, time for oneself and respite; (2) an opportunity for connection in the context of loneliness and social isolation; (3) a perceived relief from hand stiffness and spasticity; and (4) Experiencing one's own body as a resource rather than solely as a site of dysfunction.

**Conclusions:** Preliminary data suggest that participants value hand massage beyond itch relief, encompassing physical comfort, emotional well-being and human connection during hemodialysis. These results offer insight into dimensions of nurse-delivered care that may be insufficiently captured by standardized measures. Further interviews will allow to deepen and refine these preliminary themes. Effects on CKD-aP remain to be established by the trial.

**Table 1**

| | | Patients (n=22) (drop out n=3) |
|---|---|---|
| **Characteristics** | | |
| Age in years, mean (SD) [min-max] | | 70.09 (11.55) [44-88] |
| Sex, % | Men | 59% |
| | Women | 41% |
| Marital Status, % | Single | 9% |
| | Married | 46% |
| | In couple | 9% |
| | Divorced | 32% |
| | Widowed | 4% |
| Number of years on dialysis, mean (SD) [min-max] | | 3.40 (4.35) [0.28-19] |
| Main comorbidities, % | Hypertension | 77% |
| | Diabetes | 50% |
| | Congestive heart failure | 45% |
| | Peripheral vascular disease | 41% |
| | Coronary artery disease | 36% |
| Baseline itch NRS, mean (SD) [min-max] | | 4.90 (2.17) [1-8] |
| Baseline itch WI-NRS, mean (SD) [min-max] | | 5.86 (2.59) [1-10] |

## LO-15

### Postpartum recovery of renal parameters after preeclampsia in women without preexisting maternal conditions: a literature review

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**Background:** Preeclampsia is a multisystem maternal disorder characterised by new-onset hypertension after 20 weeks of gestation, accompanied by proteinuria +/- other signs of organ dysfunction. Although these abnormalities generally resolve after delivery, the exact timeframe required for complete recovery remains unclear. In some women, cardiovascular and renal impairment persist for several months. We undertook a systematic review of available literature to establish the time to full recovery in previously healthy women who developed renal impairment during pregnancies complicated by preeclampsia.

**Methods:** We searched Pubmed-MEDLINE and Google Scholar for eligible studies, between 2000 and 2026. Retrospective and prospective studies involving previously healthy women with preeclampsia which reported kidney outcomes were included. Articles published in English, French, Italian and German were considered. Of all reports screened, 7 met our inclusion criteria. A descriptive synthesis was performed. Complete renal parameter data were available for 547 preeclamptic patients.

**Results:** Renal parameters and diagnostic thresholds varied widely across studies. Proteinuria persisted in 0-58% of women at 3 months postpartum. Reduced eGFR (or elevated serum creatinine) was less frequent and was reported in 2-17% of patients at 3 months. Only one study extended follow-up beyond 3 months, showing persistent hypertension in 18% and proteinuria in 2% of women at 2 years. These wide ranges largely reflect the marked heterogeneity of the included studies.

**Conclusion:** While no established timeline exists for renal recovery after preeclampsia and most women showed resolution

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of abnormal renal parameters within 3 months, a significant minority had persistent dysfunction. Given the marked study heterogeneity, prospective cohort studies with standardised criteria and longer follow-up are needed to define the true recovery and eventual outcome of women whose renal function has not yet returned to normal.

## LO-16

### Proteomic Profiling of Recurrent endocapillary IgA Nephropathy in Kidney Allografts Reveals a complement-dominated signature

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**Background:** Recurrent IgA nephropathy (IgAN) is a major cause of kidney allograft dysfunction, but its intragraft molecular characteristics remain poorly defined. We aimed to characterize proteomic changes across the histological severity of recurrent IgAN.

**Methods:** We performed label-free quantitative mass spectrometry on 19 kidney allograft biopsies from 18 transplant recipients. Biopsies were classified as recurrent IgAN without endocapillary proliferation (IgAN_MildRecu), recurrent IgAN with endocapillary proliferation (IgAN_ProlifRecu). IgAN without recurrence (IgAN_NoRecu), or non-IgAN transplant(Other-KTx) served as a contol groups. Differential protein abundance was assessed using DEqMS with a nested model comparing mild recurrence with controls and proliferative recurrence with mild recurrence. Pathway enrichment was evaluated using Reactome and KEGG gene set enrichment analyses.

**Results:** IgAN_ProlifRecu had higher proteinuria than the other groups (median, 2.6 versus 0.19 g/day; $P$ = 0.03). IgAN_MildRecu was predominantly characterized by alterations in metabolic pathways, including purine, glucose, glycogen, and sulfur amino acid metabolism. In contrast, IgAN_ProlifRecu showed marked enrichment of complement and coagulation pathways. Mapping onto the KEGG complement and coagulation cascade revealed proteins belonging to the alternative, classical/lectin, and terminal complement pathways. Protein-level anal-ysis confirmed increased abundance of membrane attack complex components C5–C9 and complement factor H-related protein 5 (CFHR5) in proliferative compared with mild recurrence, with CFHR5 showing an approximately fourfold increase (log2 fold change ≈2; adjusted $P$<0.05). Terminal complement components and CFHR5 showed a stepwise increase across IgAN_NoRecu, IgAN_MildRecu, and IgAN_ProlifRecu. IgAN_ProlifRecu additionally exhibited increased abundance of immune-cell-associated proteins, including CD45, CD16A, and cathep-sin G.

**Conclusions:** Recurrent IgAN exhibits stepwise proteomic changes, progressing from predominantly metabolic alterations in mild disease to extensive complement activation and immune-cell infiltration in proliferative lesions. Terminal complement components and CFHR5 may represent biomarkers and therapeutic targets in severe recurrent IgAN.

## LO-17

### PTLD CART-cell therapy in a kidney transplant recipient

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Post-transplant lymphoproliferative disorder (PTLD) is a rare but severe complication following kidney trans-plantation. Treatment depends on aggressiveness, CD20 expression, and response to immunosuppression re-duction. Rituximab is first-line for CD20+ disease, but CD20– or rituximab-refractory cases require alternative chemotherapy. CAR T-cell therapy, increasingly used in classical lymphomas, has shown promise in renal transplant recipients with PTLD, though concerns persist regarding cytokine release syndrome (CRS), immune effector cell-associated neurotoxicity syndrome (ICANS), and allograft rejection.

We report a 55-year-old male kidney transplant recipient with IgA nephropathy, maintained on mycophenolate and tacrolimus, who was diagnosed with late, EBV-negative PTLD. Initial management with immunosuppression reduction and one rituximab dose failed. Subsequent R-CHOP chemotherapy induced an excellent response, followed by R-ICE for suspected meningeal involvement. Consolidation with anti-CD19 CAR T-cell therapy was then administered. Immunosuppression was temporarily tapered to prednisone 10 mg/day monotherapy, before leukapheresis and maintained at reinfusion. Following CAR T-cell infusion, the combination of prednisone and low-dose tacrolimus was resumed. The treatment was well tolerated, with no CRS, ICANS, or graft rejection observed. Although the patient ultimately lost his graft >1 year later due to chronic allograft dysfunction, he remains in complete remission two years after CAR T-cell infusion.

This case demonstrates that CAR T-cell therapy can induce durable remission in renal transplant recipients with late PTLD without precipitating graft rejection, provided immunosuppression is appropriately maintained at reduced doses. These findings support the feasibility of CAR T-cells in this high-risk population and highlight the importance of individualized immunosuppressive management to balance oncologic control and graft preservation.

## LO-18

### Renal denervation in dialysis patients with resistant hypertension: a promising treatment option

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**Case:** We present a 50-year-old female patient on chronic hemodialysis with severe treatment-resistant arterial hy-pertension. Despite seven antihypertensive drugs, including two diuretics, 24-hour ambulatory blood pressure monitoring (ABPM) showed a mean systolic blood pressure (SBP) of 185 mmHg and diastolic blood pressure (DBP) of 96 mmHg. Hypertension was attributed primarily to renoparenchymal disease. There was no evi-dence of medication non-adherence or renovascular disease on duplex sonography. Due to recurrent hyper-tensive crises requiring hospitalization, catheter-based renal denervation (RDN) was considered. As the patient declined further

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medication, trans radial RDN was performed after shared decision-making using a dedicated radiofrequency catheter system. A total of 20 and 32 ablations were applied to the right and left renal arteries, respectively. Initially, blood pressure remained unchanged. Six weeks after RDN, however, a marked reduc-tion was observed, with home measurements below 100 mmHg, allowing gradual reduction of antihypertensive medication. Eight weeks after RDN, ABPM on dual therapy showed a mean BP of 138/84 mmHg.

**Discussion:** Hypertension in end-stage kidney disease (ESKD) is multifactorial, involving sodium retention, activation of the renin–angiotensin–aldosterone system, endothelial dysfunction, and sympathetic over activity. Sympathetic activity increases progressively with declining renal function and is particularly pronounced in ESKD. Inter-rupting renal sympathetic pathways may therefore represent a biologically plausible therapeutic approach. However, sham-controlled RDN trials largely excluded patients with advanced chronic kidney disease, and ro-bust evidence in patients receiving dialysis remains limited. Small studies suggest that RDN may be feasible and associated with reductions in blood pressure and sympathetic activity in advanced kidney disease.

**Conclusion:** This case demonstrates a marked antihypertensive response following RDN in a hemodialysis patient with treatment-resistant hypertension. RDN may represent a potential therapeutic option in carefully selected pa-tients with ESKD, although prospective studies are needed to establish its safety, efficacy, and clinical benefit in the dialysis population.

## LO-19

### Sex-dependent macrophage states and tubular repair after renal ischemia-reperfusion injury.

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**Background:** Sex differences influence outcomes after acute kidney injury (AKI), but the mechanisms linking sex to repair and fibrosis remain incompletely understood. We hypothesized that female sex promotes a more regulated and reparative innate immune response after acute kidney ischemia-reperfusion injury (IRI), leading to better renal outcomes.

**Methods:** Male, intact female and ovariectomized female C57BL/6 mice, with and without sex hormone replacement, underwent unilateral renal IRI and were analyzed at the acute (day 2), transitional (day 7) and repair phases (days 14 and 28). Tubulo-interstitial injury, repair and fibrosis were assessed by histology, immunohistochemistry and bulk RNA sequencing. Renal infiltrating leukocytes were characterized by flow cytometry, immunofluorescence and single-cell RNA sequencing, complemented by regulatory network and ligand-receptor analyses.

**Results:** Females developed less chronic tubulo-interstitial injury than males, with reduced maladaptive tubular repair and preserved tubular metabolic programs. This protection was lost after ovariectomy and restored by estradiol administration. Immune profiling revealed marked sex-dependent differences in tissular myeloid recruitment and macrophage states. Male kidneys had a greater macrophage infiltration, enriched in hypoxia-and scar-associated phenotypes, whereas female kidneys had a higher relative proportion of regulatory and resident-like macrophages. These immune differences appeared mostly during the repair phase and paralleled the divergent fibrotic trajectories.

**Conclusions:** Sex shapes the chronic inflammatory response after kidney IRI. Female kidney protection is as-sociated with reduced maladaptive tubular repair, lower macrophage accumulation and a shift away from scar-associated macrophage states. Manipulation of sex hormone-signaling resulted in intermediate tissue and immune phenotypes. These findings identify sex hormones and macrophage polarization as potential determinants of AKI-to-CKD transition and support sex-specific therapeutic strategies after AKI.

## LO-20

### Single-nucleus analysis reveals renal STAT1/CEBPB signaling as a modifiable relay of proinflammatory FGF23 effects in diabetic kidney disease

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**Background:** fibroblast Growth Factor 23 (FGF23) increases during chronic kidney disease, especially in diabetic kidney disease (DKD), speculatively due to lack or resistance to its negative regulator insulin. Here, we investigated cell-specific renal actions of FGF23 in mice with early DKD.

**Methods:** DBA/2J mice received sex-adapted doses of streptozotocin or vehicle to induce DKD. After measurement of glomerular filtration rate (GFR), mice received FGF23 300ng or vehicle for 1h. 10X Gene Expression kit v4 was used for renal single-nuclei kidney profiling of males, followed by analysis of differentially expressed genes and regulon activity. Precision-cut kidney slices of mice were treated with Cebpb inhibitor ST-101 and FGF23 for bulk RNAseq and gene set enrichment analysis.

**Results:** Males developed DKD with increased urinary albumin/creatinine and blood urea nitrogen but preserved GFR. Females failed to induce DKD, thus, nuclei from male mice' kidneys were sequenced. Clusters of all major renal cell types were annotated. FGF23 induced segment-specific target gene expression patterns that were conserved in both diseased and healthy kidney, such as *Cyp24a1* expression in both proximal tubule (PT)-segment 1 and distal convoluted tuble (DCT) segment 1. Disease-specific FGF23 effects included expression of *Wsb1* in PT-S1 and *Pdk4* in PT-S2, PT-S3, DCT1 and DCT2. Regulon activity revealed DKD-specific induction of STAT1 and CEBPB activity by FGF23 across all tubular segments. Validation experiments confirmed induction of CEBP gene set enrichment by FGF23 *ex vivo*. Addition of ST-101 before FGF23 treatment decreased both CEBP activity, proinflammatory gene sets and renal immune-like cell abundance in experimental DKD.

**Conclusion:** The present study links FGF23 signaling to STAT1-CEBPB transcription factor activity, which is a known pro-inflammatory mechanism that can be pharmacologically modified. We thus reveal a novel potential therapeutic target to alleviate the proinflammatory consequences of pathologically elevated circulating concentrations of FGF23 during kidney disease.

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## LO-21

### The impact of recipient age on the incidence of rejection and infection after kidney transplantation

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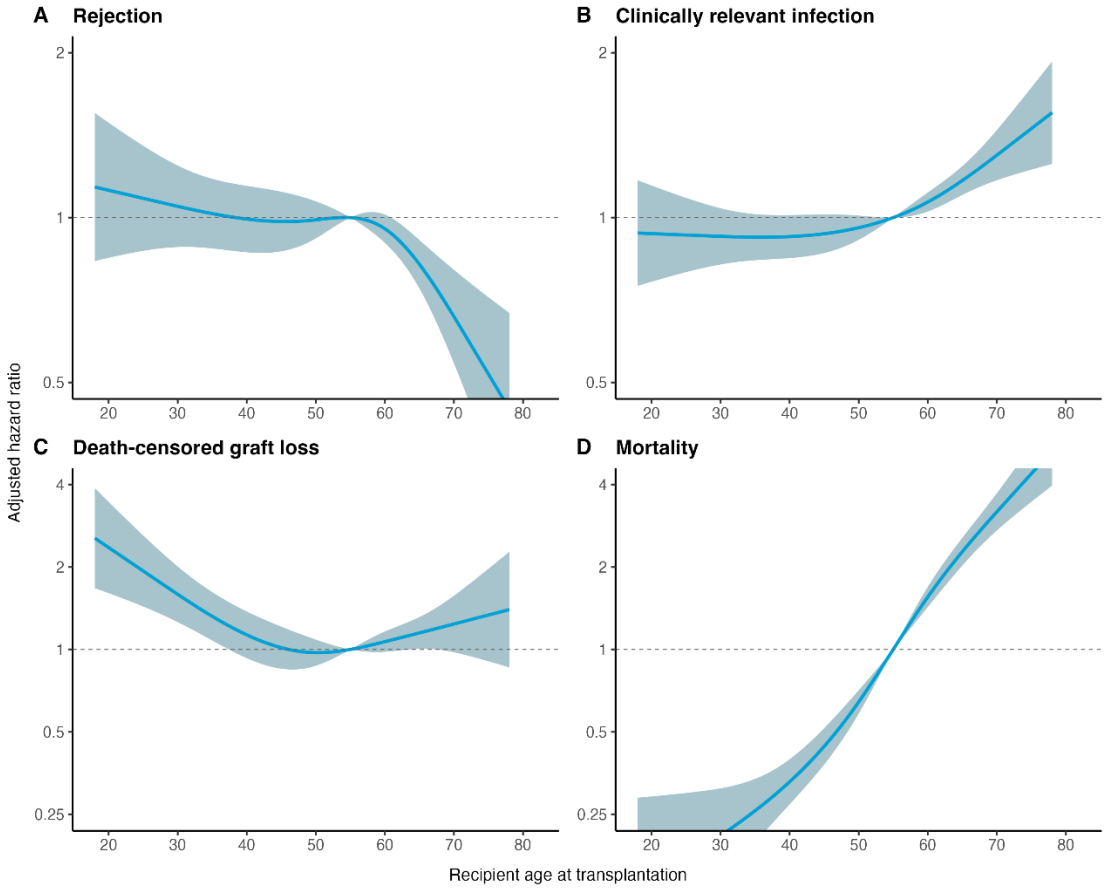
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**Background:** Older kidney transplant recipients may have a lower risk of rejection because of immunosenescence but an increased susceptibility to infection. We investigated associations of recipient age with early and long-term outcomes after kidney transplantation.

**Methods:** We included adult kidney-only transplant recipients enrolled in the Swiss Transplant Cohort Study (2008–2023). Outcomes were first biopsy-proven rejection and clinically relevant infection within 1 year, death-censored graft loss, and mortality. Age was modelled using restricted cubic splines (RCS) in adjusted cause-specific Cox models. For graft loss, Aalen–Johansen estimates accounted for death with a functioning graft as a competing event.

**Results:** Among 4,119 recipients, 940 (22.8%) experienced rejection and 2,219 (53.9%) infection within 1 year. In adjusted RCS analyses, rejection hazard decreased with age (overall P = 0.0001; nonlinearity P = 0.007) whereas infection hazard increased (overall P<0.0001; nonlinearity P = 0.010). Recipient age was also associated with death-censored graft loss (overall P = 0.0002), with higher hazards at both younger and older ages (nonlinearity P = 0.0007), while mortality increased strongly with age (overall P<0.0001; nonlinearity P = 0.14) (Figure 1). Competing-risk analysis demonstrated a marked age-dependent increase in death with a functioning graft, whereas the cumulative incidence of graft loss varied comparatively little across age groups. At 10 years, the cumulative incidence of death with a functioning graft increased from 2.8% in recipients aged 18-40 years to 47.4% in those aged >70 years, while the corresponding cumulative incidence of graft loss was 14.5% and 16.3%, respectively. Although the age association with first-year rejection was predominantly driven by events within the first 90 days, landmark analyses suggested a persistent inverse association between attained age and sub-sequent rejection during longer-term follow-up.

**Conclusions:** Recipient age was associated with divergent rejection and infection risks and distinct long-term graft and patient outcomes. These findings support age-adapted immunosuppressive and surveillance strategies.





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## LO-22

### When fasting turns harmful: severe hypokalaemia and rhabdomyolysis due to liquorice ingestion during Ramadan

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**Background:** Liquorice-induced apparent mineralocorticoid excess is a rare but important cause of hypokalaemia and metabolic alkalosis. Severe complications such as rhabdomyolysis are uncommon and usually occur in the presence of additional risk factors.

**Case presentation:** A 61-year-old man presented with diffuse myalgia following prolonged Ramadan fasting. Laboratory testing revealed severe hypokalaemia, metabolic alkalosis, and rhabdomyolysis. Medical history included coronary artery disease, type 2 diabetes, obstructive sleep apnoea, and prior sleeve gastrectomy. Further history revealed daily consumption of approximately 1 litre of the liquorice-based beverage "Erq Sous" mixed with sodium bicarbonate. Renin and aldosterone levels were suppressed, consistent with liquorice-induced apparent mineralocorticoid excess. Autoimmune myositis and statin-associated necrotizing myopathy were excluded. Treatment consisted of potassium replacement, discontinuation of diuretics and precipitating agents. Electrolytes and muscle enzymes normalised with therapy.

**Discussion:** Liquorice inhibits 11β-hydroxysteroid dehydrogenase type 2, resulting in cortisol-mediated mineralocorticoid receptor activation. In this patient, fasting-related volume depletion, diuretic therapy, and bicarbonate intake likely amplified the metabolic disturbance.

**Conclusion:** This case highlights liquorice ingestion as an under-recognised cause of severe hypokalaemia and rhabdomyolysis and underscores the importance of detailed dietary history, particularly in a selected patient population during Ramadan fasting.



Fig. 1: MRI image of the thighs showing significant muscle edema, particularly in the quadriceps femoris muscles on both sides (indicated by the arrows)

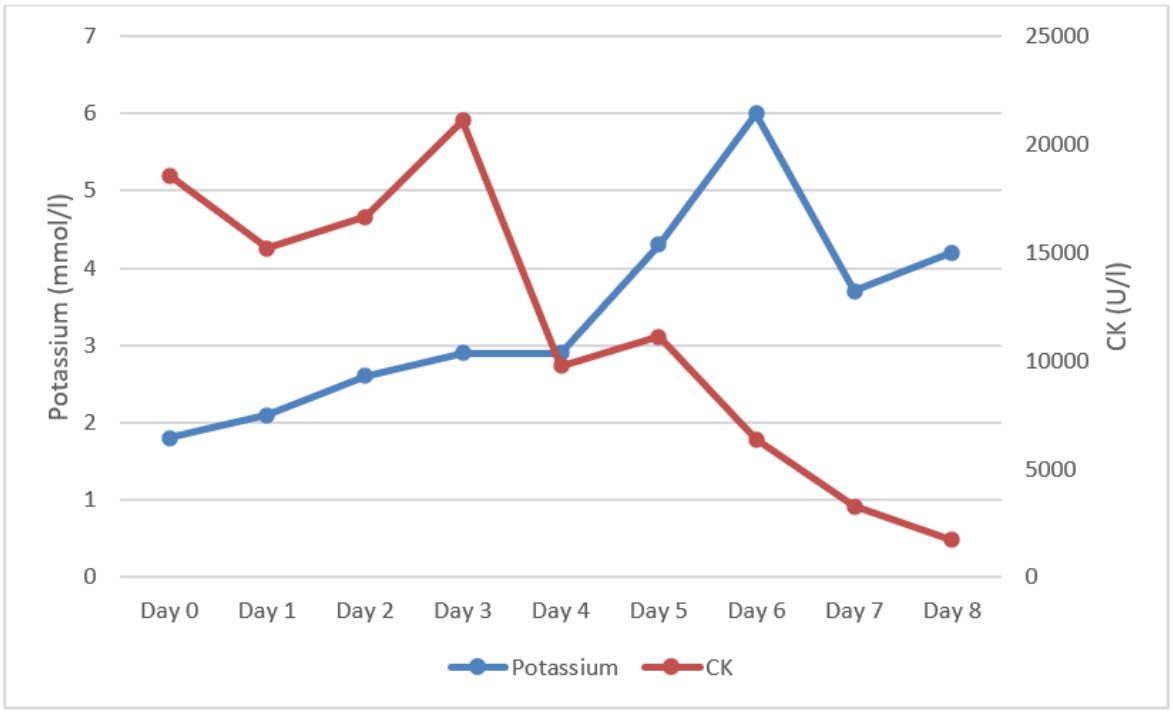



Fig. 2: Potassium (mmol/l) and CK (U/l) levels during the hospitalization


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## SHORT ORAL PRESENTATIONS

### SO-01

### Successful treatment of refractory GAVE with bevacizumab enabling kidney transplantation

Dr. Nina Weber[1], Dr. Bernhard Kaspar Morell[1], Dr. Adrian Schmidt[1], Ms. Rebecca Guidotti[1]

*1. Stadtspital Zürich*

Gastric antral vascular ectasia (GAVE) is an uncommon cause of chronic gastrointestinal bleeding, frequently associated with cirrhosis and chronic kidney disease. Patients often develop severe iron deficiency anemia and transfusion dependency. Endoscopic therapies such as argon plasma coagulation, radiofrequency ablation, and rubber band ligation are standard treatments; however, some patients remain refractory with persistent bleeding. We report a dialysis dependent kidney transplant candidate with refractory GAVE who remained profoundly anemic and transfusion dependent despite multiple endoscopic interventions, rendering him inactive on the transplant waiting list. Following treatment with bevacizumab, bleeding stabilized, transfusion requirements resolved, and hemoglobin improved sufficiently to allow successful activation and subsequent kidney transplantation.

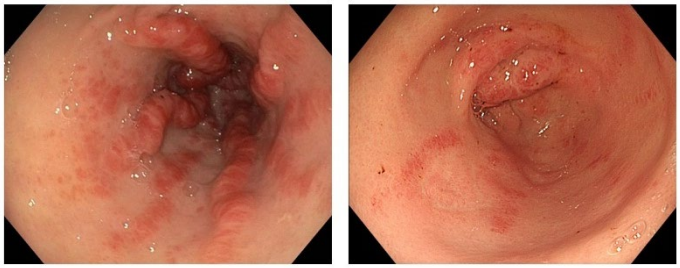


Figure 2 a) b)

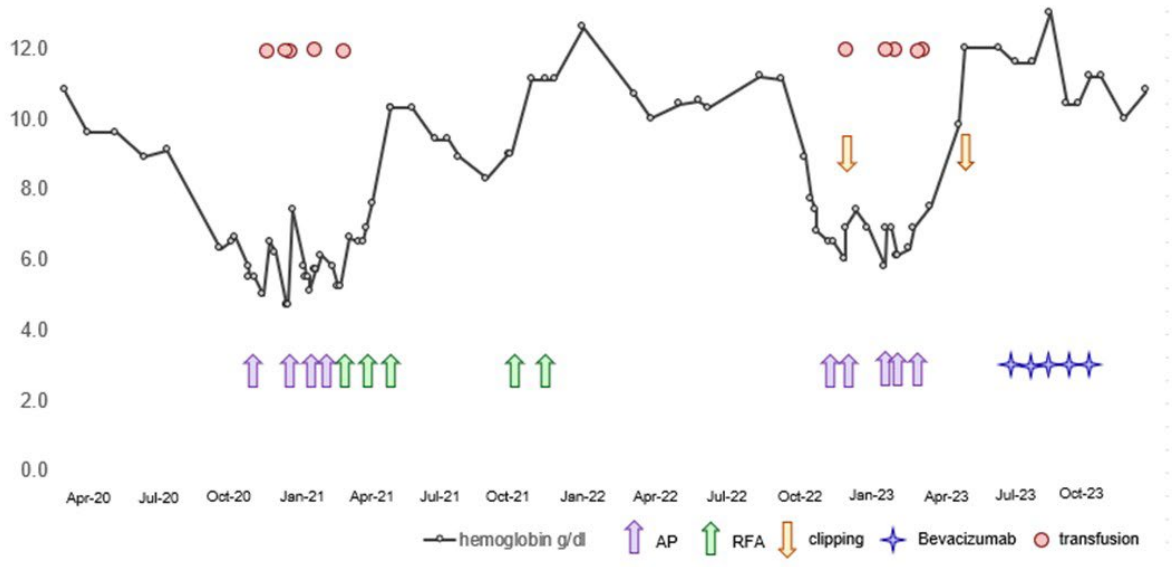



Figure 1


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## SO-02

### A novel clinical prediction model to support kidney transplant decision-making

Dr. Simon Schwab[1], Prof. Daniel Sidler[2], Dr. Fadi Haidar[3], Dr. Christian Kuhn[4], Prof. Stefan Schaub[5], Dr. Linard Hoessly[6], Dr. Michael Koller[6], Dr. Katell Mellac[6], Mr. Agim Thaqi[7], Dr. Ueli Stuerzinger[6], Mr. Bruno Tischhauser[6], Mr. Andreas Elmer[1], Dr. Nicola Franscini[1], Dr. Nathalie Kruegel[1], Prof. Thomas Müller[1], Ms. Lisa Straumann[1], Dr. Isabelle Binet[4], Prof. Dela Golshayan[8], Dr. Kerstin Huebel[9], Prof. Thomas Fehr[10], Dr. Franz Immer[1]

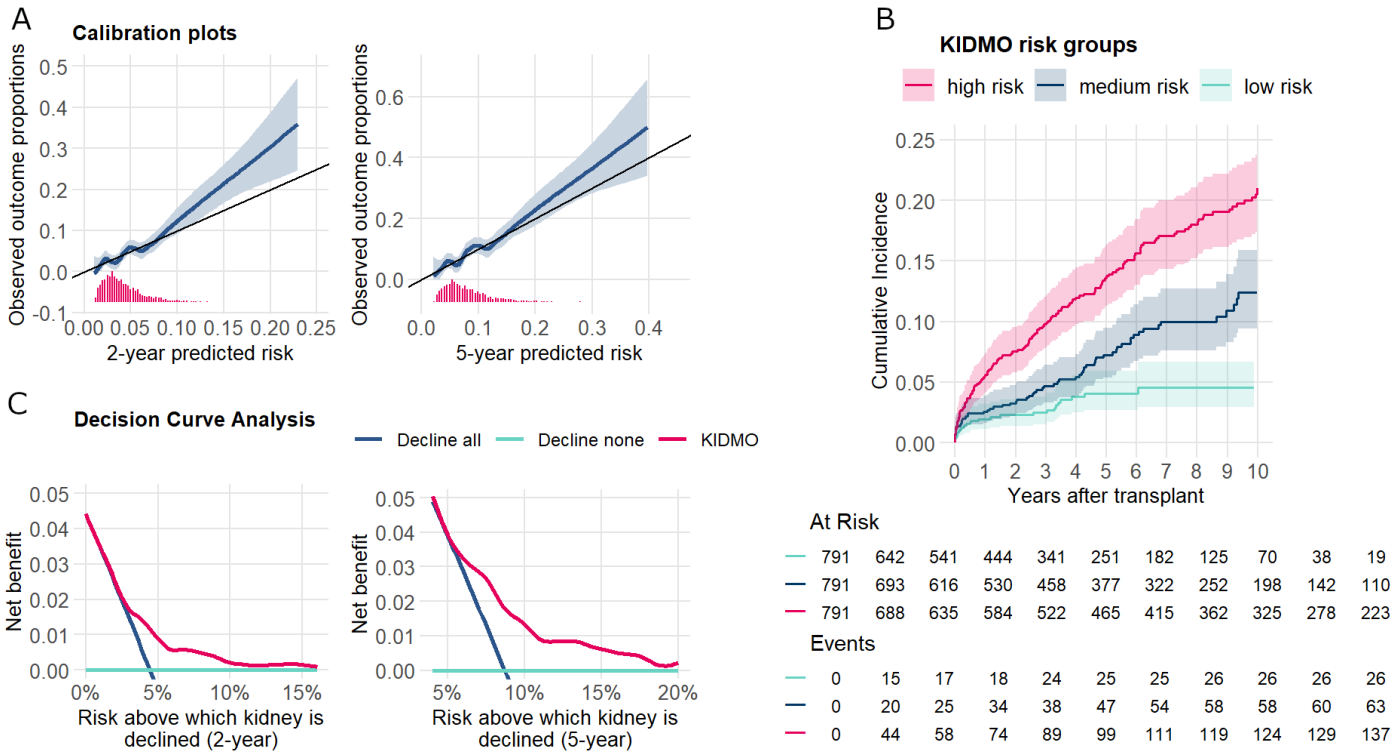
*1. Swisstransplant, Bern, 2. Department of Nephrology and Hypertension, Inselspital, Bern University Hospital and University of Bern, Bern, 3. Division of Nephrology, Geneva University Hospitals, 4. Department of Nephrology and Transplantation Medicine, HOCH Health Ostschweiz, Cantonal Hospital St.Gallen, Switzerland, 5. Clinic for Transplantation Immunology and Nephrology, University Hospital Basel, Basel, 6. Swiss Transplant Cohort Study, University Hospital Basel, Basel, Switzerland, 7. Federal Office of Public Health (FOPH), 8. Centre hospitalier universitaire vaudois, 9. Department of Nephrology, University Hospital Zurich, Zurich, 10. Zurich Kidney Center, University of Zurich, Zurich*

**Background:** Risk prediction of future graft loss at the time of organ offer can support decision-making in deceased-donor kidney transplantation, but existing models often combine graft loss with death or rely on post-transplant variables, limiting their clinical applicability. We developed and validated a clinical prediction model to estimate 2- and 5-year graft loss risk at the time of organ offer while accounting for competing mortality.

**Methods:** KIDMO (kidney prediction model) was a retrospective, multicenter prognostic model development study. The study included six Swiss transplant centers and all kidney recipients from deceased donors with transplantations between May 5, 2008, and December 31, 2021. The primary outcome was the time from trans-plantation to graft loss, with death as a competing risk.

**Results:** Among 2,373 kidney recipients, 236 experienced graft loss, and 336 died with functioning grafts. Cu-mulative incidence of graft loss was 4.4% (95% CI, 3.6% to 5.3%) at 2 years and 8.7% (95% CI, 7.4% to 10.0%) at 5 years. On internal validation, the model achieved a C-statistic of 0.65 (95% CI, 0.60 to 0.71) at 2 years and 0.63 (95% CI, 0.60 to 0.68) at 5 years, with good overall calibration (Fig. 1A). Cumulative incidence curves for graft loss were well separated across risk strata (Fig. 1B). Internal–external cross-validation showed heterogeneous performance across centers, indicating the need for careful transport and recalibration in new settings. Decision curve analysis demonstrated a higher net benefit across relevant decision thresholds (Fig. 1C).

**Conclusions:** This clinical prediction model provides individualized risk estimates of graft loss using routinely available data and may support organ acceptance decisions. Independent external validation is required before routine clinical implementation.





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## SO-03

### A sex-stratified pilot study on renal osteodystrophy and antiresorptive effects in the BXD consortium of recombinant inbred mice

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*1. Inselspital, Department of Nephrology, Bern, Switzerland, 2. Karolinska, 3. Department of Nephrology, Inselspital, Bern University Hospital, University of Bern, Bern, Switzerland, 4. Inselspital Bern*

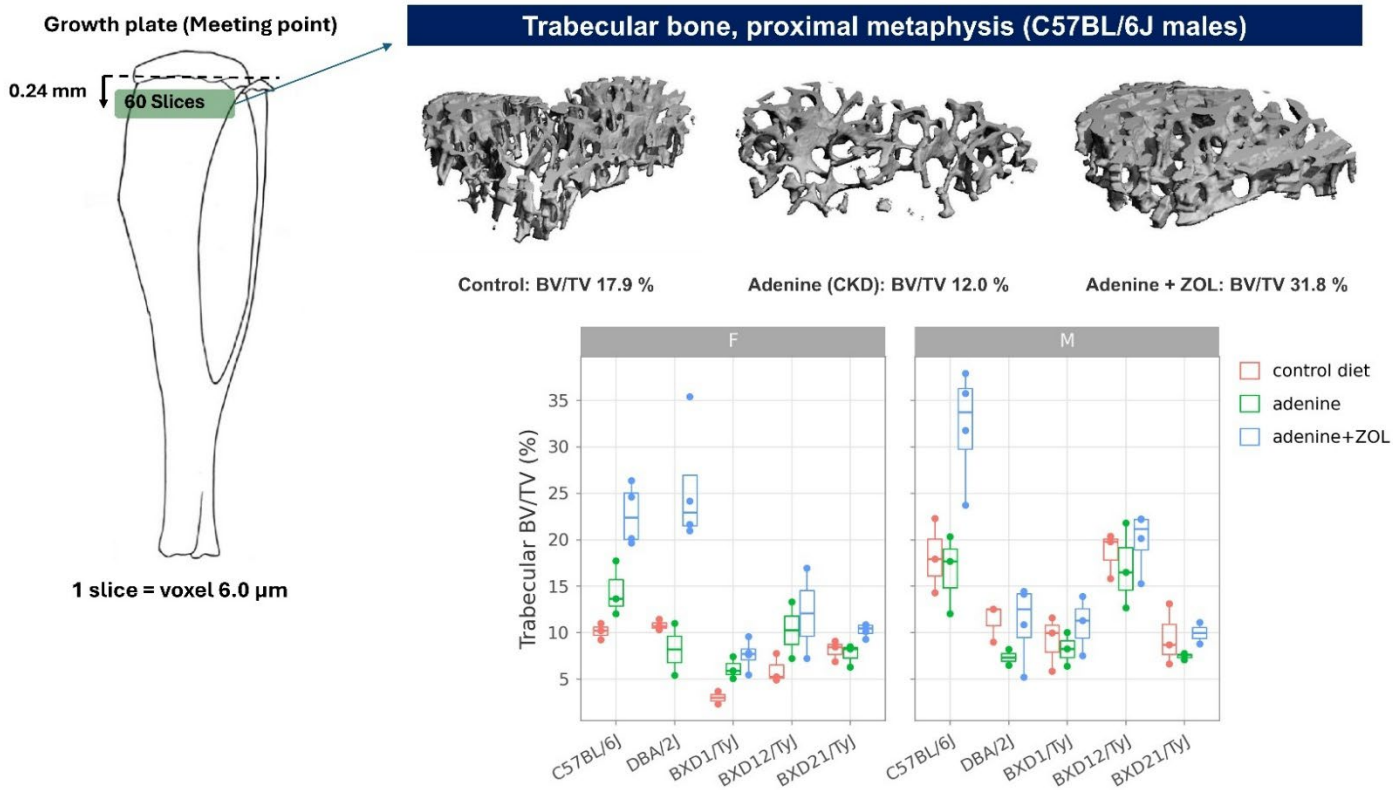
**Background:** Renal osteodystrophy (ROD) is the skeletal manifestation of chronic kidney disease-mineral and bone disorder. Genetic factors may contribute to its heterogeneity. We used BXD mice to study how genetic background and sex influence bone changes and the response to zoledronate.

**Methods:** A total of 95 mice from 5 BXD strains were studied with n = 2-4 mice per sex, group and strain. Group 1 received control diet, group 2 received 0.2% adenine diet up to 6 weeks, and group 3 received 0.2% adenine and single-dose zoledronate. Blood urea nitrogen (BUN) was measured to monitor renal impairment. Tibial micro-CT provided trabecular and cortical measurements. We performed femoral dynamic histomorphometry (n = 94) and three-point bending tests. For 33 mice lacking double labels, mineral apposition rate was imputed to 0.1 µm/day for calculations of derived parameters. Heritability ($H^2$) was estimated using a sum-of-squares method.

**Results:** Mean BUN levels increased with adenine diet from approximately 19-20 mg/dL at baseline to 38.7 mg/dL (Group 2) and 37.9 mg/dL (Group 3) by day 35. Thinner cortices were observed in Group 2 than in Group 1 in both sexes of every strain (0.135 ± 0.025 versus 0.162 ± 0.015 mm). For trabecular bone volume fraction, Group 3 averaged 17.0 ± 9.0%, compared with 10.5 ± 4.8% in Group 2 (micro-CT means ± SD across strains and sexes). Group-adjusted $H^2$ for trabecular bone volume fraction was 0.68 in males and 0.58 in females. Male/female $H^2$ values for cortical thickness, cortical porosity and bone formation rate per bone surface were 0.50/0.34, 0.51/0.13 and 0.38/0.43. Biomechanical $H^2$ ranged from 0.39 to 0.88.

**Conclusions:** All strains showed cortical thinning with adenine alone. Cortical porosity and bone formation varied between strains. Larger BXD cohorts could investigate genetically encoded mechanisms underlying ROD and zoledronate response.

Figure 1: Strain, sex and group effects on trabecular bone.





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## SO-04

### Anti-nephrin autoantibodies superimposed on a collagen type IV disease phenotype: dual nephropathy and the rituximab maintenance dilemma

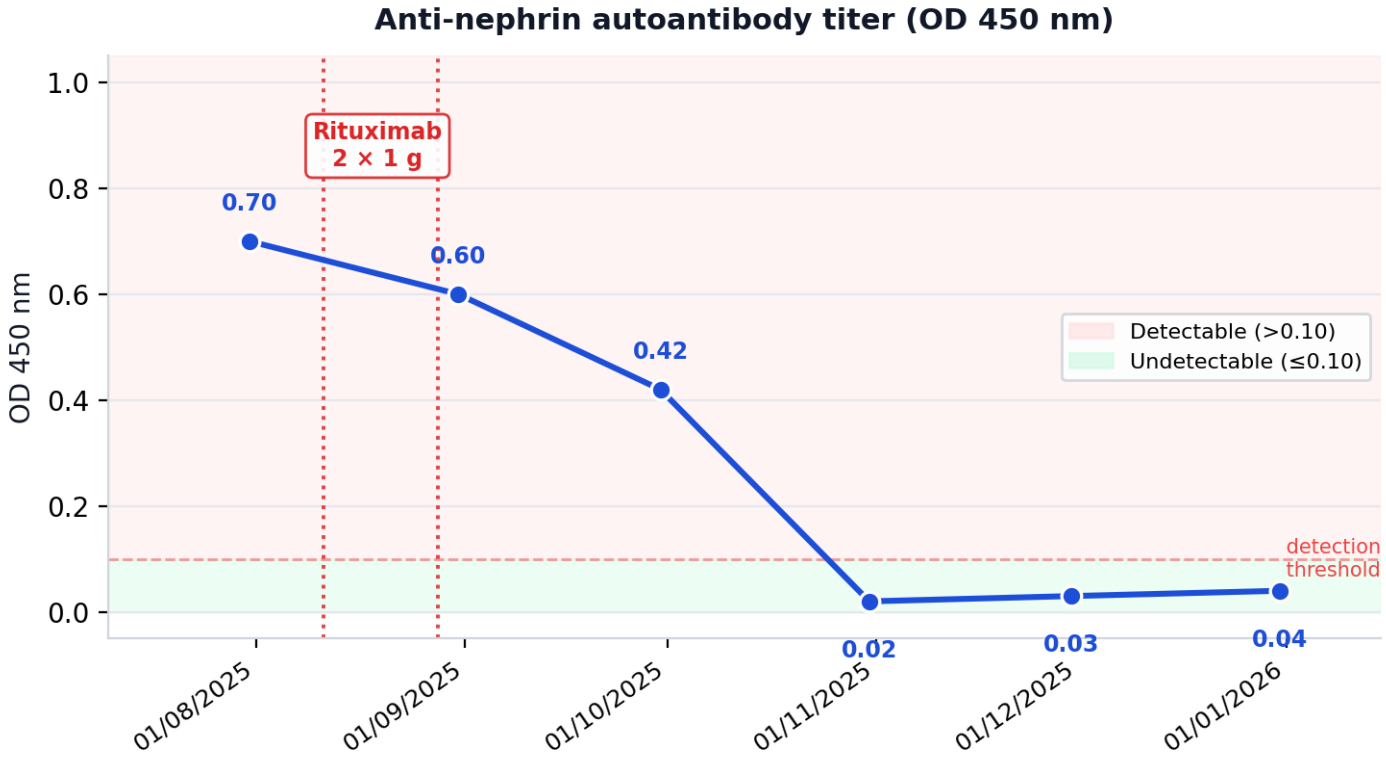
Dr. Benjamin Koch[1], Dr. Petra Rhyn[1], Dr. Hoan Nguyen[1], Dr. Larysa Lebid[1], Prof. Thomas Müller[1], Dr. Jan Brügger[2]

*1. nephro.swiss AG, Centre for nephrology and dialysis, Horgen, 2. nephro.swiss AG, Centre for nephrology and dialysis, Horgen, Switzerland*

**Background:** Anti-nephrin autoantibodies are established markers of disease activity in minimal change disease (MCD) and primary focal segmental glomerulosclerosis (FSGS), correlating with slit-diaphragm dysfunction and nephrotic syndrome. Their coexistence with hereditary collagen type IV (COL4) nephropathy has not been reported, posing unique diagnostic and therapeutic challenges.

**Case presentation:** A 42-year-old woman presented with acute kidney injury (AKI stage 3; minimum eGFR 12 ml/min/1.73 m²) and nephrotic-range proteinuria (5–9 g/day). Bilateral sensorineural hearing loss since age 5 and a family history of microhematuria (mother, sister) raised suspicion for a hereditary nephropathy. Kidney biopsy showed minimal glomerular changes on light microscopy; electron microscopy revealed thin and partially split glomerular basement membranes (GBM), suspicious for COL4-associated hereditary nephritis (Alport spectrum). Genetic testing (COL4A3/4/5 MLPA) yielded no clearly pathogenic variant; an HNF1B variant of uncertain significance was noted. Secondary immune causes were excluded (ANA, ANCA, anti-GBM, anti-PLA2R, anti-THSD7A all negative; complement normal). Anti-nephrin autoantibodies were markedly elevated (OD 450 nm 0.70). Treatment with prednisone and rituximab (2 × 1 g, August 2025) induced complete remission; anti-nephrin titers declined to near-zero by October 2025 (Fig. 1). At follow-up (April 2026), eGFR was 72 ml/min/1.73 m² with no proteinuria off all medications.

**Conclusion:** This case illustrates the rare coexistence of a COL4 nephropathy phenotype and anti-nephrin antibody-associated podocytopathy – likely 2 distinct entities in 1 patient. Rituximab achieved complete, medication-free remission. The key unresolved therapeutic question is whether preemptive rituximab redosing every 6 months is warranted to prevent relapse, or whether close monthly monitoring of proteinuria and eGFR with treatment triggered only by relapse is sufficient – a distinction of particular relevance given the underlying hereditary structural nephropathy.




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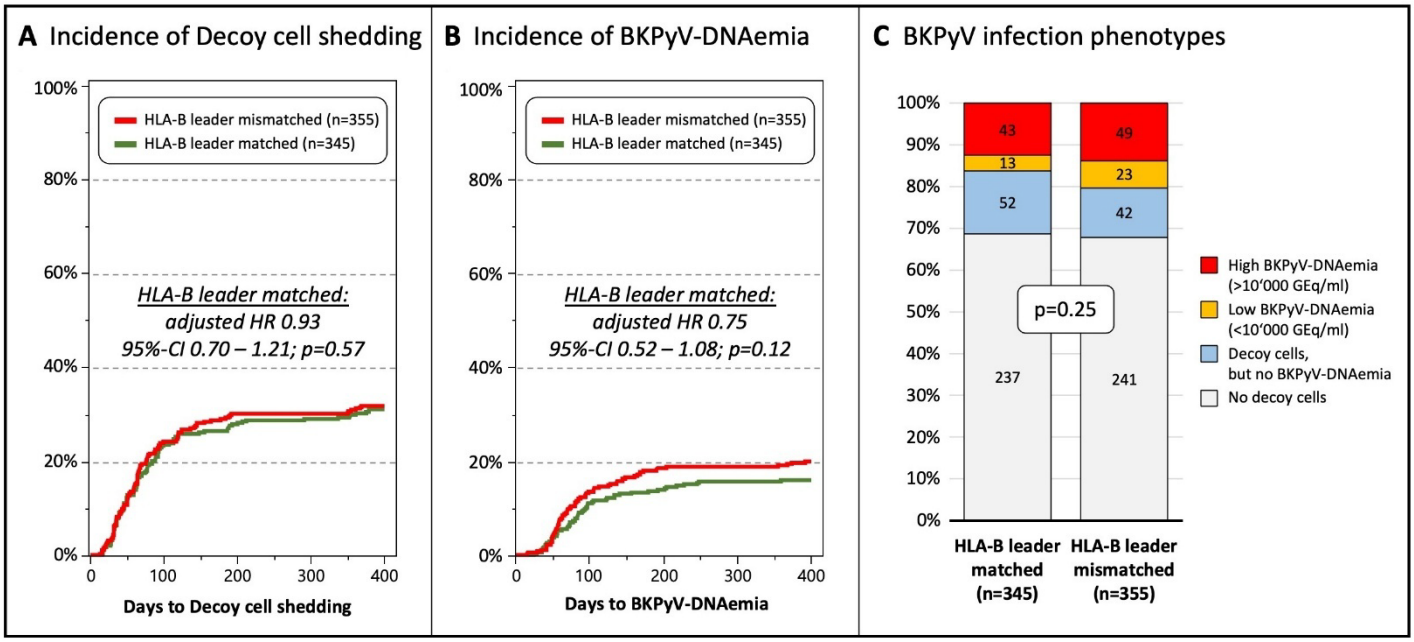
## SO-05

### Association of HLA-B leader constellation with BK polyomavirus and cytomegalovirus infections in renal allograft recipients

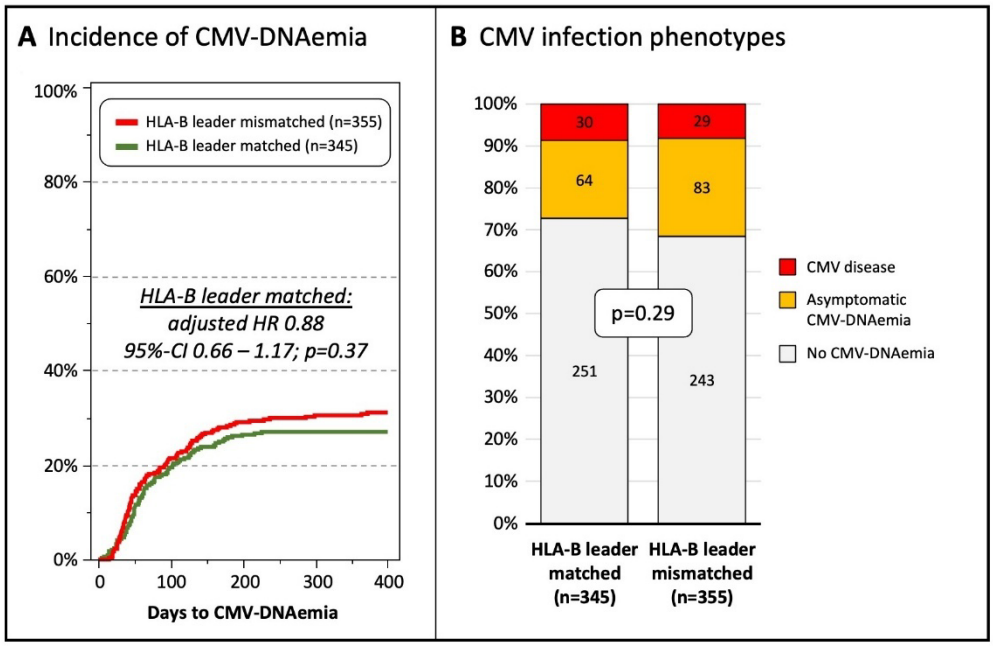
Ms. Leonie Ballmer[1], Prof. Stefan Schaub[2], Dr. Caroline Wehmeier[2], Dr. Matthias Diebold[2], Dr. Gideon Hoenger[3], Prof. Hans H. Hirsch[4]

*1. Clinic for Transplantation Immunology and Nephrology, University Hospital Basel, Basel, Switzerland, 2. Clinic for Transplantation Immunology and Nephrology, University Hospital Basel, Basel, 3. HLA-Diagnostics and Immunogenetics, Department of Laboratory Medicine, University Hospital Basel, Basel, 4. Translational Virus Immunity Pathology, Immunology Research, Department of Medical Biology, UiT The Arctic University of Norway, Tromsø, Norway*

The HLA-B leader peptide can influence natural killer (NK) cell education and function. NK cells contribute to immune responses against common viruses after kidney transplantation such as BK polyomavirus (BKPyV) and cytomegalovirus (CMV). The impact of the HLA-B leader on BKPyV and CMV infections is incompletely understood. In this study, we investigated 700 kidney allograft recipients regarding the association between recipient/donor HLA-B leader constellation and the occurrence as well as the severity of BKPyV and CMV infections. The HLA-B leader constellation was determined by high-resolution HLA typing of the recipients and donors (i.e. MM, MT, TT). Three hundred and forty-five of 700 patients (49%) had an HLA-B leader match, 355/700 (51%) a mismatch. The one-year incidence of decoy cell shedding and BKPyV-DNAemia was not different between the two groups (Figure 1A/B). In addition, we found no statistically significant differences regarding the BKPyV infection phenotypes between the two groups (p = 0.25) (Figure 1C). In multivariable models, an HLA-B leader match was not associated with decoy cell shedding (HR 0.93 [95%-CI 0.70-1.21], p = 0.57), BKPyV-DNAemia (HR 0.75 [95%-CI 0.52-1.08], p = 0.12), and occurrence of presumptive BKPyVAN (OR 0.88 [95%-CI 0.56-1.38], p = 0.58). Individual recipient/donor HLA-B leader constellations (i.e. MT/MT, MT/TT, TT/MT, or TT/TT) were also not associated with any BKPyV infection events. In addition, we observed no association between an HLA-B leader match or individual constellations and the occurrence of CMV-DNAemia/CMV disease (Figure 2). In conclusion, the HLA-B leader constellation is neither associated with the occurrence nor the severity of BKPyV and CMV infections within the first year posttransplant in kidney allograft recipients. This argues against the use of HLA-B leader information to guide screening strategies for BKPyV and CMV.




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## SO-06

### ATTEND-NN: Language Barriers and Appointment Burden Are Associated with Non-attendance at Non-nephrology Clinics in Patients Receiving Dialysis

Dr. Nina Weber[1], Ms. Rebecca Guidotti[1]

*1. Stadtspital Zürich*

**Background:** Dialysis patients require care from multiple specialties, but non-attendance at non-nephrology outpatient appointments is poorly studied. ATTEND-NN assessed the frequency of no-shows and associated factors among dialysis patients at Stadtspital Zürich.

**Methods:** We retrospectively analyzed 83 patients receiving hemodialysis or peritoneal dialysis for ≥90 days in 2023. Non-nephrology outpatient appointments were extracted from electronic health records and clinical data from the Swiss Dialysis Registry. Associations with ≥1 no-show were assessed using uni- and multivariable logistic regression. Time from dialysis initiation to transplant listing and overall survival through 31 December 2025, were compared between groups.

**Results:** Overall, 24/83 patients (28.9%) had ≥1 no-show. Patients with no-shows had more outpatient appointments per year than those without (median 19.5 [IQR 9.3– 32.7] vs. 7.0 [IQR 3.0–15.0], p<0.001). In multivariable analysis, greater appointment burden was associated with higher odds of ≥1 no-show (OR 1.06, 95% CI 1.02–1.10, p = 0.007), as was limited German language proficiency (OR 4.55, 95% CI 1.39–14.29; p = 0.013). Among 19 patients listed for transplantation after dialysis initiation, time to listing did not differ between groups (median 1.0 vs. 1.3 years, p = 0.840). During follow-up, 15 patients died; overall survival was not significantly different (log-rank p = 0.168).

**Conclusions:** Nearly one third of dialysis patients missed at least one non-nephrology appointment. Non-attendance was independently associated with greater appointment burden and limited German language proficiency, rather than comorbidity or dialysis-related characteristics, suggesting that healthcare utilization and communication barriers may be relevant. These findings suggest that more selective and coordinated scheduling, together with targeted language support, may reduce appointment burden and improve access to necessary specialist care. Larger studies are needed to determine whether non-attendance translates into adverse patient outcomes.

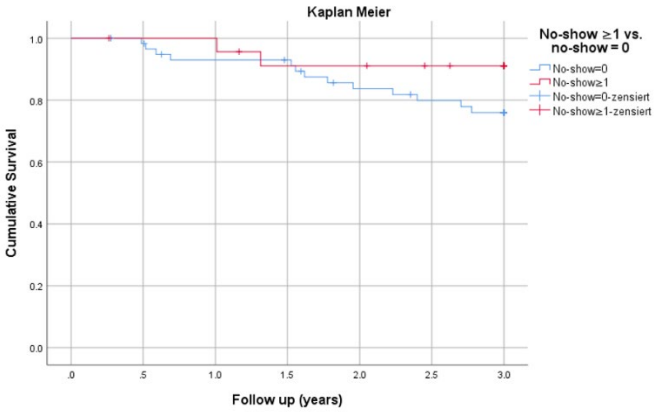



**Table 1. Baseline characteristics of dialysis patients by no-show status**

| Variable | NoShow=0 (n=59) | NoShow≥1 (n=24) | p-value |
|---|---|---|---|
| Age, years, mean±SD | 69.0±14.6 | 61.7±14.2 | 0.042 |
| Male sex, n (%) | 35 (59.3) | 17 (70.8) | 0.326 |
| Transplant candidate | 16 (27.1) | 10 (41.7) | 0.195 |
| Migration background | 20 (33.9) | 13 (54.2) | 0.087 |
| Nor or limited German language skills, | 10 (16.9) | 12 (50.0) | 0.002 |
| BMI, median (IQR) | 25.8 (23.0-30.7) | 24.5 (20.7-27.7) | 0.094 |
| Charlson Comorbidity Index, median (IQR) | 4.0 (3.0-6.0) | 4.5 (3.0-6.0) | 0.558 |
| Outpatient appointments/year, median (IQR) | 7.0 (3.0-15.0) | 19.5 (9.3-32.7) | <0.001 |
| Dialysis vintage at baseline, years, median (IQR) | 2.7 (0.4-3.7) | 1.1 (0.0-3.4) | 0.119 |

BMI, body mass index; IQR, interquartile range; SD, standard deviation


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## SO-07

### Belatacept Conversion in Simultaneous Pancreas-Kidney and Liver-Kidney Transplant Recipients: A Single-Center Cohort and Literature Synthesis

Dr. Dusan Harmacek[1], Dr. Elena Rho[1], Dr. Seraina von Moos[2], Dr. Severin Simmler[1], Dr. Thomas Welte[1], Dr. Fabian Rössler[3], Prof. Thomas Schachtner[1], Dr. Lukas Weidmann[1]

*1. Division of Nephrology, University Hospital Zürich, Zürich, Switzerland, 2. Division of Nephrology, Cantonal Hospital Lucerne, 3. Division of Surgery and Transplantation, University Hospital Zurich*

**Background:** Calcineurin inhibitor (CNI) nephrotoxicity drives long-term kidney-function decline after simultaneous pancreas-kidney (SPK) and liver-kidney (LK) transplantation, motivating belatacept use. However, *de novo* belatacept has been linked to excess pancreas and liver allograft rejection, and conversion beyond 6 months is poorly studied in these recipients.

**Methods:** We retrospectively studied 6 SPK and 6 LK recipients converted from CNI- to belatacept-based immunosuppression and compared their course with the published SPK/LK conversion literature. Outcomes were estimated glomerular filtration rate (eGFR) at conversion and 12 months, individual pre- and post-conversion eGFR slopes, donor-specific antibody (DSA) dynamics and rejection, metabolic and pancreatic/hepatic parameters, infections, and graft and patient survival.

**Results:** Conversion occurred a median of 81 months (interquartile range 44-110) after transplantation, always with a transient low-dose CNI or mTOR-inhibitor (n = 1) overlap (median 4 months; ≥3 months in 9/12). Median eGFR rose from 45.4 to 55.5 ml/min/1.73m$^2$ in SPK and from 38.8 to 46.3 in LK recipients, and the median individual slope changed from -3.9 to +5.6 ml/min/year. No new DSA appeared and pre-existing DSA mostly declined or stabilized. Glycated hemoglobin stayed stable, no patient became insulin-dependent, and lipase and transaminases showed no relevant rise. Two infections (*Pneumocystis* pneumonia, adenovirus) were possibly belatacept-aggravated. Over a median 25 months, 8/12 (67%) remained on belatacept.

**Conclusions:** In selected SPK and LK recipients, belatacept conversion with a transient low-dose CNI/mTOR-inhibitor overlap was feasible, with stable-to-improved kidney function and no pancreatic or hepatic safety signal; the uncontrolled design supports feasibility and safety rather than efficacy.

## SO-08

### Deep visual proteomics reveals distinct proximal tubular and glomerular injury programs in experimental diabetic kidney disease

Dr. Xiang Zheng[1], Dr. Mariavittoria d'Acierno[1], Dr. Lena Rosenbaek[1], Prof. Markus Rinschen[2], Dr. Qi Wu[1], Prof. Robert Fenton[1]

*1. Department of Biomedicine, Aarhus University, 2. Department of Biomedicine, Aarhus University; Department of Medicine, University Medical Center Hamburg-Eppendorf; Hamburg Center for Kidney Health and Cardiovascular Research Center, University Medical Center Hamburg-Eppendorf*

**Background/Aim**. Diabetic kidney disease (DKD) is the leading cause of chronic kidney disease worldwide, yet most proteomic studies analyse whole-kidney tissue, masking molecular differences between nephron compartments. We tested whether proximal tubules (PT) and glomeruli follow distinct proteomic trajectories during diabetic injury and whether compartment-specific signatures are conserved in human disease.

**Methods**. Diabetes was induced in male mice by streptozotocin (STZ) and compared with vehicle-treated controls after 16 weeks (n = 12). A deep learning-powered Deep Visual Proteomics workflow was used to segment and microdissect PT and glomeruli for data-independent acquisition mass spectrometry. Compartment-specific proteomes were analysed alongside bulk kidney tissue and compared with human proteomic data from the Kidney Precision Medicine Project (KPMP).

**Results**. STZ-treated mice developed sustained hyperglycaemia, albuminuria, tubular injury, and tubulointerstitial fibrosis. Proteomic profiling revealed marked compartment-specific remodelling. PT showed reduced abundance of proteins involved in proteostasis, cell-cycle regulation, cytoskeletal organisation, and epithelial integrity, accompanied by increased mitochondrial and lipid-metabolic pathways. In contrast, glomeruli exhibited broad suppression of oxidative metabolic processes, including fatty-acid oxidation, tricarboxylic acid cycle, and ATP-generating pathways, without evidence of compensatory metabolic upregulation. These divergent responses were largely obscured in bulk kidney analyses. Cross-species comparison demonstrated significant concordance between the PT proteome and human CKD tubulointerstitial proteomic data, with 14 of the 20 highest-priority PT candidates, including LARS2 and ANXA2, altered in the same direction. No significant concordance was observed for glomeruli.

**Conclusions**. Compartment-resolved proteomics reveals that PT and glomeruli follow fundamentally different injury trajectories in experimental DKD. PT exhibited a combined injury-and-adaptation programme, whereas glomeruli showed progressive metabolic decline without a corresponding compensatory response. These signatures were largely invisible in bulk tissue and were partially conserved in human CKD. The findings provide new insight into nephron segment-specific disease progression and demonstrate the value of compartment-resolved proteomics for understanding DKD.

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Figure 1

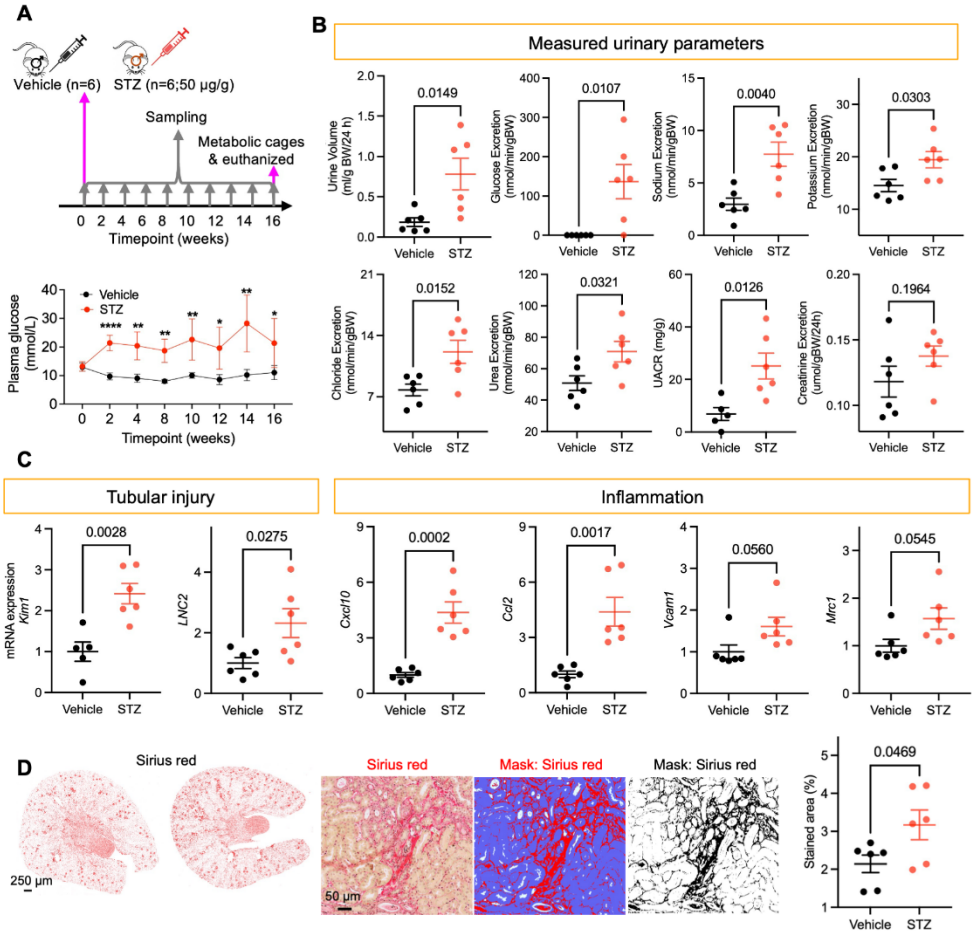



**Figure 1. Mouse STZ-induced diabetes recapitulates key hallmarks of diabetic kidney disease.** (A) Study design and plasma glucose concentrations throughout the 16-week study in vehicle and STZ-treated mice (n=6 per group). Statistical analyses were performed using a two-way ANOVA followed by Tukey multiple comparison testing. P values between conditions are denoted as follows: *P<0.05, **P<0.01, ***P<0.001, ****P<0.0001. (B) Urine volume, urinary excretion of glucose, sodium, potassium, chloride, urea, and creatinine, and urinary albumin to creatinine ratio (UACR) in vehicle and STZ-treated mice. (C) Relative kidney mRNA transcript levels of *Kim1*, *Lcn2*, *Cxcl10*, *Ccl2*, *Vcam1*, and *Mrc1* determined by RTqPCR. (D) Representative images of Sirius Red stained kidney sections showing interstitial fibrosis in vehicle and STZ-treated mice.

Figure 2

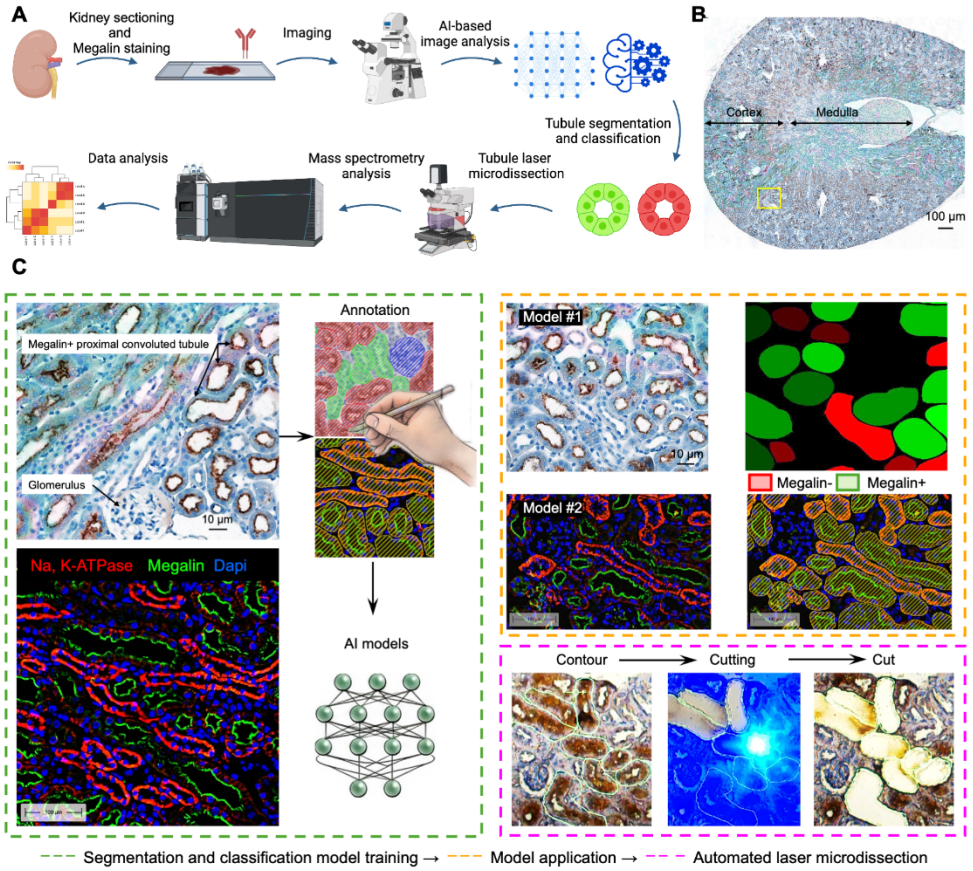



**Figure 2. Deep visual proteomics pipeline enables accurate isolation of PT and glomeruli.** (A) Schematic of the Deep Visual Proteomics (DVP) workflow, in which immunohistochemistry guided image segmentation directs automated laser microdissection of PT and glomeruli from kidneys for downstream proteomic profiling. (B) Representative whole kidney Megalin immunohistochemistry image; boxed region corresponds to region in panel C. (C) Higher magnification of the boxed region showing Megalin positive PT and Megalin negative glomeruli. Deep learning models trained on manually annotated tubules and glomeruli generated compartment masks (dotted orange box) for automated laser microdissection (dotted pink box).


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## Figure 3

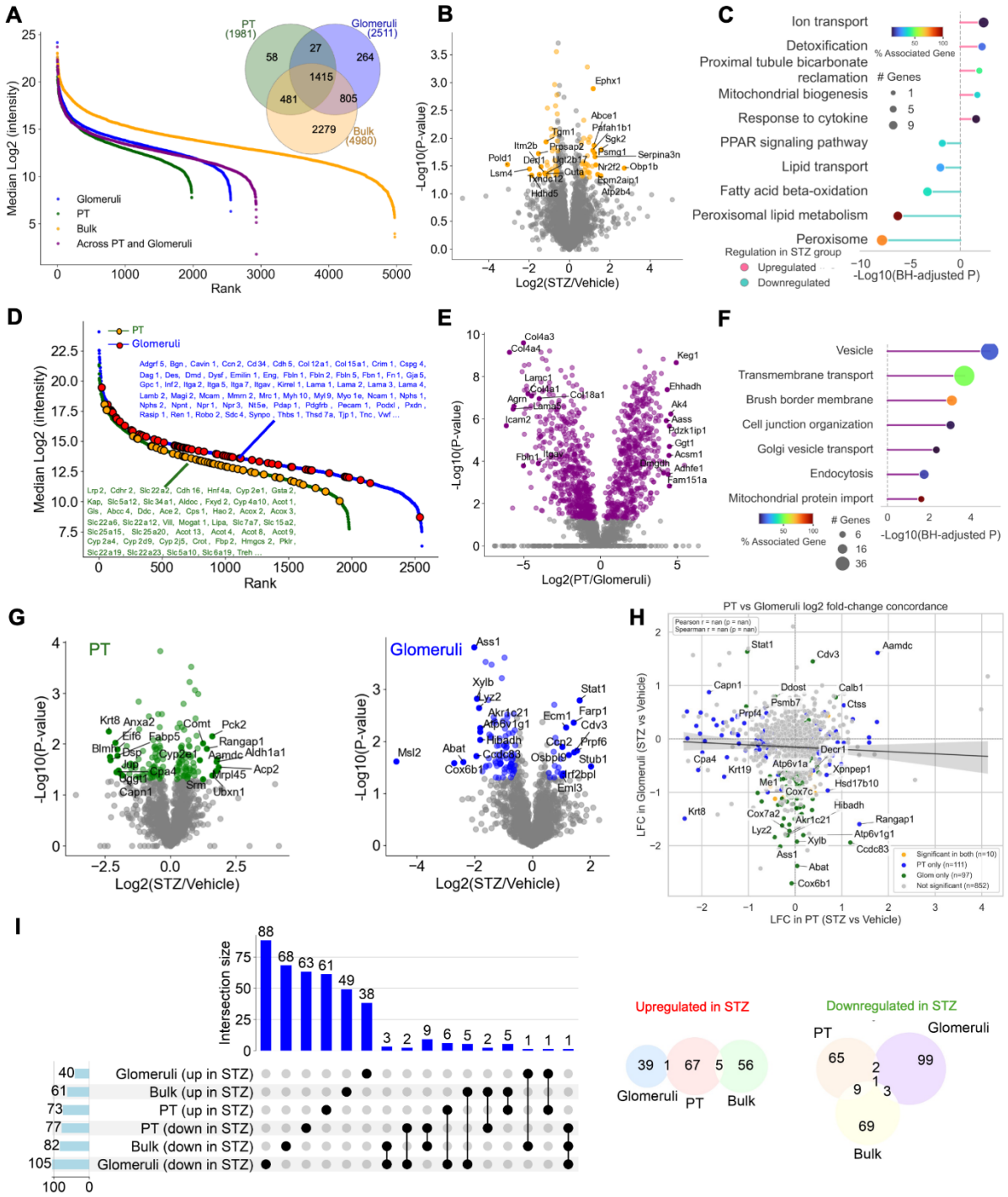



**Figure 3. Compartment resolved proteomics reveals divergent PT and glomerular alterations in DKD.** (A) Rank abundance plots of protein groups identified in bulk kidney, glomeruli, and PT, and protein numbers identified across the combined PT and glomerular datasets. (B) Volcano plot illustrating proteomic comparison of bulk kidney from STZ-treated versus vehicle mice. The top 20 differentially regulated proteins are highlighted. (C) Gene ontology and pathway enrichment for significantly changed proteins in bulk kidney. (D) Rank abundance comparison of PT and glomerular proteomes in kidneys from vehicle-treated mice show canonical compartment markers for PT, including Lrp2, Slc34a1, Slc22a6, Cyp2e1 and glomeruli, including Nphs1, Nphs2, Podxl, Pecam1. (E) Volcano plot illustrating proteomic comparison of PT and glomeruli in kidneys from vehicle-treated mice. The top 20 differentially regulated proteins are highlighted. (F) Pathways enriched in the PT relative to glomeruli. (G) Volcano plot illustrating proteins significantly altered in PT and glomeruli from STZ-treated versus vehicle mice. (H) Overlap of proteins significantly altered in both PT and glomeruli. (I) UpSet and venn diagram of significantly changed proteins across bulk kidney, PT, and glomeruli.


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## Figure 4

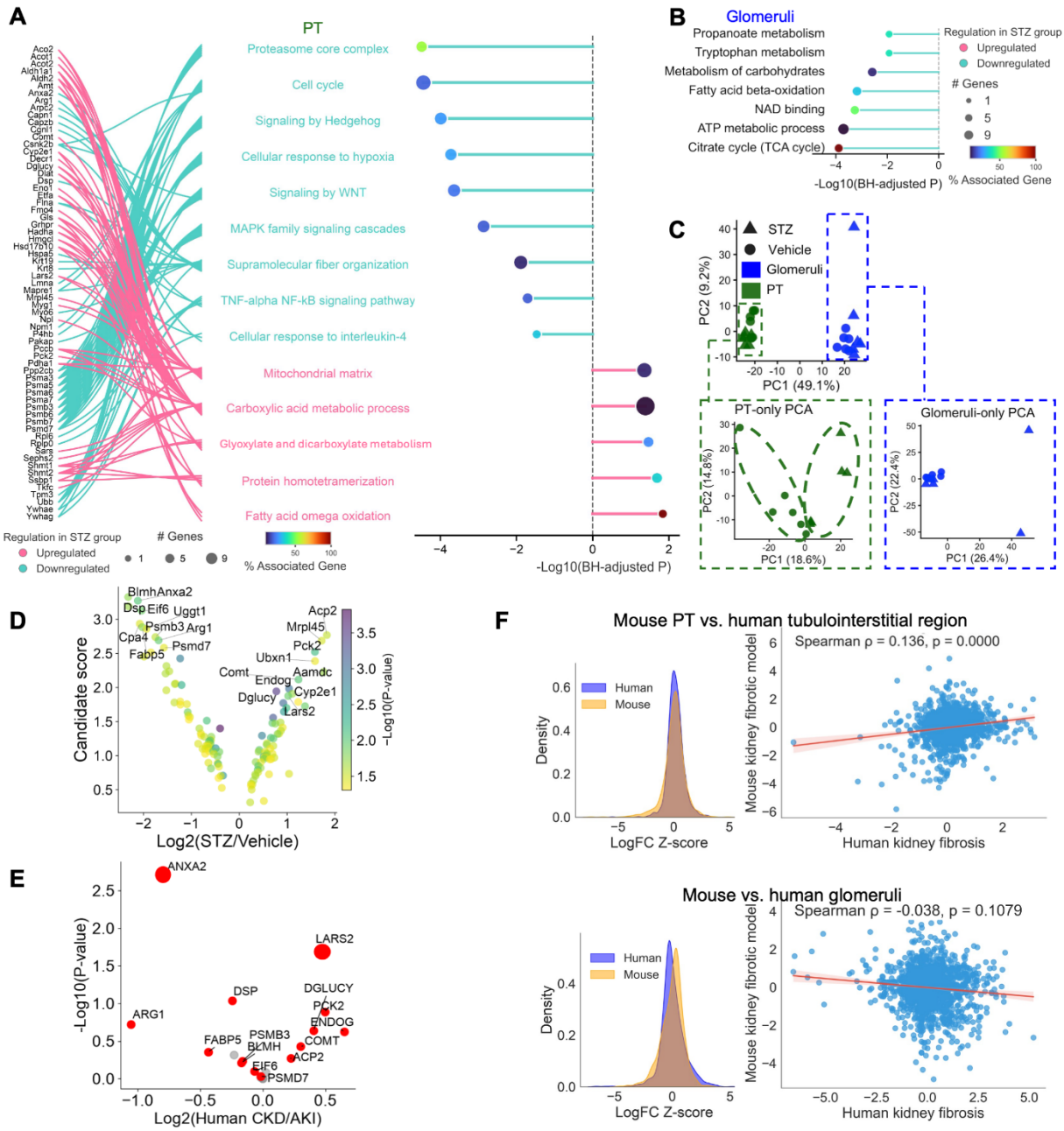



**Figure 4. A PT enriched injury signature correlates with human chronic kidney disease.** (A-B) Pathway enrichment in PT and glomeruli. In (A), sankey diagram (left) connects genes to significantly enriched pathways (right), with ribbon colors indicating direction of regulation. Differential abundance in all panels was defined by two sample *t test* (P<0.05) and an absolute fold change ≥1.5. Pathway enrichment was conducted in ClueGO, applying Benjamini–Hochberg correction and retaining terms with FDR < 0.05. (C) PCA of PT and glomeruli samples (STZ vs. vehicle). (D) PT enriched candidates ranked by a composite prioritization score integrating PT fold change, PT specificity relative to glomeruli and bulk kidney, pathway relevance, and biomarker feasibility (see Methods). Top 10 ranked increased and decreased candidates are shown. (E) Direction of change for the top 20 prioritized candidates in human chronic kidney disease data from the Kidney Precision Medicine Project. Red points indicate candidates with concordant direction of change between the STZ mouse and human datasets (14 of 20 candidates). (F) Spearman correlation of z scored log2 fold changes between the STZ proteome and the human KPMP tubulointerstitial regional proteomic dataset, shown separately for PT and glomerular comparisons.


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## SO-09

### Development of the urine solubility index (USI): an AI-assisted composite score for urinary stone risk stratification and monitoring – study protocol

Dr. Benjamin Koch[1], Dr. Petra Rhyn[1], Dr. Hoan Nguyen[1], Dr. Larysa Lebid[1], Ms. Stephanie Bull-Larsen[2], Prof. Thomas Müller[1], Dr. Jan Brügger[3]

*1. nephro.swiss AG, Centre for nephrology and dialysis, Horgen, 2. Universitätsspital Zürich, 3. nephro.swiss AG, Centre for nephrology and dialysis, Horgen, Switzerland*

**Background:** Urinary stone disease affects 5–12% of the Western population, with recurrence rates exceeding 50% at 10 years. Current risk stratification relies on individual 24h urine parameters or ion-activity product indices from spot urine (Tiselius AP(CaOx) and AP(CaP)). A composite score enabling integrated, longitudinal risk monitoring in clinical practice is lacking. AI-based platforms may facilitate the development of such scores through automated multi-source data extraction and improved visualization.

**Methods:** We developed the Urine Solubility Index (USI), a weighted composite score derived from 10 parameters of 24h urine collection: calcium, oxalate, citrate, uric acid, volume, sodium, phosphate, magnesium, pH, and protein intake. Positive USI values denote a protective urinary environment. USI calculation and visualization is powered by the mena health AI platform, which automates data extraction from diverse electronic sources and renders single-measurement profiles and longitudinal trends. We present the protocol of a prospective observational validation study comparing USI (24h urine) with Tiselius AP(CaOx) and AP(CaP) indices (spot urine) in patients undergoing standard nephrolithiasis workup.

**Results:** The mena health platform enabled rapid USI derivation from routine laboratory data. In a proof-of-concept case followed over 21 months (4 measurements), USI improved from −11.1 to +22.0 following targeted dietary counseling. Key modifiable risk factors were persistently low urine volume (0.80 L/24h; target >2.0) and acidic pH (5.0; target 5.5–7.0), partially offset by low calcium (1.6 mmol/24h) and uric acid excretion (1.84 mmol/24h). Serial USI tracking provided clear graphical feedback on treatment response for both clinician and patient.

**Conclusions:** The USI offers a clinically interpretable, multiparametric framework for kidney stone risk stratification and longitudinal follow-up. The mena health AI platform, by enabling efficient data integration from multiple sources and clear graphical visualization, substantially accelerates the development of novel composite scores. The planned validation study will benchmark USI against established Tiselius ion-activity product indices.

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## 24h-Urin-Stoffwechselprofil - Verlauf

| Sammeldatum | Körpergewicht | Geschlecht | Urin-Kreatinin | Kreatinin pro kg |
|---|---|---|---|---|
| 10. Aug. 2026 | 76.0 kg | männlich | 9.8 mmol/24h | 129 umol/kg/24h |

**Sammelqualität** 118-276 umol/kg/24h — **Adäquat**

**Urine Solubility Index (USI)** — **+22.0**

### Urine Solubility Index (USI)

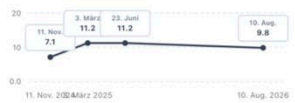
Höhere Werte zeigen ein protektiveres Profil

4 Messungen




### Urin-Kreatinin

mmol/24h — 4




### Kreatinin pro kg

umol/kg/24h — 4




### Calcium

mmol/24h — 4

Target < 6.2




### Oxalat

umol/24h — 4

Target < 500




### Citrat

mg/24h — 3

Target > 320




### Harnsäure

mmol/24h — 4

Target < 4.46




### Volumen

L/24h — 4

Target > 2.00




### Natrium

mmol/24h — 4

Target < 100




### Salzzufuhr

g/day — 4

Target < 5.7




### Phosphat

mmol/24h — 4

Target < 22




### Magnesium

mmol/24h — 4

Target > 3.30




### pH

2

Target 5.5-7.0




### Proteinzufuhr

g/kg/day — 4

Target 0.80-1.00





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## SO-10

### Establishing imaging mass cytometry for high-dimensional translational research in nephrology

Dr. Cyril Carvalho[1], Dr. Nathalie Merkel[1], Dr. Marine Nachit[1], Ms. Manh-Nhi Gass[1], Dr. Anna Rinaldi[1], Dr. Morgane Hilpert[2], Prof. Pietro E Cippà[1]

*1. Nephrology Laboratory, Department of Biomedicine, University of Basel and University Hospital Basel, 2. Flow Core Facility, DBM, University of Basel*

**Background:** Human kidney tissue presents unique challenges for multiplex imaging due to its structural complexity, intrinsic autofluorescence, and the limited amount of material available from clinical biopsies. Imaging mass cytometry (IMC) overcomes many limitations of fluorescence-based approaches by enabling simultaneous detection of more than 40 protein markers while preserving spatial information.

**Methods:** We established a kidney-specific IMC platform integrating panel design, antibody validation, metal conjugation, tissue staining, image acquisition, and analysis. A core panel was developed to capture renal architecture, cellular composition, and injury-associated states. Markers were selected to identify major nephron segments, glomerular, vascular, stromal, and immune compartments while simultaneously assessing tissue injury, repair, and immune infiltration. Standardised workflows were implemented for cell segmentation, phenotyping, and spatial analysis of human formalin-fixed paraffin-embedded kidney tissue.

**Results:** The newly developed platform enabled simultaneous characterisation of major renal compartments and cellular states in clinical biopsies obtained from patients with different clinical conditions, including acute kidney injury and allograft rejection. Renal epithelial, glomerular, vascular, stromal, and immune populations were robustly identified while preserving tissue architecture and spatial context. The resulting data supported comprehensive characterisation of kidney tissue at single-cell resolution and provided a stable reference framework for future biological investigations.

**Conclusions:** We established a kidney-specific IMC platform for high-dimensional spatial profiling of human kidney tissue. The development of this platform required extensive optimisation across experimental and analytical workflows but enables comprehensive characterisation of renal microenvironments from rare and valuable biopsy material. We developed a core panel to serve as a stable spatial reference for kidney tissue architecture, cellular composition, and injury-associated states while providing a foundation for future specialised panels targeting immune profiling, mitochondrial and metabolic adaptations, cellular senescence, fibroblast heterogeneity, and epithelial injury and repair. This platform creates new opportunities for translational nephrology research across diverse kidney diseases.

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## SO-11

### Failure to thrive and Hyponatremia in Infancy: Contrasting 2 rare salt-wasting tubulopathies

Mr. Julien Caccia[1], Dr. Sibylle Tschumi[1]

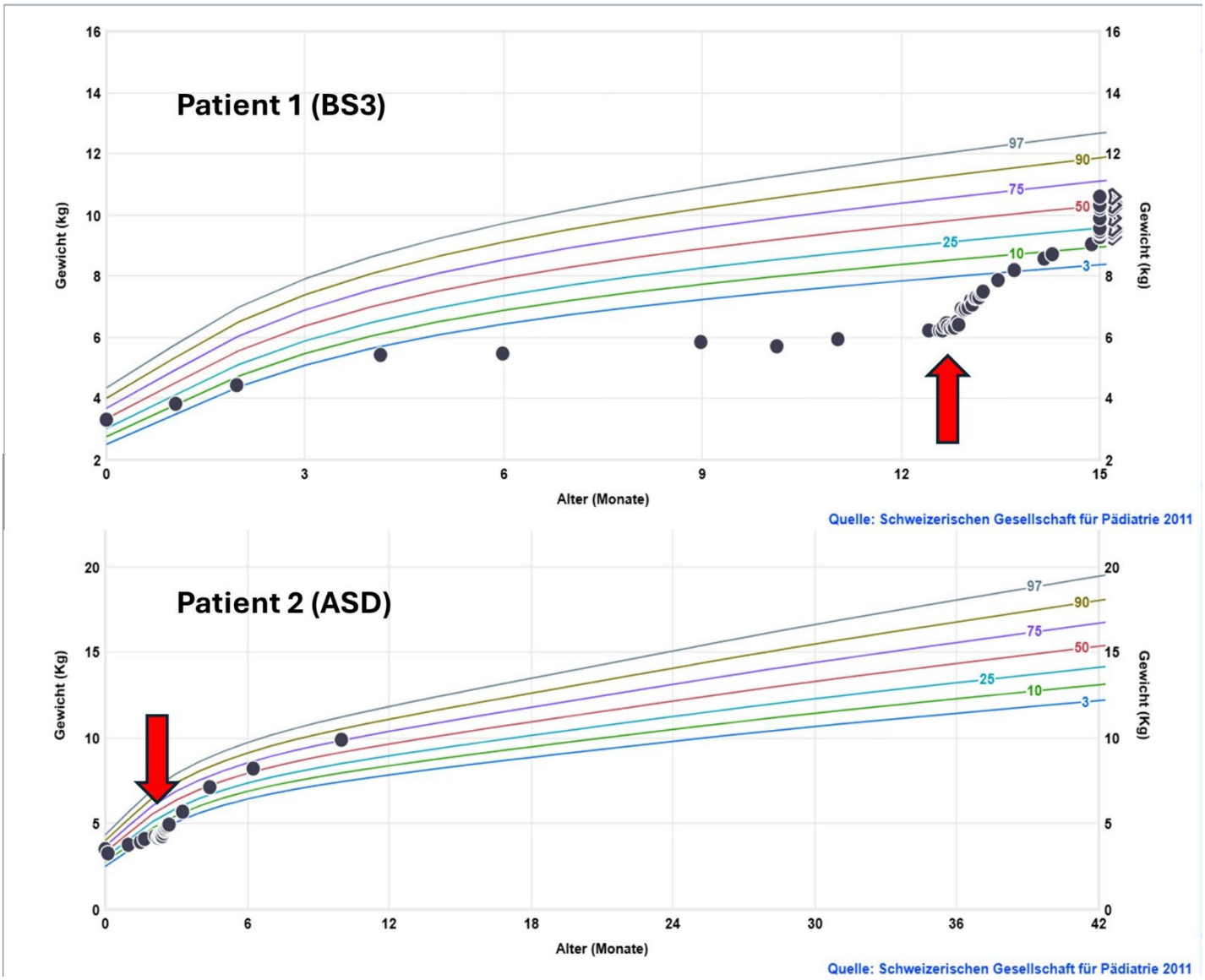
1. *pediatric nephrology, University Children's Hospital Bern, Inselspital*

Failure to thrive and electrolytes disturbances in infants encompasses a broad clinical range from renal tubulopathies to adrenal disorders among others. We report two male infants presenting with hyponatremia due to distinct salt-losing conditions.

**Patient 1.** A 12-month-old male presented with severe failure to thrive since he was 2–3 months old. Laboratories showed hyponatremia (Na 130 mmol/L), hypokalemia (K 2.1 mmol/L), hypochloremia, and metabolic alkalosis. Plasma renin was elevated, aldosterone was normal; blood pressure was normal. Genetic testing confirmed a coumpound heterozygous *CLCNKB* mutation (Bartter syndrome type 3). Treatment with salt supplementation and ibuprofen dramatically improved growth (fig. 1) and normalized electrolytes.

**Patient 2.** A 2-month-old male presented with failure to thrive, vomiting and dehydration. Laboratories showed hyponatremia (Na 125 mmol/L), normal to mild elevated Potassium (4.5-5.3 mmol/L), and normal blood gas analysis. Plasma renin was extremely elevated while aldosterone was normal; Blood pressure was normal. Genetic testing confirmed a homozygous *CYP11B2* mutation (aldosterone synthase deficiency). Treatment with salt supplementation and Fludrocortisone improved growth (fig. 1) and normalized electrolytes. Despite shared hyponatremia, contrasting potassium, renin-aldosterone and acid–base profiles guided the diagnosis which was confirmed by genetics. Treatment with salt supplements and targeted medications (Ibuprofen for BS3 or fludrocortisone for ASD) corrected the electrolytes disturbances and normalized weight gain. Prompt recognition of these rare salt-losing conditions is critical; untreated, they can cause life-threatening electrolyte imbalances, while appropriate therapy promotes normal development.

**Figure 1.** follow-up weight curve before and after initiation of therapy




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## SO-12

### GLP-1 Receptor Agonists Are Associated With Reduced Tolvaptan-Induced Polyuria in Autosomal Dominant Polycystic Kidney Disease

Dr. Matteo Bargagli[1], Dr. Simeon Schietzel[1], Prof. Daniel Fuster[2]

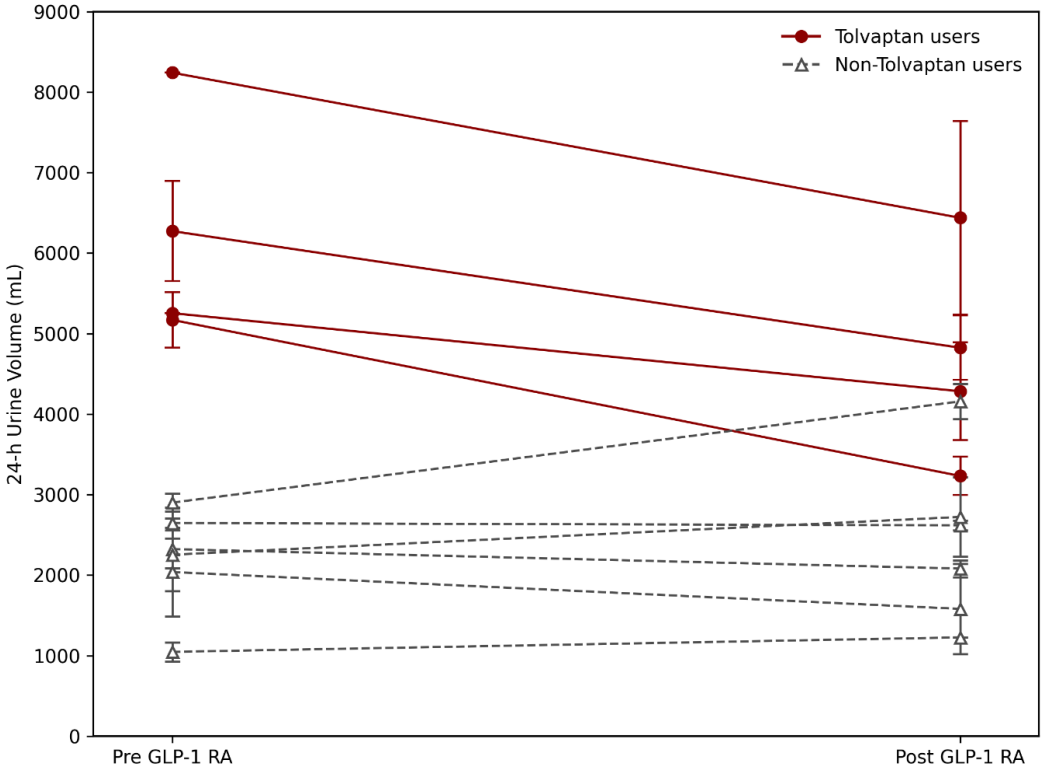
1. *Inselspital, Department of Nephrology, Bern, Switzerland, 2. Inselspital Bern*

**Background:** Tolvaptan is the only approved disease-modifying therapy for autosomal dominant polycystic kidney disease (ADPKD), but its use is frequently limited by aquaretic adverse effects. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) exert renal effects beyond appetite regulation, but their impact on water handling in ADPKD is unknown. We investigated changes in 24-hour urine volume after GLP-1 RA initiation in patients with ADPKD with and without concomitant tolvaptan treatment.

**Methods:** We analyzed patients initiating GLP-1 RA therapy within the prospective Bern ADPKD Registry. For each patient, mean values across available visits before and after GLP-1 RA initiation were calculated. Patients were stratified according to concomitant tolvaptan treatment, and within-patient changes in urinary parameters were compared between groups.

**Results:** Ten patients were included, of whom 4 received tolvaptan and 6 did not. Nine patients received semaglutide and 1 liraglutide. Following GLP-1 RA initiation, 24-hour urine volume decreased consistently in tolvaptan-treated patients, with a significantly greater change compared with patients not receiving tolvaptan (−1,540 ± 432 vs +196 ± 612 mL/day; P = 0.01). Urine osmolality increased in all tolvaptan-treated patients after GLP-1 RA initiation, while decreasing in 5 of 6 patients without tolvaptan. Free water clearance also decreased significantly more in tolvaptan-treated patients (P = 0.02). Changes in urinary sodium and urea excretion were inconsistent, arguing against reduced solute intake as the primary explanation.

**Conclusions:** GLP-1 RA initiation was associated with a pronounced antiaquaretic effect in ADPKD patients receiving tolvaptan, but not in those without V2-receptor blockade. These hypothesis-generating findings suggest a previously unrecognized interaction between GLP-1 receptor activation and tolvaptan-induced aquaresis and warrant mechanistic studies and prospective clinical trials.




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## SO-13

### Grafalon versus Thymoglobulin in Kidney Transplantation: A Retrospective Single-Center Cohort Study of Efficacy, Safety, and Graft Outcomes

Ms. Pabithra Pushparasa[1], Dr. Thomas Menter[2], Dr. Helmut Hopfer[2], Dr. Caroline Wehmeier[1], Dr. Jakob Brune[1], Prof. Michael Dickenmann[1], Prof. Pietro E Cippà[1], Dr. Marine Nachit[1], Prof. Stefan Schaub[1], Dr. Matthias Diebold[1]

1. *Clinic for Transplantation Immunology and Nephrology, University Hospital Basel, Basel, 2. Institute of pathology, University of Basel, Basel*

**Background**: Thymoglobulin® (Sanofi, France) and Grafalon® (Neovii Pharmaceuticals AG, Switzerland; formerly ATG-Fresenius®) are widely used as induction therapy in kidney transplantation. Comparative evidence on efficacy, safety, and graft outcomes in human leukocyte antigen (HLA) donor-specific antibody (DSA)-positive recipients remains limited.

**Methods**: We analyzed 92 adult HLA-DSA-positive recipients transplanted between 2014 and 2025 who received Grafalon (n = 50) or Thymoglobulin (n = 42). The primary endpoint was time to first biopsy-proven rejection within 12 months after transplantation. Secondary endpoints were microvascular inflammation, T-cell-mediated rejection and death-censored graft survival. Safety endpoints included cytomegalovirus and BK polyomavirus DNAemia, urinary decoy cell positivity and hematological side effects. Time-to-event outcomes were analyzed using Kaplan–Meier methods and longitudinal blood counts using linear mixed-effects models.

**Results**: During the first year, biopsy-proven rejection, graft survival, and infectious complications were similar between groups. Rejection-free survival showed no significant difference between groups (Hazard ratio 1.07, 95% confidence interval 0.43–2.67; *p = 0.878*), (Fig. 1). The median number of infections did not differ among the groups (2 [interquartile range (IQR) 1–2] vs. 2 [IQR 1–4]; *p = 0.209*), even after exclusion of lower urinary tract infections. During the first 30 postoperative days (Fig.2), lymphocyte counts were significantly lower with Thymoglobulin, although their trajectories did not significantly differ. In contrast, platelet and leukocyte counts showed significant treatment-by-time interactions, despite comparable mean levels. Hemoglobin trajectories differed significantly (*p<0.001*), with Grafalon patients requiring more red blood cell transfusions (3 [IQR 1–5] vs. 0 [IQR 0–3]; *p = 0.007*). Extending the analysis to six months, the difference in hemoglobin persisted until approximately day 60, after which the trajectories converged (Fig.3).

**Conclusion:** In HLA-DSA-positive kidney transplant recipients, Grafalon and Thymoglobulin showed comparable efficacy, safety, and graft outcomes during the first post-transplant year. Grafalon was associated with lower hemoglobin levels and a greater need for erythrocyte transfusion.

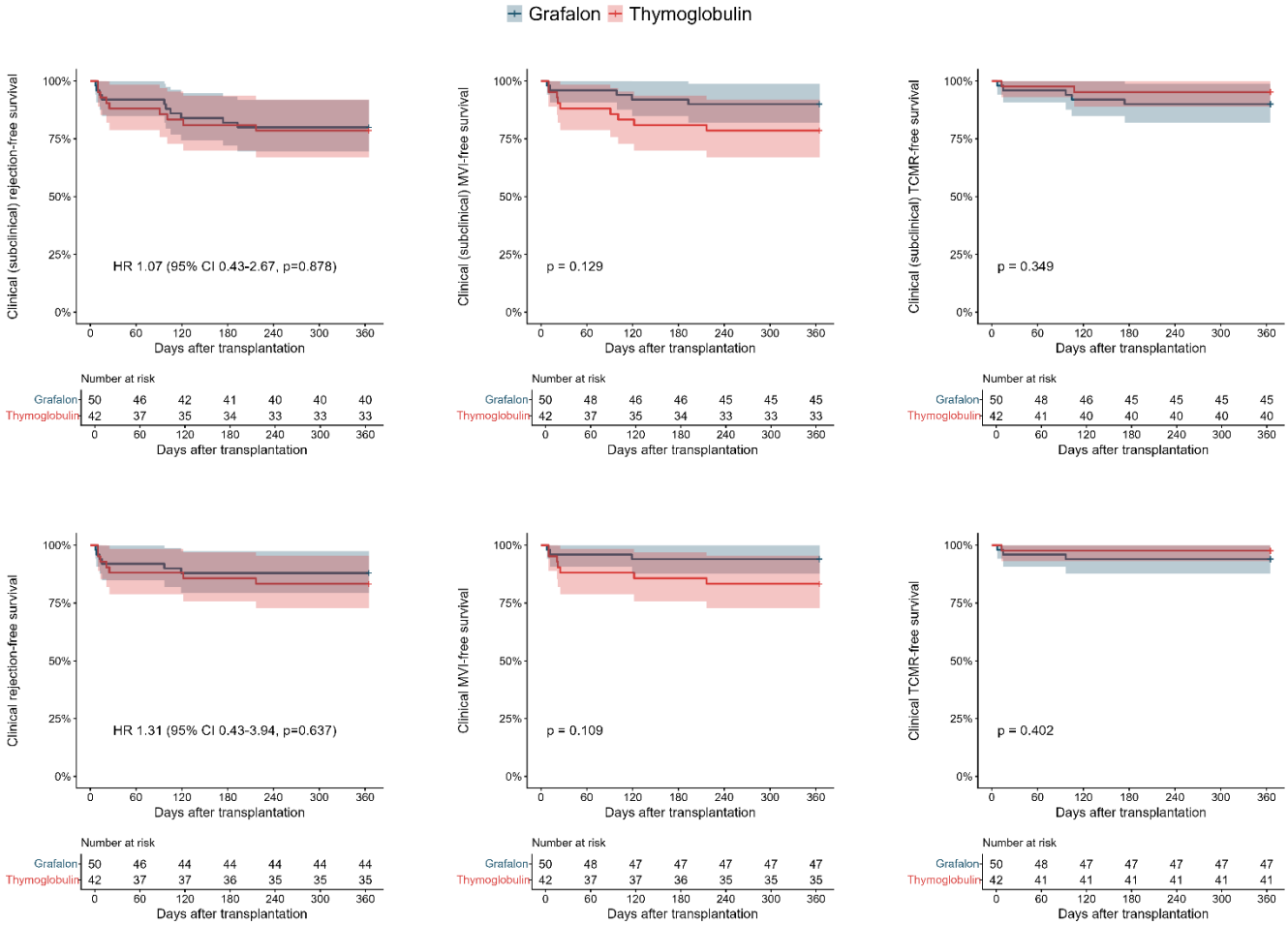



**Figure 1**


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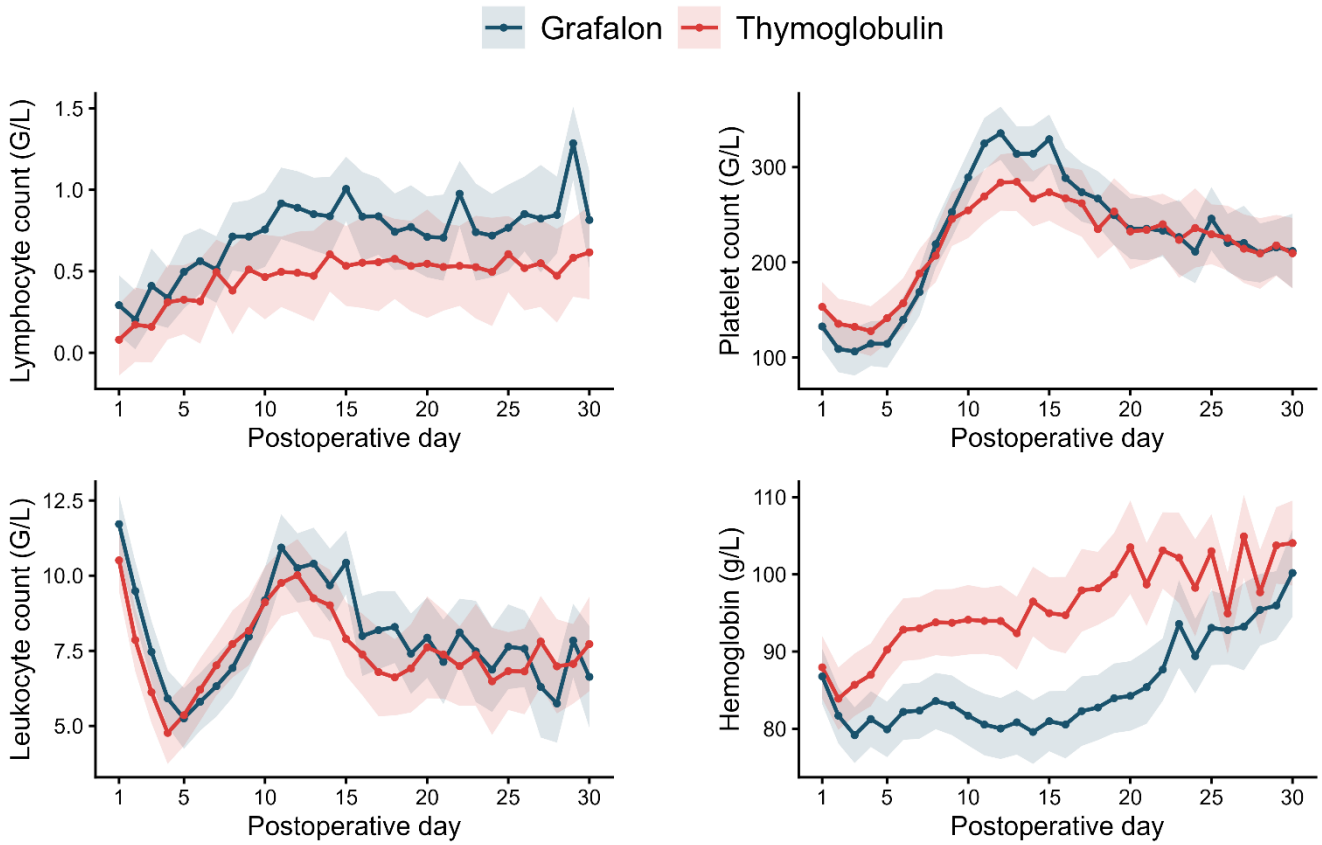



**Figure 2**

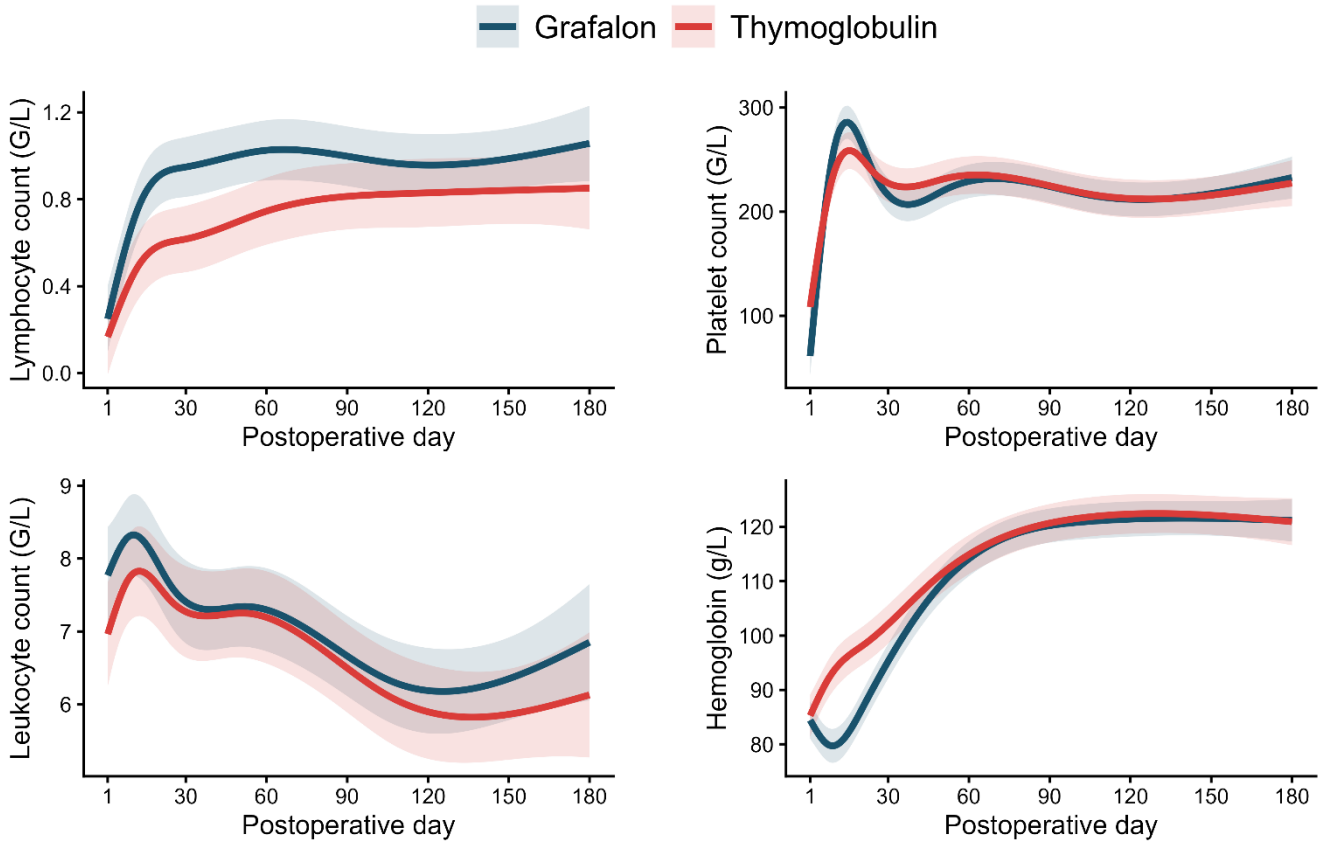



**Figure 3**


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## SO-14

### Immune cell identification in a routinely-processed kidney allograft biopsy using imaging mass cytometry

Dr. Marine Nachit[1], Dr. Nathalie Merkel[1], Dr. Cyril Carvalho[2], Ms. Manh-Nhi Gass[1], Dr. Anna Rinaldi[1], Prof. Pietro E Cippà[3], Dr. Matthias Diebold[3]

*1. Nephrology Laboratory, Department of Biomedicine, University of Basel and University Hospital Basel, 2. Nephrology Laboratory, Department of Biomedicine, University of Basel and University Hospital Basel, Switzerland, 3. Clinic for Transplantation Immunology and Nephrology, University Hospital Basel, Basel*

**Background:** Kidney allograft rejection is still a major issue in transplanted patients resulting in adverse graft outcomes and overall worse prognosis. Diagnosis is currently based on Banff classification relying on histopathological assessment that does not provide characterization nor spatial organization of implicated immune cells. The current diagnostic framework does not fully capture the underlying biological heterogeneity of kidney allograft rejection thus limiting the development of new mechanism-based therapies.

**Methods:** We established an immune panel including known myeloid and lymphoid lineages markers (CD3, CD20, CD11b, CD68, CD45 and others) as well as structural kidney markers. We used imaging mass cytometry (IMC) to analyze a kidney allograft biopsy obtained from a transplanted patient with suspected antibody-mediated rejection.

**Results:** The antibody panel enabled the detection of distinct immune cell types, including B cells, T cells, and macrophages, within the kidney tissue. These preliminary analyses allowed the spatial mapping of these populations, revealing their co-localization patterns and their enrichment next to specific renal structures.

**Conclusion:** This proof-of-concept study demonstrates that IMC can be applied to routinely processed human kidney allograft sections without specific pre-processing requirements beyond the usual workflow. This enabled characterization of immune cell phenotypes and their distribution within infiltrates. Studies in larger cohorts are now needed to determine whether IMC provides added value for the assessment of kidney allograft rejection.

## SO-15

### Impact of plasma-derived exosomes on B-cell activation and differentiation towards galactose-deficient IgA1 production in IgA nephropathy

Ms. Alica Cutkova[1], Mrs. Katerina Zachova[2], Prof. Milan Raska[2]

*1. Department of Immunology, Faculty of Medicine and Dentistry, Palacky University Olomouc, Olomouc, Czech Republic, 2. Department of Immunology, Faculty of Medicine and Dentistry, Palacky University Olomouc and University Hospital Olomouc, Olomouc, Czech Republic*

**Background:** IgA nephropathy (IgAN) is characterized by overproduction of galactose-deficient IgA1 (Gd-IgA1), its recognition by Gd-IgA1-specific autoantibodies, and subsequent formation of nephritogenic immune complexes. Exosomes mediate intercellular communication and may modulate immune-cell functions. This study investigated whether exosomes isolated from the blood (plasma) of patients with IgAN affect B-cell activation, differentiation, or Gd-IgA1 production.

**Methods:** Exosomes were isolated from IgAN patient's and healthy controls plasma by ultracentrifugation and were characterized by dynamic light scattering and flow cytometry (FC). Their uptake by the RD cell line (CCL-136) and RAW 264.7 cells was assessed by fluorescence microscopy. PBMCs from patients with IgAN (n = 5) and healthy controls (n = 5) were cultured for 7 days with autologous or heterologous exosomes. B-cell isotypes, CD38 expression, and λ light-chain surface expression were analyzed by FC. Concentrations of IgA, IgG, and Gd-IgA1 in cell culture supernatants were measured by ELISA.

**Results:** Isolated vesicles exhibited a size distribution consistent with exosomes and were efficiently internalized by RD and RAW cells. Exposure of PBMCs to exosomes did not significantly alter the proportions of $IgA^+$, $IgM^+$, $IgG^+$, or Gd-$IgA1^+$ B cells and had no consistent effect on CD38 or λ light-chain expression. Similarly, no significant differences in IgA, IgG, or Gd-IgA1 secretion were detected.

**Conclusions:** The inability of circulating plasma-derived exosomes from patients with IgAN to modulate peripheral Gd-$IgA1^+$ B-cell activation, differentiation, or Gd-IgA1 secretion indicates that systemic exosomes are not major drivers of Gd-IgA1 production. These findings support the concept that Gd-IgA1 production is primarily determined by genetic background and the local mucosal immune microenvironment, as well as by its activation in response to various inflammatory or infectious stimuli. Our findings do not exclude the possibility that exosomes may modulate other mechanisms involved in IgAN pathogenesis.

## SO-16

### Incidence of human papillomavirus-related cancers in solid organ transplant recipients: a retrospective cohort study in Geneva

Dr. Nathalie Hammer[1], Dr. Luca Pesenti[2], Dr. Manuela Undurraga[2], Dr. Fadi Haidar[1]

1. *Division of Nephrology, Geneva University Hospitals, 2. Division of Gynaecology, Geneva University Hospitals*

**Background:** Solid organ transplant recipients (SOTR) have an increased risk of human papillomavirus (HPV)-related malignancies due to chronic immunosuppression. However, epidemiological data in Switzerland remain limited, particularly for non-gynaecological HPV-related cancers. We aimed to characterize the incidence of these cancers in adult abdominal SOTR in our center.

**Methods:** We conducted an STCS-endorsed monocentric retrospective cohort study including adult recipients of kidney, liver, pancreas, pancreatic islet, or combined transplants performed at Geneva University Hospitals between May 2008 and March 2025. STCS data were cross-referenced with electronic medical records to identify HPV-related cancers, precancerous lesions, HPV testing, and vaccination status. The primary outcome was the 10-year cumulative incidence of HPV-related cancer, estimated using competing-risk analysis.

**Results:** Among 1,086 SOT recipients, 94 possible HPV-related malignancies were identified. After diagnostic review, 7 post-transplant cancers remained (2 molecularly confirmed, 5 probable), including 6 in liver recipients. Cancers involved ENT, penile, and uterine sites, with a median time to diagnosis of 3.4 years (IQR 1.6–6.9) (Figure 1). The 10-year cumulative incidence was 0.95% (95% CI 0.42–1.93), corresponding to 116 (95% CI 55–243) cases per 100,000 person-years. Mortality did not differ by transplant type (log-rank $\chi^2 = 2.4$, $p = 0.70$; Figure 2a), whereas the composite endpoint of death and graft loss differed significantly (log-rank $\chi^2 = 15.2$, $p = 0.004$; Figure 2b), with graft loss contributing substantially to the overall event burden.

**Conclusion:** This explorative study provides the first epidemiological characterisation of HPV-related cancers in Swiss SOTR, showing an incidence approximately 20-fold higher than the general population, and highlights the need for improved HPV genotyping, vaccination records, and surveillance protocols warranting a multidisciplinary post-transplant approach and multicentre prospective studies.

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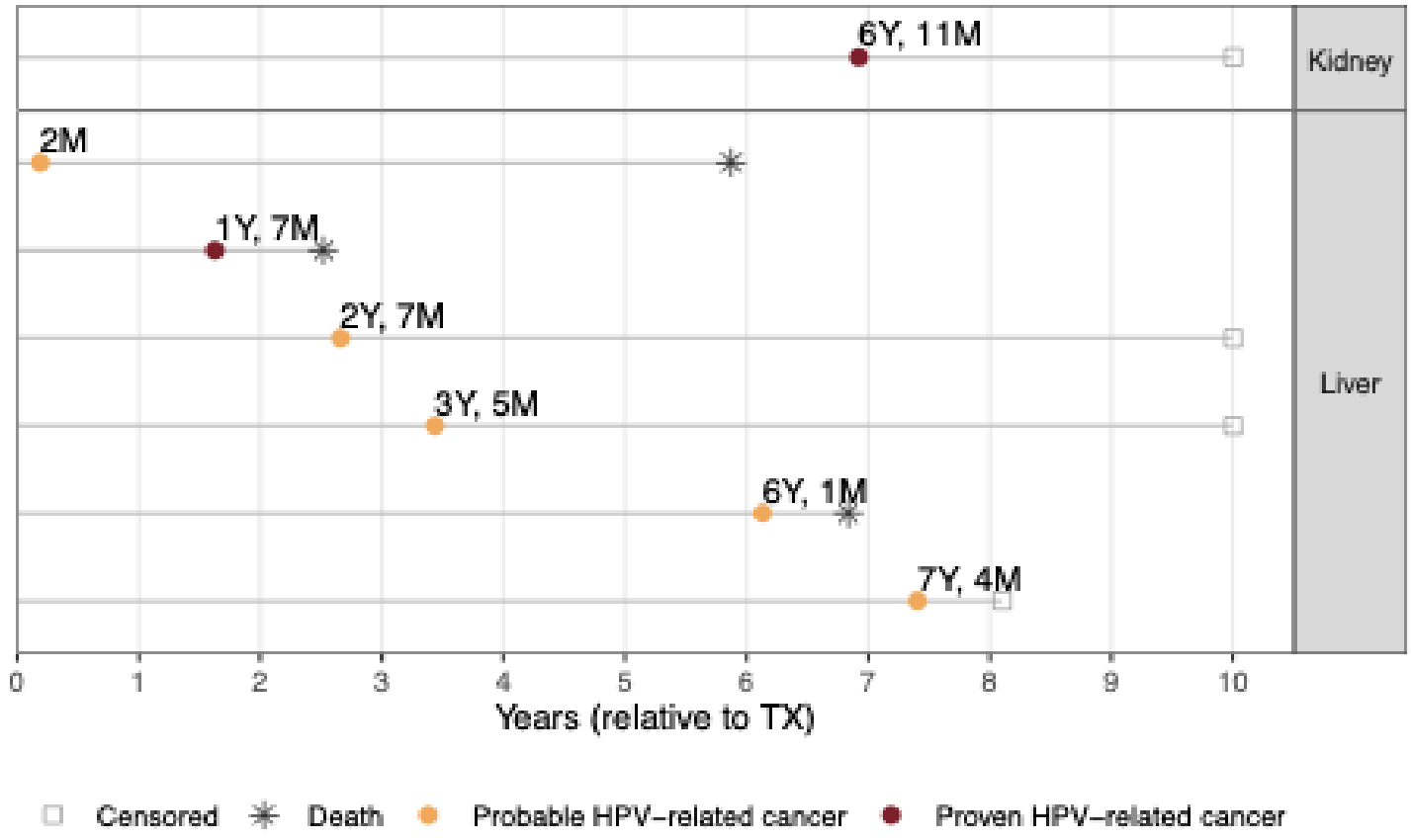



**Figure 1.** Incidence of HPV-related cancers

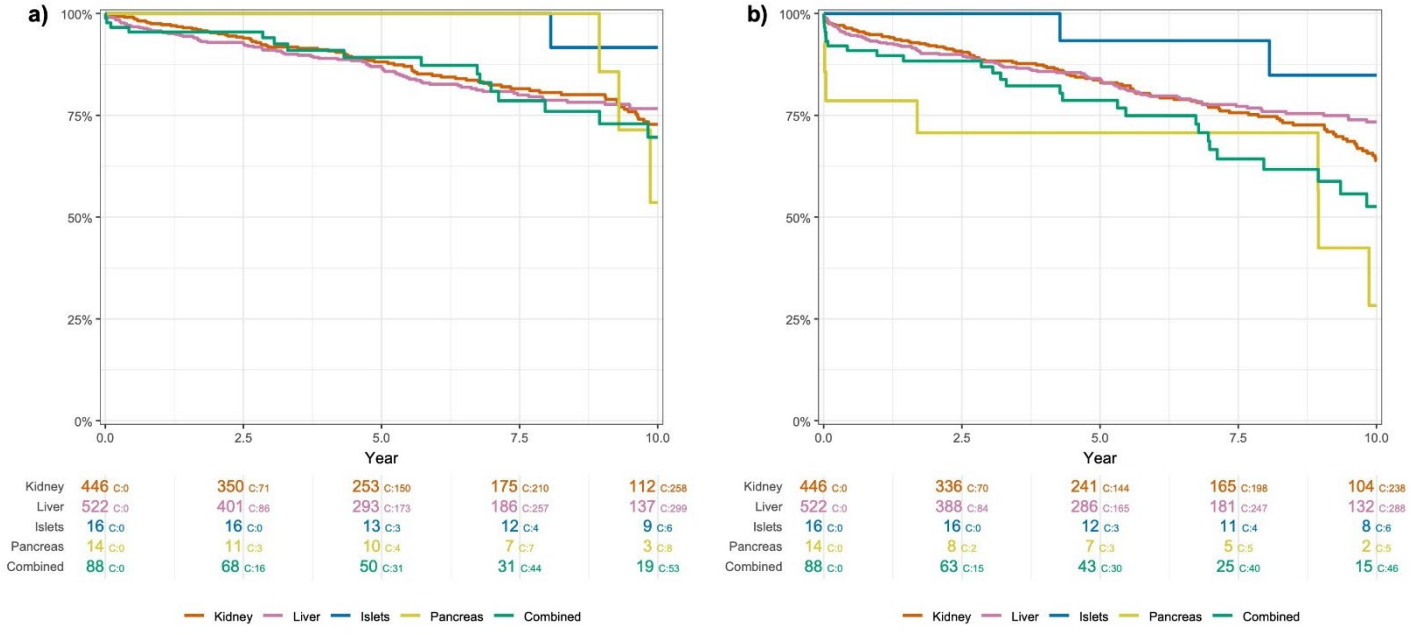



**Figure 2.** Kaplan-Meier survival analysis among organ transplant types with corresponding censored data. Overall mortality is depicted in a) and overall mortality and graft loss in b).


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## SO-17

### Indapamide and Chlorthalidone to Reduce Urine Supersaturation for Secondary Prevention of Kidney Stones: a Randomized, Double-blind, Crossover Trial (INDAPACHLOR Trial)

Dr. Martin Scoglio[1], Dr. Matteo Bargagli[1], Prof. Daniel Fuster[2]

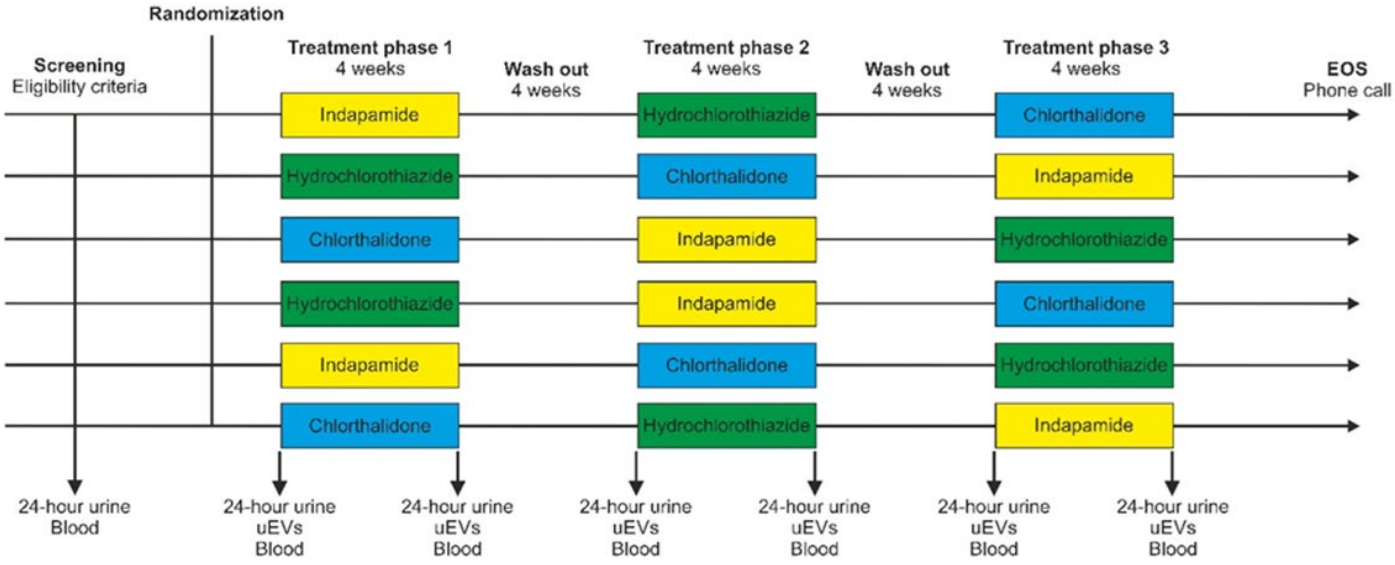
1. *Department of Nephrology, Inselspital, Bern University Hospital, University of Bern, Bern, Switzerland, 2. Inselspital Bern*

**Background:** Kidney stones constitute a major global healthcare problem. The majority of kidney stones consist of calcium and a high urine calcium is the predominant metabolic abnormality in these patients. The NOSTONE trial demonstrated that hydrochlorothiazide (HCT) has limited efficacy in preventing recurrence. Our objective is to assess whether indapamide or chlorthalidone are more effective than HCT in reducing urine supersaturations (RSRs) for calcium oxalate and calcium phosphate, which are validated predictors of stone recurrence.

**Methods:** INDAPACHLOR is a single-center, randomized, double-blind, cross-over trial evaluating the efficacy of indapamide or chlorthalidone compared to hydrochlorothiazide in lowering urine RSRs for calcium oxalate and calcium phosphate in individuals with idiopathic calcium kidney stones. Participants will be allocated to indapamide 2.5 mg once daily, chlorthalidone 25 mg once daily, and hydrochlorothiazide 50 mg once daily in a random sequence. The three consecutive active treatment periods of 28 days each will be separated by wash-out periods of 28 days. The primary outcomes are the changes of the RSR calcium oxalate and RSR calcium phosphate from baseline to day 28 of each treatment period. The study targets enrollment of 99 participants to achieve 80% power for detecting a 20% reduction in RSR calcium oxalate or calcium phosphate when treated with indapamide or chlorthalidone compared to HCT.

**Results:** The INDAPACHLOR trial is registered at ClinicalTrials.gov (NCT06111885). Approval of the cantonal Ethics Commission Bern, Switzerland was obtained on May 28 2024 (approval # 2024_00477), and authorization from the Competent Authority Swissmedic was received on June 27 2024 (approval # 701824). Patient recruitment commenced in December 2024. As of end-August 2026, a total of 96 patients have been recruited. Of these, 70 participants already completed the study.

**Conclusion:** No conclusions available (study in progress).




Study summary

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| TABLE 2: Inclusion and Exclusion Criteria | |
|---|---|
| **Inclusion Criteria** | |
| Patients fulfilling all the following inclusion criteria are eligible for the study: | |
| 1 | Written, informed consent |
| 2 | Age 18 years or older |
| 3 | Recurrent kidney stone disease (2 or more stone episodes in the last 10 years prior to randomization) |
| 4 | Past kidney stone containing 50% or more of calcium oxalate, calcium phosphate or a mixture of both |
| **Exclusion Criteria** | |
| The presence of any one of the following exclusion criteria will lead to exclusion: | |
| 1 | Patients with secondary causes of recurrent calcium kidney stones including severe eating disorders (anorexia or bulimia), chronic bowel disease, intestinal or bariatric surgery, sarcoidosis, primary hyperparathyroidism, chronic urinary tract infection |
| 2 | Patients taking the following medications: Thiazide or loop diuretics, carbonic anhydrase inhibitors (including topiramate), xanthine oxidase inhibitors, alkali, active vitamin D (calcitriol or similar), calcium supplementation, bisphosphonates, denusomab, teriparatide, sodium-glucose co-transporter 2 (SGLT2) inhibitors, strong CYP3A4 inhibitors or inducers (may affect indapamide metabolism), lithium. |
| 3 | Patients with chronic kidney disease (defined as CKD-EPI eGFR < 30ml/min) |
| 4 | Patients with glomerulonephritis |
| 5 | Patients with the following biochemical imbalances: severe hypercalcemia (>2.8 mmol/L), therapy-resistant hypokalemia or conditions with increased potassium loss, severe hyponatremia (<130 mmol/L), symptomatic hyperuricemia |
| 6 | Patients with hepatic encephalopathy or severe liver insufficiency |
| 7 | Patients with severe cardiac insufficiency |
| 8 | Patient with a recent cerebrovascular event |
| 9 | Patients with a solid organ transplant |
| 10 | Pregnant and lactating women (A urine pregnancy test must be performed for women of child-bearing potential, defined as women who are not surgically sterilized/hysterectomized, and/or who are postmenopausal for less than 12 months) |
| 11 | Previous (within 3 months prior to randomization) or concomitant participation in another interventional clinical trial |
| 12 | Previous participation in the INDAPACHLOR Trial |
| 13 | Inability to understand and follow the protocol |
| 14 | Allergy to any of the study drugs |

Inclusion and exclusion criteria

## SO-18

### Kidney Allograft Proteomics Reveals Shared Inflammatory and Distinct T Cell– and Antibody-Mediated Rejection Signatures

Dr. Matthieu Halfon[1], Dr. Maxime Jan[2], Dr. Dusan Harmacek[3], Dr. Mélanie Metrich[4], Prof. Olivier Bonny[1], Dr. Susanne Dettwiler[5], Dr. Birgit Maria Helmchen[5], Prof. Manuel Pascual[6], Prof. Fadi Fakhouri[7], Prof. Dela Golshayan[7], Prof. Manfredo Quadroni[8]

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**Background:** T cell–mediated rejection (TCMR) and antibody-mediated rejection (ABMR) are major causes of kidney allograft injury. Although histologically distinct, their molecular overlap and specific intragraft proteomic features remain incompletely characterized. We aimed to define and compare the proteomic signatures associated with TCMR and ABMR.

**Methods:** Label-free quantitative mass spectrometry was performed on kidney allograft biopsies from recipients with TCMR, ABMR, or without rejection. Differential protein abundance was assessed using DEqMS. Proteins were considered significantly altered when the adjusted *P* value was <0.05 and the absolute log2 fold change was >1.

**Results:** Compared with non-rejection controls, 428 proteins were differentially abundant in TCMR, including 214 increased and 214 decreased proteins. In ABMR, 193 proteins were differentially abundant, including 98 increased and 95 decreased proteins. Seventy-five increased proteins were shared between TCMR and ABMR, indicating a common intragraft inflammatory response. TCMR showed a broader immune signature, characterized by enrichment of phagosome, natural killer cell–mediated cytotoxicity, antigen processing and presentation, and leukocyte transendothelial migration pathways. ABMR also demonstrated antigen-presentation, cytotoxic and myeloid inflammatory features but was distinguished by a more prominent humoral signature involving complement and coagulation pathways, immunoglobulin-related proteins, C3, C4B and JCHAIN. Decreased proteins in both rejection phenotypes were predominantly involved in small-molecule, amino-acid, carbon and energy metabolism, consistent with loss or dysfunction of kidney tubular metabolic functions.

**Conclusions:** TCMR and ABMR share a substantial intragraft inflammatory proteomic core but display different dominant bio-

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logical patterns. TCMR is characterized by a broader cell-mediated immune response, whereas ABMR exhibits a more prominent complement and humoral signature. Both rejection phenotypes are accompanied by suppression of renal metabolic pathways, highlighting the combination of immune activation and parenchymal dysfunction in kidney allograft rejection.

## SO-19

### Less pain, same answers? Diagnostic agreement and patient experience with capillary versus venous blood sampling in children

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**Background:** Venous puncture (VP), the gold standard of blood sampling, is a common but distressing procedure in children. Less invasive blood sampling methods like capillary blood sampling (CBS) could reduce procedure-related pain and anxiety. This may be particularly relevant for children requiring repeated blood testing, including those with chronic kidney disease, in whom preservation of peripheral veins is important for potential future vascular access. This study evaluates the diagnostic concordance of CBS and VP for routine nephrology parameters and compares associated pain and anxiety.

**Methods:** In this prospective single-center cross-sectional study at the University Children's Hospital Basel (UKBB),

100 children (0–18 years) requiring clinically indicated blood sampling or venous access were enrolled. Paired capillary and venous samples were collected simultaneously. Agreement between sampling methods was assessed using Pearson's correlation coefficient, Lin's concordance correlation coefficient (CCC), and Bland–Altman analysis. In a subgroup of 50 patients, standardized questionnaires assessed pain and anxiety.

**Results:** The median age was 13.5 years (IQR 9.9–15.3). Creatinine demonstrated the highest concordance between sampling methods (CCC = 0.97; 95% CI, 0.96–0.98), followed by cystatin C (CCC = 0.83; 95% CI, 0.77–0.88), whereas concordance was more variable for electrolytes and glucose (CCC range, 0.24–0.56). Pain and anxiety scores were significantly lower with CBS than VP (p <0.001 each).

**Conclusions:** CBS provides strong diagnostic agreement with VP for creatinine while significantly reducing pain and anxiety. These findings support CBS as a feasible, patient-centered alternative to VP for selected laboratory assessments, particularly in children requiring frequent blood sampling or preservation of venous access.

## SO-20

### Long-term effects of palopegteriparatide on bmd and tbs in adults with chronic hypoparathyroidism: pooled results from the path forward and pathway trials

Dr. Karin Senn[1]

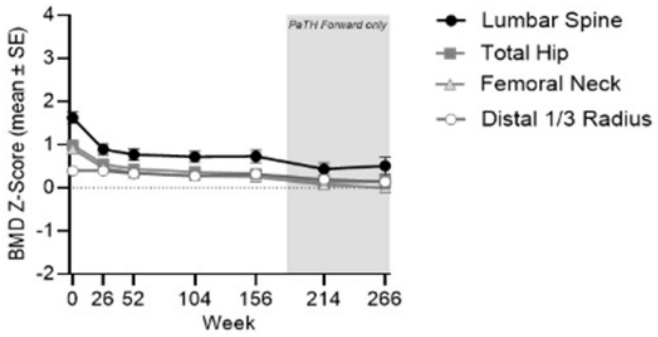
1. *Ascendis Pharma Switzerland GmbH*

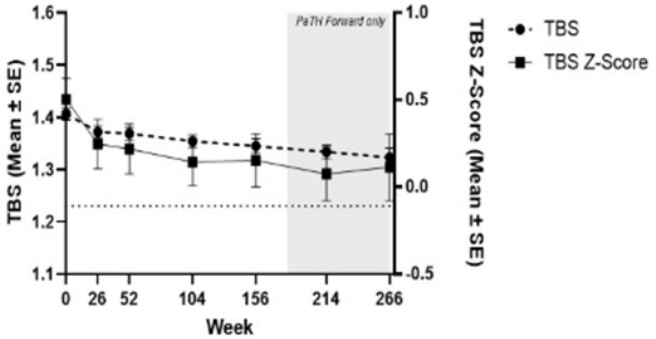
**Background:** The phase 2 PaTH Forward and phase 3 PaTHway trials demonstrated safety and efficacy of palopegteriparatide in chronic hypoparathyroidism for up to 5 years. This post-hoc, pooled analysis reports long-term changes in bone mineral density (BMD).

**Methods:** Data were from PaTH Forward and PaTHway, comprising randomized, double-blind, placebo-controlled periods through Weeks 4 and 26 and open-label extension (OLE) periods through Weeks 266 and 182, respectively. Bone mass and microarchitecture were assessed by DXA and trabecular bone score (TBS). TBS was assessed using the TBS iNsight algorithm. Safety included treatment-emergent adverse events.

**Results:** At baseline, participants (N = 139) had a median (range) hypoparathyroidism duration of 9 (1-56) years and mean (SD) age of 49 (12), 80% were female (of which 35% were postmenopausal), and 129 completed their respective trials. Elevated baseline mean BMD T- and Z-scores declined and remained above zero through the OLE. Lower baseline T- and Z-scores were generally associated with smaller BMD declines over time. Longitudinal changes in DXA BMD were consistent with TBS and TBS Z-scores. Mean TBS Z-scores reflected normal trabecular microarchitecture ≥1.310 through Week 266. Magnitude of TBS Z-score change over time was generally smaller among participants with lower baseline TBS Z-scores. Fractures were uncommon and deemed unrelated to palopegteriparatide. Two participants (1.4%) had vertebral fracture events.

**Conclusions:** Treatment with palopegteriparatide led to early normalization of elevated baseline BMD and TBS, which was sustained. This suggests, over a 5-year period, palopegteriparatide normalized bone mass and microarchitecture consistent with age- and sex-matched norms.




TBS values reflect trabecular microarchitecture that is normal (≥ 1.310), partially degraded (1.230 to 1.310), or degraded (≤ 1.230)
Shevroja E, et al. Update on the clinical use of trabecular bone score (TBS) in the management of osteoporosis. *Osteoporos Int.* 2023 Sep;34(9):1501-1529


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## SO-21

### Mechanisms and signatures of immune tolerance following combined kidney and hematopoietic stem cell transplantation

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Transplantation tolerance has clinically been achieved by combined kidney and hematopoietic stem cell transplantation (CKHSCT) from the same donor, leading to a state of transient or persistent hematological chimerism. The SWISS–TOLERAN–CE.CH study has successfully established such a protocol in Switzerland, enabling immunosuppression withdrawal in human leukocyte antigen (HLA)-matched transplants. However, the immunological mechanisms and biomarkers underlying transplant tolerance remain unclear. We hypothesis that distinct central and peripheral immunological tolerance mechanisms operate over the course of immune reconstitution, modulated by donor-recipient interactions.

During the SWISSTOLERANCE.CH a biobank was generated with peripheral blood mononuclear cells before, 6 months and 12 months and at long term follow up time points after transplantation was generated. These samples were analyzed using high-dimensional spectral flow cytometry and single-cell RNA sequencing (scRNA-seq) with feature barcoding.

All patients remained tolerant with a functional kidney graft up to 10 years post-transplantation, despite loss of macrochimerism in 4 patients (Fig. 1). Flow cytometry revealed early depletion of adaptive immune cells with distinct temporal and phenotypic reconstitution dynamics (Fig. 2A-C): notably the T cell compartment shifted to distinct memory phenotypes, whereas the B cell compartment post-transplant was dominated by a naïve/transitional phenotype (Fig. 2D-F). In our scRNA-seq analysis, we refined this analysis by separating donor and recipient cells based on single-nucleotide polymorphisms (Fig. 2G,H). We are currently investigating the transcriptional and clonal dynamics associated with tolerance acquisition in this dataset.

CKHSCT induces profound, long-term immunological changes with implications for alloreactive and infectious immune responses. The observed dynamics, including expansion of transitional B cells, suggest a role for additional regulatory mechanisms that we are further investigating and extending to a deeper recipient-donor resolution. A better understanding of the immunological mechanisms underlying transplantation tolerance is essential to improve clinical protocols, help biomarker discovery and allow translation of these protocols into HLA-mismatched individuals.

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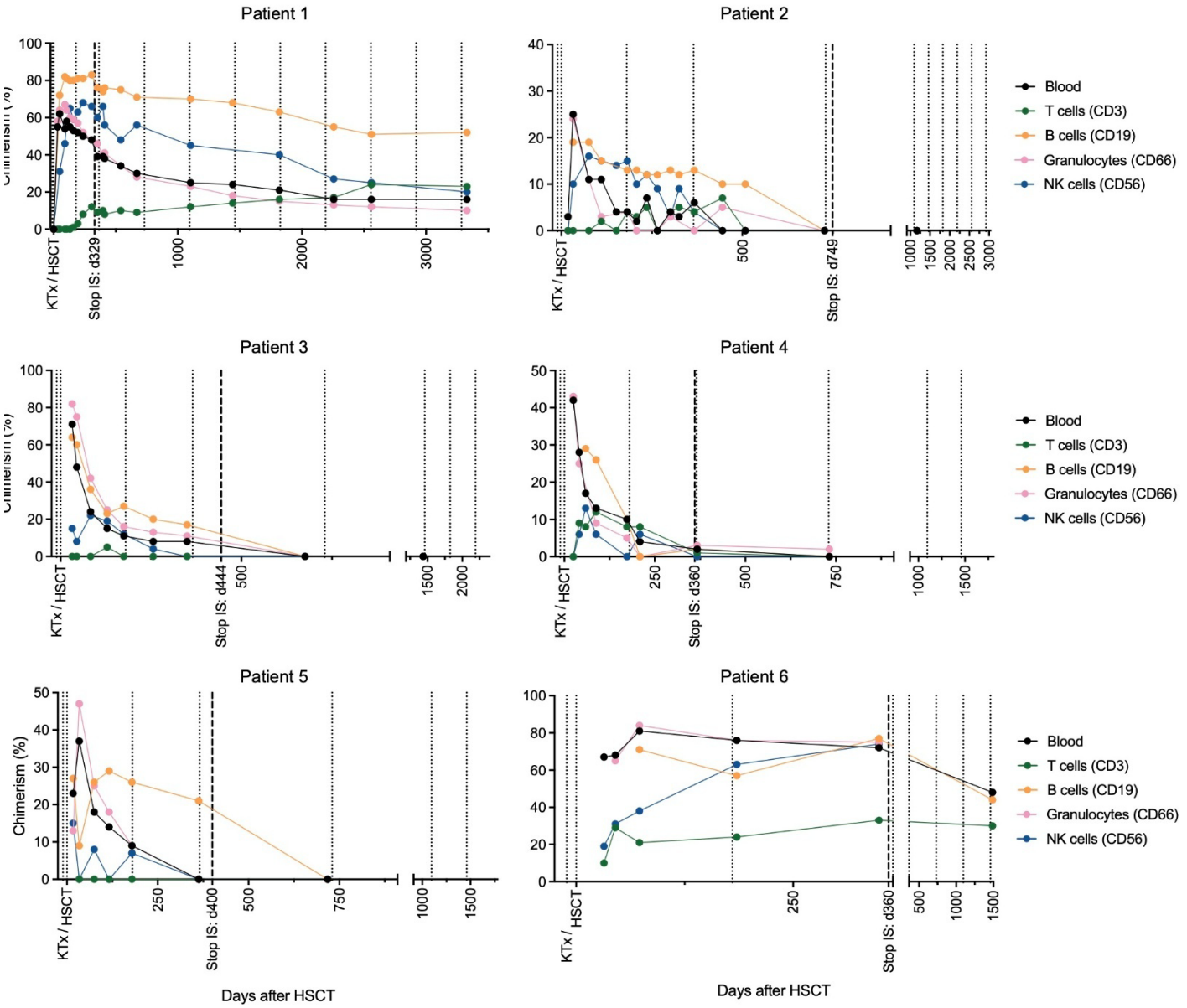



**Figure 1 – Donor Chimerism over time.**
Whole blood (black), as well as lineage-specific (CD3+ green, CD19+ orange, CD56+ blue, CD66+ pink) donor chimerism is shown over time after HSCT for each patient. Dotted lines indicate time intervals of 6 and 12 months post-transplant and yearly thereafter.
IS – immune suppression. KTx – Kidney Transplantation, HSCT – hematopoietic stem cell transplantation.


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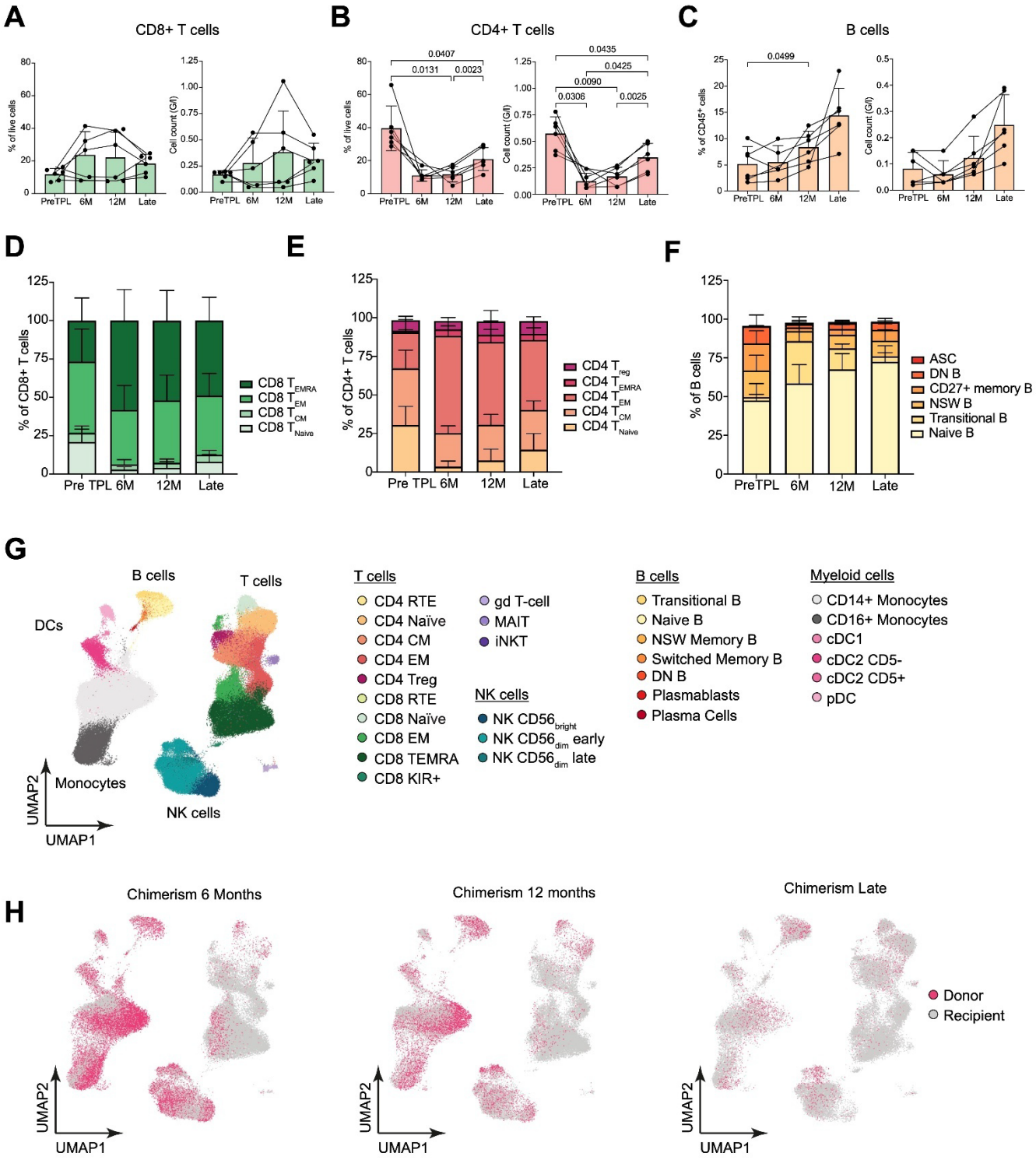



**Figure 2 – Immune reconstitution following CKHSCT and tolerance acquisition by flow cytometry and scRNA-seq analysis**
(**A-C**) Frequencies and absolute cell counts of CD8+ T cells (A), CD4+ T cells (B) and B cells (C) in peripheral blood before (PreTPL), 6 months (6M) after, 12 months (12M) after and several years after CKHSCT (Late) as determined by flow cytometry.
(**D-F**) Stacked bar graphs showing the composition of the circulating CD8+ T cell (D), CD4+ T cell (E) and B cell (F) compartment at the indicated time points.
(**G**) Uniform Manifold Approximation and Projection (UMAP) plot showing the main immune cell subset in scRNA-seq with feature barcoding data from sorted CD45+ peripheral blood cells from all patients before, 6 months, 12 months and several years after CKHSCT (Late).
Cell subsets were identified using a Louvain-Clustering and supervised marker and TCR analysis with manual annotation.
(**H**) UMAP showing the recipient (grey) and donor (red) cells at the indicated time points (6 months, 12 months and several years after CKHSCT (Late)). Cell origin was determined using single nucleotide polymorphism (SNP) calling.
In A-C statistical testing was performed using a mixed-effect model with a Tukey multiple comparison testing. Shown are statistically significant ($p<0.05$) adjusted p-values.


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## SO-22

### Model-based meta-analysis to inform chronic kidney disease drug development

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1. *Clinical Pharmacology Modelling and Simulation, GSK, 2. Certara USA, Inc., Princeton NJ*

**Background:** Chronic kidney disease (CKD) represents a major unmet medical need, with heterogeneous etiologies and severities that demand tailored therapeutic strategies. This study uses the CODEx CKD Database and model-based meta-analysis (MBMA) to quantify the relationship between early surrogate endpoints, particularly urinary albumin-to-creatinine ratio (UACR), and long-term composite renal outcomes, to inform clinical trial design and patient stratification.

**Methods:** The database was developed using systematic review methods aligned with the Cochrane Handbook and PRISMA guidelines. Average-level safety and efficacy data from randomized controlled trials of ACE inhibitors, angiotensin receptor blockers, SGLT2 inhibitors and endothelin receptor antagonists were extracted. MBMA was applied to characterize the association between 6-month UACR changes and composite clinical endpoints at 1–2 years, including progression to end-stage renal disease and renal death, while accounting for inter-study heterogeneity in CKD etiology and severity.

**Results:** Renal function-related endpoints were harmonized and converted to UACR after unit adjustments, and clinical endpoints were standardized by estimating hazard ratios for active versus control arms. Analyses showed that early reductions in UACR at 6 months were positively associated with improved clinical outcomes at 12 months. A 30% reduction in the geometric mean ratio of UACR fold change in the treatment versus control arm corresponded to a 14% decrease in the hazard of the composite clinical endpoint. These findings support UACR as a meaningful endpoint for CKD drug development, enabling futility analyses, adaptive trial designs and earlier Go/No-Go decisions with smaller sample sizes. The results are consistent with previous studies while addressing limitations by relying on harmonized published aggregate data.

**Conclusions:** Integrating the CODEx CKD Database with MBMA provides a framework to evaluate surrogate endpoint relationships in CKD, and early insights into UACR–clinical outcome associations may enhance trial efficiency and support development of targeted therapies for CKD and its subtypes.

## SO-23

### Nearly 20 years on peritoneal dialysis: potential factors associated with long-term technique survival – a clinical case

Dr. Baiba Adamaite[1], Dr. Ināra Bušmane[2], Dr. Anda Grigāne[2], Prof. Jūlija Voicehovska[2], Dr. Eva Bormane[2], Dr. Kaspars Kivlenieks[2], Dr. Santa Līga Starostniece[2], Dr. Lana Zeltiņa[1], Dr. Dace Trumpika[3]

*1. Kidney diseases and Renal replacement clinics,Latvian renal and multiorgan replacement treatment association., 2. Kidney diseases and Renal replacement clinics, Latvian renal and multiorgan replacement treatment association, 3. Kidney diseases and Renal replacement clinics, Latvian renal and multiorgan replacement treatment association.*

**Background:** Peritoneal dialysis (PD) is an effective kidney replacement therapy (KRT) modality for patients with kidney failure. It enables home-based treatment, helps preserve residual kidney function, and allows patients to maintain professional and social activities. The mean duration of PD treatment is approximately 3.5–3.9 years. Cases in which a patient successfully remains on PD for 20 years or longer are rare.

**Methods and results:** This is a rare clinical case report about a young patient with traumatic spinal cord injury at Th7–Th9 level developed bladder atony, bilateral hydronephrosis, obstructive nephropathy, and stage 5 chronic kidney disease. PD was initiated in 2006, initially as continuous ambulatory PD and subsequently converted to automated PD in 2012 because of fluid overload. During 20 years of PD, only one episode of peritonitis occurred. Residual urine output remained approximately 1000 mL/day for many years but declined to 400–500 mL/day from 2024. Severe secondary hyperparathyroidism (PTH 2355 pg/mL) was treated with bilateral parathyroidectomy in 2024, resulting in sustained normalization of PTH, calcium, and phosphate levels. Progressive heart failure with marked volume overload, pleural and pericardial effusions, and pulmonary congestion developed in 2024–2025. Despite intensification of APD, signs of inadequate dialysis persisted. An AVF was created in July 2025, and intermittent hemodialysis was initiated in January 2026. Heart failure subsequently progressed, requiring repeated thoracenteses and pleural drainage.

**Conclusions:** Peritoneal dialysis (PD) can remain an effective KRT modality for a very prolonged period. Long-term success may be facilitated by young age at initiation, absence of diabetes, preserved residual kidney function, only one episode of peritonitis, and good patient adherence. Individualized PD prescription adjustments enabled continued treatment despite declining residual kidney function. Ultimately, progressive loss of dialysis adequacy and heart failure necessitated transition to intermittent hemodialysis, highlighting the importance of individualized care and long-term follow-up.

## SO-24

### Oral Potassium Supplementation and Clinical Outcomes in Patients with Cardiorenal Syndrome: A Retrospective Cohort Study

Dr. Elena Rho[1], Dr. Christian Martinetti[2], Dr. Thomas Welte[1], Prof. Britta George[3], Prof. Thomas Schachtner[1], Dr. Lukas Weidmann[1], Prof. Frank Ruschitzka[4], Dr. Michelle Frank[4], Dr. Dusan Harmacek[1]

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**Background:** Serum potassium follows a U-shaped relationship with mortality in cardiorenal syndrome (CRS). Evidence and recommendations on oral potassium supplementation for diuretic-associated hypokalemia in a genuinely cardiorenal population is comparatively sparse.

**Methods:** We screened 551 patients who from 2015 to 2022 had been seen at least once in the nephrology and in the cardiology outpatient clinics with concomitant chronic kidney disease (CKD) and heart failure (HF), resulting in a cohort of 223 patients. Characteristics related to type and cause of renal and cardiac disease, therapy with a specific focus on K supplementation and diuretic use, incidence of hospitalisation for decompensated heart failure (HF) with acute kidney injury (AKI), eGFR-Slope and mortality were recorded.

**Results:** Oral potassium supplementation was present in 50/223, 22% patients. Supplemented patients had more atrial fibrillation (66% vs 44.5%, p = 0.007) and markedly higher loop- (94% vs. 69.4%, p <0.001) and thiazide-diuretic (28% vs. 5.2%, p<0.001) use at baseline. Over a median follow-up of 7.7 years (77 deaths among 223 patients), potassium supplementation was associated with higher all-cause mortality (log-rank p = <0.001; adjusted HR = 2.04, 95% CI 1.23–3.37, p = 0.006, Figure 1) and more heart-failure hospitalizations (IRR = 3.5,

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p<0.001). These associations were unchanged by adjustment for mean serum potassium and diuretic use, none of which were independent predictors of either outcome (Table 1). In a subgroup with available data on measured potassium (n = 166), the minimum K value was lower in the supplemented patients (3.25 ± 0.49 vs. 3.56 ± 0.45, p = 0.0002), but medium and highest K levels were similar in the two groups.

**Conclusions:** In this cardiorenal cohort, oral potassium supplementation marked a higher-risk phenotype (more atrial fibrillation, more intensive diuretic therapy) and was associated with worse survival and more heart-failure hospitalizations, an association that was not explained by use of diuretics.

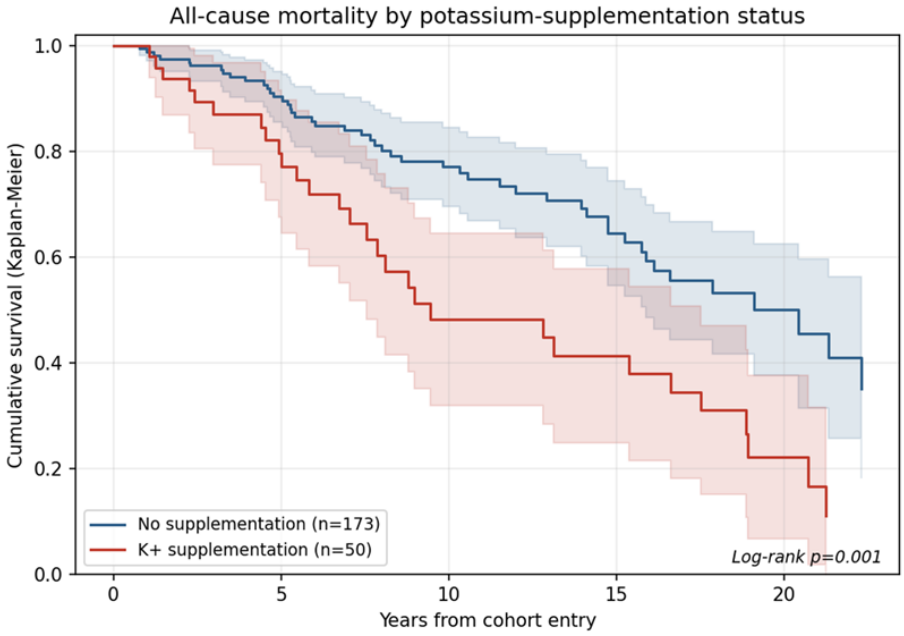



*Figure 1. Kaplan-Meier estimates of all-cause survival by potassium-supplementation status, with 95% Greenwood confidence bands.*

| Variable | HR | 95% CI | p |
|---|---|---|---|
| K+ supplementation (unadjusted) | 2.17 | 1.37–3.45 | **<0.001** |
| K+ supplementation (adjusted) | 2.04 | 1.23–3.37 | **0.006** |
| Age, per year | 1.07 | 1.04–1.10 | **<0.001** |
| Male sex | 0.81 | 0.49–1.35 | 0.426 |
| Best eGFR, per mL/min/1.73m2 | 1.01 | 0.99–1.02 | 0.288 |
| Diabetes mellitus | 1.06 | 0.65–1.73 | 0.823 |
| RAASi/ARNI use | 0.66 | 0.40–1.07 | 0.089 |
| Loop diuretic use | 0.95 | 0.53–1.70 | 0.852 |

*Table 2. Cox proportional-hazards model for all-cause mortality, unadjusted and adjusted (n=220, 76 deaths) for age, sex, best eGFR, diabetes, RAASi/ARNI use, and loop-diuretic use.*


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## SO-25

### Pegcetacoplan for 76 weeks maintains proteinuria reduction and eGFR stabilization for adolescent patients: VALIANT + VALE subgroup analysis

Dr. Tobias Vogler[1], Prof. Bradley P Dixon[2], Dr. Larry A Greenbaum[3], Prof. Gema Ariceta[4], Dr. Yael Borovitz[5], Dr. Antonio Mastrangelo[6], Dr. Nabil Melhem[7], Prof. Naoya Fujita[8], Dr. Nicole CAJ van de Kar[9], Dr. Dean Wallace[10], Dr. Eli Khankin[11], Dr. Anwesha Mukherjee[11], Dr. Elena Surova[12], Mr. Johan Szamosi[12], Dr. Marina Vivarelli[13], Prof. Christoph Licht[14], Prof. Carla Nester[15]

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**Background**: C3 glomerulopathy (C3G) and primary immune-complex mediated membranoproliferative glomerulonephritis (pIC-MPGN) are primarily driven by dysregulation of the alternative complement pathway affecting both children/adolescents and adults. Underlying causes of complement dysregulation are heterogenous and both genetic or acquired factors can play important roles. In the phase 3 VALIANT trial (NCT05067127), adolescents (12–17 years) receiving the C3/C3b inhibitor pegcetacoplan for C3G or pIC-MPGN over 1 year showed reduced proteinuria and stable eGFR; treatment continued in the VALE open-label extension (NCT05809531).

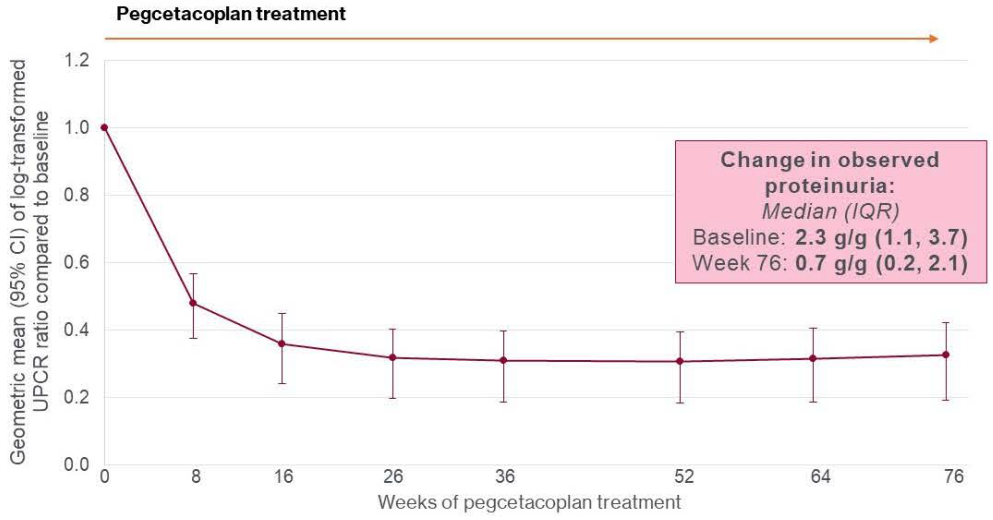
**Methods**: Of 55 adolescents enrolled in VALIANT, 53 continued pegcetacoplan through week 76, with VALIANT and VALE data pooled and aligned by cumulative treatment duration. Endpoints included change from baseline in urine protein-to-creatinine ratio (UPCR) and eGFR, plus treatment-emergent adverse effects. When available, investigators documented genetic or acquired complement abnormalities, and patients were classified by reported genetic factors, acquired factors, and disease subtype (primary IC-MPGN).

**Results**: After 76 weeks, adolescents sustained a substantial proteinuria reduction (geometric mean change from baseline [95% CI]: –67.5% [–77.0, –54.1]; Figure 1A), consistent with reductions at week 26 (–68.4%) and week 52 (–69.3%). eGFR remained stable (least squares mean [SE] change: +8.4 [4.43] mL/min/1.73 m²; Figure 1B). No new safety signals emerged. In this cohort 5 adolescents had reported genetic factors, 5 had acquired factors, and 27 had pIC-MPGN.

**Conclusions**: Sustained benefit with 76 weeks of pegcetacoplan in adolescents was consistent with outcomes in the broader VALIANT population.

**Figure 1. Proteinuria and eGFR changes for adolescent patients in VALIANT and VALE**

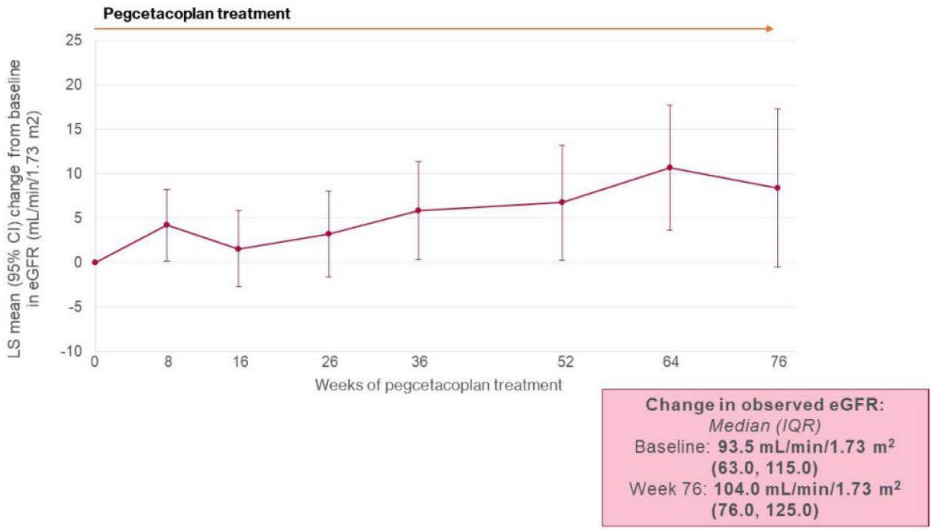
**A) Change from baseline in proteinuria over time**





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**B) Change from baseline in eGFR over time**




Abbreviations: eGFR, estimated glomerular filtration rate; LS, least squares; UPCR, urine protein-to-creatinine ratio.

## SO-26

### Phosphate Metabolism and Blood Pressure in the General Population: Modulation by Arterial Stiffness or Phosphate effect on Blood Pressure is modulated by arterial stiffness: a swiss general population-based study

Dr. Théodore Pasquier[1]

*Hôpitaux Universitaires Genève*

**Background:** Evidence linking phosphate to blood pressure (BP) is inconsistent, possibly reflecting lack of vascular phenotyping. We hypothesized that arterial stiffness modulates the phosphate–BP relationship.

**Methods:** Cross-sectional analysis of 919 adults from SKIPOGH, a multicenter family-based Swiss population cohort. Serum phosphate and 24-hour urinary phosphate excretion were related to office and 24-hour ambulatory BP using mixed-effects models (family as random effect) adjusted for age, sex, body mass index, smoking, hypertension, diabetes, LDL cholesterol, eGFR, serum calcium, heart rate and urinary sodium/potassium ratio. Interactions with carotid-femoral pulse wave velocity (PWV) and renal resistive index (RRI) were tested (n = 806).

**Results:** Mean age was 47.7±17.5 years; 48.7% were men. Urinary phosphate was not associated with any BP component. Serum phosphate was inversely associated with office systolic BP (SBP; ß = −1.66 mmHg per SD, IC95% −2.49 to −0.84) and diastolic BP, but not with 24-hour BP. PWV significantly modified the association of urinary (ß = -0.91 mmHg per SD, IC95% -1.70 to -0.13) and serum phosphate (ß = 0.80, IC95% 0.10 to 1.50) with 24-hour SBP, but not diastolic BP. At PWV 10 m/s, urinary phosphate was inversely associated with 24-hour SBP (ß = −1.71 mmHg per SD, CI95% −2.89 to −0.53), whereas serum phosphate was positively associated (ß = +1.51, IC95% 0.44 to 2.58), with no association at 5 or 7 m/s. The urinary phosphate interaction was replicated with RRI (ß = -0.87, IC95% -1.64 to -0.09). Interactions persisted after adjustment for FGF23 and parathyroid hormone.

**Conclusions:** The association between phosphate and ambulatory SBP could depend on arterial stiffness. In individuals with stiff arteries, urinary and serum phosphate seem associated with SBP in opposite directions. Vascular phenotyping may help reconcile conflicting literature.

## SO-27

### Prevalence and Determinants of Hypothyroidism in Patients on Routine Hemodialysis in Somalia.

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**Background:** Thyroid dysfunction is common in patients with end-stage kidney disease (ESKD) and may contribute to increased cardiovascular risk, malnutrition, and poor clinical outcomes. However, data from low-resource settings remain limited. This study aimed to assess the prevalence and determinants of hypothyroidism among patients receiving routine hemodialysis in Somalia.

**Methods:** A cross-sectional study was conducted at a tertiary dialysis center in Mogadishu, Somalia, between June and July 2022. Patients receiving routine hemodialysis were enrolled. Demographic and biochemical data were collected. Patients were classified as euthyroid, subclinical hypothyroid, or overt hypothyroid based on standard biochemical criteria. Logistic regression analysis was performed to identify factors associated with hypothyroidism.

**Results:** A total of 301 patients were examined. Their ages ranged from 40 to 66 years, with the median age being 54 (IQR

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= 40–66). Males were 167 (55.5%) compared to females 134 (44.5%). Hypertension was the most common comorbidity among the patients with 137 (45.5%). Diabetic kidney disease was the most common cause of renal failure in 138 patients (45.84%). The prevalence rate of hypothyroidism in hemodialysis patients in our study was 28%. In hypothyroidism patients 57.8% had subclinical hypothyroidism and 42.2% had overt hypothyroidism. 70.8% of our patients were in euthyroid status. Subclinical hypothyroidism was commonly seen in patients with diabetes, hypertension, and heart disease. We found that increasing age, decreased albumin level were related to higher risk of subclinical hypothyroidism. We also found that increasing creatinine levels were associated with lower risk of overt hypothyroidism.

**Conclusions:** Hypothyroidism is highly prevalent among patients receiving routine hemodialysis in Somalia, affecting more than one-quarter of patients, with subclinical hypothyroidism being the predominant form. Older age and markers of poor nutritional status were associated with thyroid dysfunction. These findings highlight the importance of considering routine thyroid function screening in hemodialysis populations, particularly in resource-limited settings.

**Table 1** Descriptive Statistics for Clinical Characteristics of Patients

| Variables | Frequency (%) |
|---|---|
| **Comorbidity** | |
| No known comorbid | 16 (5.3) |
| Diabetes | 101(33.6) |
| Hypertension | 137(45.5) |
| Heart disease | 43(14.2) |
| Smoker | 4(1.3) |
| **Causes of renal failure** | |
| Diabetic kidney disease | 138(45.84) |
| Hypertensive kidney disease | 100(33.22) |
| Glomerulonephritis | 28(9.3) |
| Polycystic kidney disease | 7(2.3) |
| Obstructive kidney disease | 13(4.3) |
| No known cause | 15(5) |


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**Table 2** Comparison of Patients' Feature Among Those Who Had Euthyroid and Hypothyroid (n=296)

| Variables | Euthyroid n(%) | Sub Clinical Hypothyroid n(%) | Overt Hypothyroidism n(%) | p-value |
|---|---|---|---|---|
| Age (in years)* | 52(36.5–65) | 59.5(48–70.75) | 60(42–72) | 0.039* |
| **Gender** | | | | |
| Male | 117(70.9) | 28(17) | 20(12.1) | 0.898 |
| Female | 96(73.3) | 20(15.3) | 15(11.5) | |
| **Causes of failure** | | | | |
| Diabetic kidney disease | 92(67.6) | 24(17.6) | 20(14.7) | 0.294‡ |
| Hypertensive kidney disease | 77(77.8) | 12(12.1) | 10(10.1) | |
| Glomerulonephritis | 22(84.6) | 2(7.7) | 2(7.7) | |
| Polycystic kidney disease | 6(85.7) | 1(14.3) | 0(0) | |
| Obstructive kidney disease | 7(53.8) | 4(30.8) | 2(15.4) | |
| No known cause | 9(60) | 5(33.3) | 1(6.7) | |
| **Comorbidity** | | | | |
| Diabetes | 75(76.5) | 12(12.2) | 11(11.2) | 0.388 |
| Hypertension | 93(69.9) | 25(18.5) | 17(12.6) | 0.532 |
| Heart diseases | 28(65.1) | 8(18.6) | 7(16.3) | 0.509 |
| Smoker | 3(75) | 1(25) | 0(0) | 0.734‡ |
| FT4* (ng/mL) | 0.7(0.6–0.9) | 0.6(0.6–0.8) | 0.2(0.2–0.2) | <0.001** |
| BUN* (mg/dL) | 123(93–152) | 114.5(99.25–148) | 119(81–148) | 0.372 |
| Creatinine* (mg/dL) | 7.1(5.3–9.5) | 7.75(5.25–9.925) | 5.6(3.8–7.7) | 0.003* |
| Hemoglobin* (g/dL) | 8.9(8.4–9.9) | 8.8(8.1–9.6) | 9.3(8.4–10) | 0.216 |
| Calcium* (mg/dL) | 8.6(8.2–9.2) | 8.75(7.75–9.375) | 8.6(7.8–9.2) | 0.894 |
| Albumin* (g/dL) | 3.7(3.5–4.2) | 3.5(3.125–3.9) | 3.7(3.3–4) | 0.007** |
| PTH* (pg/mL) | 258(178–389) | 244.5(160.75–392) | 251(149–301) | 0.190 |
| Potassium* (mEq/L) | 4.4(3.85–5.15) | 4.7(3.825–5.2) | 4.5(3.6–5.2) | 0.503 |
| Sodium* (mEq/L) | 139(135–140) | 137.5(135–140) | 137(135–139) | 0.248 |
| Phosphorous* (mg/dL) | 4.2(3.2–5.2) | 4.45(3.2–5.275) | 4.2(3.1–5.2) | 0.489 |
| Ferritin* (ng/mL) | 425(300–625) | 529.5(250–628) | 515(326–620) | 0.831 |

**Notes:** *Non-normal continuous data is reported as median (inter-quartile range), ‡Fisher-exact test is reported, *Significant at $\alpha<0.05$, **Significant at $\alpha<0.01$.
**Abbreviations:** FT4, free T4; n, Number of patients; PTH, Parathyroid hormone.

**Table 3** Post-Hoc Analysis Among Three Groups of Thyroid Status

| Comparison Between Three Groups | Age | FT4 | Creatinine | Albumin |
|---|---|---|---|---|
| Between euthyroid and overt hypothyroid | NS | ** | ** | NS |
| Between euthyroid and subclinical hypothyroid | ** | ** | NS | ** |
| Between subclinical and overt hypothyroid | NS | ** | ** | NS |

**Notes:** **Significant at $\alpha<0.01$.
**Abbreviations:** FT4, free T4; NS, non-significant.


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Table 4 Univariate Predictor of Hypothyroidism (n=296)

| Variables | Subclinical Hypothyroidism OR (95% CI) | p-value | Overt Hypothyroidism OR (95% CI) | p-value |
|---|---|---|---|---|
| Age (in years) | 1.02 (1–1.04) | 0.040* | 1.02 (0.99–1.04) | 0.107 |
| **Gender** | | | | |
| Male | 1.15 (0.61–2.17) | 0.668 | 1.09 (0.53–2.25) | 0.807 |
| Female | Ref | | Ref | |
| **Comorbid** | | | | |
| Diabetes | 0.61 (0.30–1.24) | 0.178 | 0.84 (0.39–1.81) | 0.663 |
| Hypertension | 1.40 (0.74–2.62) | 0.291 | 1.22 (0.59–2.49) | 0.588 |
| Heart diseases | 1.32 (0.56–3.11) | 0.524 | 1.65 (0.66–4.14) | 0.284 |
| BUN* (mg/dL) | 0.99 (0.99–1.07) | 0.867 | 0.99 (0.98–1) | 0.122 |
| Creatinine* (mg/dL) | 0.99 (0.99–1.01) | 0.878 | 0.76 (0.65–0.90) | 0.001** |
| Hemoglobin* (g/dL) | 0.87 (0.66–1.14) | 0.305 | 1.27 (0.93–1.73) | 0.134 |
| Calcium* (mg/dL) | 0.90 (0.64–1.28) | 0.573 | 0.87 (0.58–1.29) | 0.490 |
| Albumin* (g/dL) | 0.39 (0.22–0.70) | 0.002** | 0.56 (0.28–1.12) | 0.100 |
| PTH* (pg/mL) | 1 (0.99–1) | 0.844 | 1 (0.99–1) | 0.090 |
| Potassium* (mEq/L) | 1.27 (0.87–1.84) | 0.215 | 0.99 (0.65–1.53) | 0.981 |
| Sodium* (mEq/L) | 0.97 (0.90–1.04) | 0.380 | 0.94 (0.86–1.02) | 0.143 |
| Phosphorous* (mg/dL) | 1.22 (0.99–1.51) | 0.069 | 1.16 (0.91–1.48) | 0.239 |
| Ferritin* (ng/mL) | 1 (0.99–1) | 0.901 | 1 (0.99–1) | 0.757 |

Notes: *Non-normal continuous data is reported as median (inter-quartile range), *p-value significant at level of 0.05, **p-value significant at level of 0.01.
Abbreviations: BUN, blood urea nitrogen; CI, confidence interval; OR, odds ratio; Ref, Reference category; PTH, parathyroid hormone.

## SO-28

### Reversible IL-6-Responsive Gd-IgA1+ B Cells in Adalimumab-Associated Secondary IgA Nephropathy

Mrs. Katerina Zachova[1], Mrs. Hana Flogelova[2], Mrs. Alica Cutkova[1], Mrs. Jana Jemelkova[1], Mr. Tomas Tichy[3], Prof. Frantisek Mrazek[1], Prof. Josef Zadrazil[4], Prof. Milan Raska[1]

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**Background:** IgA nephropathy (IgAN), the most common primary glomerulonephritis, is characterized by mesangial deposition of galactose-deficient IgA1 (Gd-IgA1)-containing immune complexes. Elevated IL-6 levels in serum and urine are associated with disease activity and promote production of pathogenic Gd-IgA1 by patient-derived plasmablasts. Although both inflammatory bowel disease (IBD) and anti-TNF therapy have been associated with IgAN, distinguishing therapy-associated immune dysregulation from coincidental disease remains challenging.

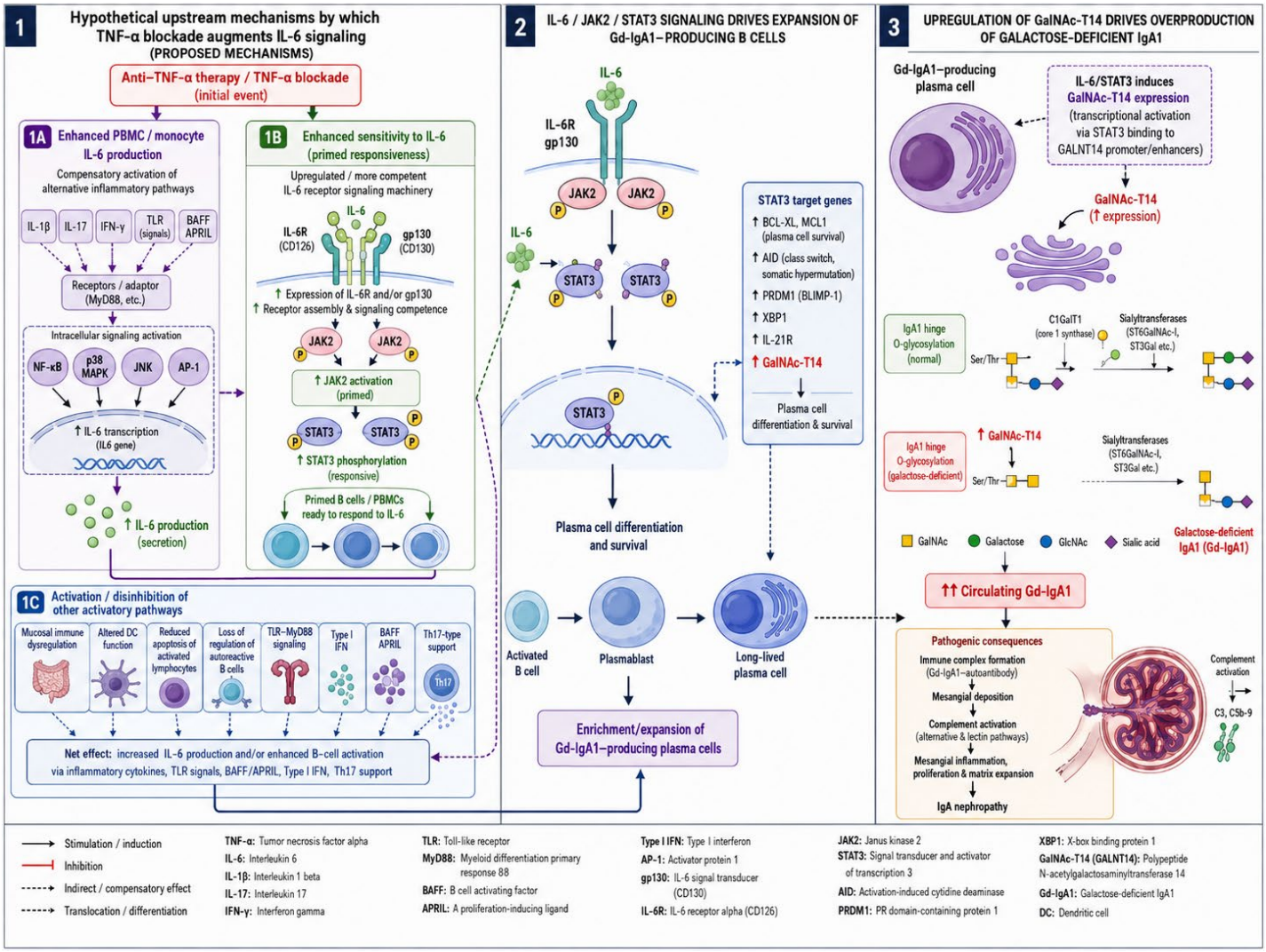
**Case Presentation:** A 17-year-old female with 7-year history of ulcerative colitis developed microscopic hematuria, proteinuria (1–1.5 g/day), and mild renal dysfunction after 3 years of adalimumab therapy. Renal biopsy confirmed IgA nephropathy (Oxford classification M1E0S1T1C1). HLA genotyping revealed homozygous *DRB111:03, DQA105:05* and *DQB103:01*, alleles reported in association with increased anti-TNF immunogenicity. Following adalimumab discontinuation and initiation of systemic corticosteroid therapy followed by methotrexate, renal function improved, resulting in sustained clinical remission.

**Methods:** Peripheral blood mononuclear cells (PBMCs) were analyzed during anti-TNF therapy, and after its discontinuation.

**Results:** Baseline proportions of $CD19^+$, $IgA^+$, and $Gd\text{-}IgA1^+$ cells remained unchanged. In contrast, IL-6 *in vitro* stimulation of PBMC sampled during anti-TNF therapy drove a striking expansion of $Gd\text{-}IgA1^+$ cells, reaching 92.4% of total $IgA^+$ population, accompanied with increased GalNAc-T14 mRNA expression, whereas healthy controls and conventional IgAN patients show minimal IL-6 responsiveness. Exaggerated response progressively normalized after withdrawal of adalimumab.

**Conclusions:** The exaggerated IL-6–driven expansion of $Gd\text{-}IgA1^+$ B cells and increased GalNAc-T14 expression during adalimumab therapy may provide a novel means of identifying therapy-associated immune dysregulation and support more individualized anti-TNF treatment decisions.


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## SO-29

### Sparsentan as Rescue Therapy for Treatment Refractrory PLA2R-negative Membranous Glomerulonephritis with ACE Inhibitor Intolerance

Dr. Martina Pechula Thut[1]

1. *Spital Zollikerberg*

**Introduction:** Sparsentan, a dual endothelin type A/angiotensin II typ 1 receptor antagonist (DEARA), is currently approved exclusively for IgA nephropathy; data on its use in membranous glomerulonephritis are lacking. I report the off-label use of sparsentan in a patient with treatment-refractory, PLA2R-negative membranous nephropathy who was intolerant of conventional RAS blockade.

**Case Presentation:** A man born in 1964 was diagnosed with PLA2R-negative membranous glomerulonephritis by biopsy in 2013 (eGFR 61 ml/min, proteinuria 1.2g/d), initially well controlled on an ACE inhibitor. In 2015 the disease progressed, with nephrotic-range proteinuria (4g/d) and worsening renal function (eGFR 39 ml/min). Because of a myocardial infarction shortly beforehand, steroid therapy was avoided and tacrolimus was used instead for 18 months, resulting in partial remission (proteinuria 1g/d, stable renal function). When proteinuria later rose again to 2.5g/d (eGFR 47ml/min), and because an ACE inhibitor allergy that had since developed precluded conventional RAS blockade, sparsentan was initiated on December 1, 2025. This led to a rapid and sustained reduction in proteinuria to 0.6 g/d after 6 weeks and to 0.35 g/d after 8 months, with stable renal function (eGFR 35-37 ml/min). Treatment was well tolerated.

**Conclusion:** In a patient with treatment-refractory, PLA2R-negative membranous glomerulonephritis and ACE inhibitor intolerance, sparsentan led to near-complete remission of proteinuria with stable renal function. This suggests a potential therapeutic role for sparsentan beyond IgA nephropathy and warrants further investigation in membranous nephropathy.


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## SO-30

### Structure and function analysis of (likely) pathogenic NPHS2 missense variants in cell culture and zebrafish

Ms. Blanda Beci[1], Dr. Carolin Eul[1], Dr. Sucheta Sridhar[1], Dr. Luisa Bertgen[1], Prof. Britta George[2]

1. *Universität Zürich, 2. Universitätsspital Zürich*

Steroid-resistant nephrotic syndrome (SRNS) is a rare and severe kidney disease characterized by proteinuria often resulting in kidney failure. Monogenic causes of SRNS highlight the podocyte, particularly the slit diaphragm protein podocin encoded by *NPHS2*. This project aims to assess the molecular pathomechanisms of *NPHS2* missense variants by investigating their effects on podocin trafficking, localization, and function. A systematic structure-function analysis of 13 (likely) pathogenic podocin missense variants was performed in HEK-293T cells stably expressing eGFP-tagged wildtype or mutant podocin. Protein expression was analyzed by Western blot and localization of podocin was assessed by colocalization with an endoplasmic reticulum (ER) tracker, or a BFP labeled plasma membrane.Most variants showed impaired intracellular trafficking and ER retention. Seven of the 13 variants co-localized with the ER tracker and four with the plasma membrane marker. In contrast, two variants displayed an intermediate phenotype and partially reached the plasma membrane. These findings highlight the impact of the identified variants on podocin trafficking at the cellular level and raise the question of their functional consequences *in vivo*. Therefore, functional consequences of podocin deficiency will be evaluated *in vivo*, with a generated CRISPR/Cas9-mediated *nphs2* knockout zebrafish model. Podocin and key pathways involved in kidney development are highly conserved between zebrafish and humans. The knockout (KO) phenotype will be characterized by immunofluorescence staining, as well as with functional assays. Electron microscopy revealed foot process effacement and partially loss of slit diaphragms for the homozygous *nphs2* KO fish 5 days post fertilization (dpf). Rescue experiments with human podocin will assess functional conservation and may establish a platform for future testing of pathogenic *NPHS2* variants.

## SO-31

### Temporal trends in characteristics and outcomes of patients initiating dialysis in Switzerland from 2014 to 2025

Ms. Rebecca Guidotti[1], Prof. Patrice Ambühl[2]

1. *Stadtspital Zürich, 2. non*

**Background:** Patient characteristics and treatment patterns may have changed over time, with potential implications for outcomes. We investigated temporal trends in patients initiating dialysis in Switzerland between 2014 and 2025.

**Methods:** We analyzed Swiss Dialysis Registry data on all incident dialysis patients between 2014 and 2025 (n = 10’108), with a mean follow-up of 3.0±2.4 years. Patients were grouped into four eras (2014–2016, 2017–2019, 2020–2022, and 2023–2025). Baseline characteristics were compared across eras, and time-to-event outcomes were analyzed using Kaplan–Meier methods and multivariable Cox regression adjusted for prespecified covariates.

**Results:** Patient characteristics and treatment patterns changed over time. Mean age increased from 65.0±16.0 years in 2014–2016 to 67.4±16.2 years in 2023–2025 ($p<0.001$), while BMI and Charlson Comorbidity Index (CCI) remained stable. The proportion of patients receiving peritoneal dialysis decreased from 14.6% in 2014–2016 to 11.9% in 2020–2022, followed by an increase to 13.2% in 2023–2025. Vascular access patterns also changed over time, with a decrease in arteriovenous fistula use and an increase in catheter use ($p<0.001$). In unadjusted analysis, mortality did not differ significantly across the four eras ($p = 0.180$). After adjustment for age, sex, BMI, and CCI, mortality differed across dialysis eras (overall $p = 0.025$), with a lower hazard of death observed among patients initiating dialysis in 2023–2025 compared with 2014–2016 (HR 0.844, 95% CI 0.742–0.961; $p = 0.011$).

**Conclusions:** Patients initiating dialysis in 2023–2025 had lower adjusted mortality than those initiating dialysis in 2014–2016, after accounting for age, sex, BMI, and comorbidity. This finding occurred alongside substantial changes in dialysis treatment patterns, while comorbidity at initiation remained stable. Better survival despite an older population may reflect improvements in the overall care of patients with kidney failure, although the reasons remain unclear.

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**Table 1: Baseline patient characteristics according to dialysis era**

| | Era | | | | |
|---|---|---|---|---|---|
| | **2014-2016** | **2017-2019** | **2020-2022** | **2023-2025** | **p-value** |
| **Number of patients, n** | 2'555 | 2'534 | 2'570 | 2'449 | - |
| **Age (years), mean±SD** | 65.0±16.0 | 66.6±15.8 | 66.5±16.1 | 67.4±16.2 | <0.001 |
| **Sex (male), % (n)** | 66.0 (1'686) | 67.4 (1'708) | 67.0 (1'722) | 68.2 (1'669) | 0.429 |
| **Caucasian, % (n)** | 92.4 (2'330) | 93.1 (2'358) | 91.8 (2'360) | 90.3 (2'211) | <0.001 |
| **Nationality, % (n)** | | | | | 0.126 |
| Swiss | 76.0 (1'446) | 76.4 (1'933) | 75.7 (1'942) | 74.2 (1'815) | |
| EU citizen | 12.7 (242) | 13.2 (335) | 14.1 (363) | 13.4 (327) | |
| Other | 11.3 (215) | 10.4 (262) | 10.2 (262) | 12.5 (305) | |
| **BMI, mean±SD** | 26.1±5.8 | 26.2±6.2 | 26.4±5.8 | 26.1±6.1 | 0.139 |
| **Dialysis sessions/week, mean±SD** | 3.13±1.19 | 2.96±0.26 | 2.93±0.31 | 2.86±0.38 | <0.001 |
| **Dialysis hours/week, mean±SD** | 11.0±2.6 | 11.3±1.5 | 11.0±1.7 | 10.7±2.0 | <0.001 |
| **Hemoglobin, mean±SD** | 10.8±1.7 | 10.9±1.6 | 10.8±1.6 | 10.9±1.6 | 0.597 |
| **Ferritin, median (IQR)** | 284 (151-481) | 311 (170-516) | 410 (202-594) | 355 (190-578) | <0.001 |
| **Charlson Comorbidity Index, mean±SD** | 4.26±2.19 | 4.16±2.12 | 4.24±2.20 | 4.26±2.07 | 0.171 |
| **Iron substitution, % (n)** | 76.3 (770) | 74.4 (1'882) | 75.2 (1'929) | 72.7 (1'777) | 0.088 |
| **EPO substitution, % (n)** | 82.4 (831) | 80.3 (2'029) | 78.5 (2'014) | 79.1 (1'933) | 0.050 |
| **Hypertension, % (n)** | 78.5 (2'004) | 84.6 (2'140) | 82.9 (2'128) | 84.7 (2'071) | <0.001 |
| **Diabetes, % (n)** | 37.9 (966) | 37.7 (955) | 38.6 (992) | 39.6 (968) | 0.520 |
| **Cardiovascular disease, % (n)** | 49.5 (1'264) | 52.1 (1'317) | 52.0 (1'334) | 52.5 (1'284) | 0.140 |

**Table 2. Dialysis modality and vascular access at initiation according to dialysis era**

| | Era | | | | |
|---|---|---|---|---|---|
| | **2014-2016** | **2017-2019** | **2020-2022** | **2023-2025** | **p-value** |
| **Number of patients, n** | 2'555 | 2'534 | 2'570 | 2'449 | |
| **Dialysis type, % (n)** | | | | | 0.005 |
| Home HD | 1.5 (39) | 1.1 (29) | 1.1 (28) | 0.9 (23) | |
| Center HD | 82.0 (2'093) | 81.2 (2'055) | 85.0 (2'182) | 83.0 (2'032) | |
| Self Care | 1.8 (47) | 2.8 (71) | 2.0 (51) | 2.7 (67) | |
| PD | 14.6 (374) | 14.8 (375) | 11.9 (306) | 13.2 (324) | |
| **Material, % (n)** | | | | | <0.001 |
| Fistula | 43.6 (949) | 38.9 (838) | 34.9 (788) | 36.2 (768) | |
| Catheter | 49.0 (1'068) | 55.1 (1'187) | 59.8 (1'353) | 59.9 (1'272) | |
| Graft | 7.4 (162) | 5.9 (128) | 5.2 (117) | 3.9 (83) | |
| **Shunt Localisation, % (n)** | | | | | <0.001 |
| Forearm | 69.8 (776) | 72.9 (704) | 65.1 (589) | 64.0 (545) | |
| Upper arm | 30.2 (335) | 27.1 (262) | 34.9 (316) | 36.0 (306) | |
| **Catheter type, % (n)** | | | | | <0.001 |
| Temporary | 5.8 (62) | 3.5 (42) | 3.4 (46) | 1.5 (19) | |
| Tunneled | 94.2 (1'006) | 96.5 (1'145) | 96.6 (1'307) | 98.5 (1'253) | |


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## SO-32

### Two methods for histomorphometry of aortic microcalcifications in experimental kidney disease using in-situ binding of exogenous Fetuin-A

Dr. Miguel Correia[1], Dr. Sandra Stefanikova[2], Dr. Yunsu Jang[2], Dr. Stefan Rudloff[2], Dr. Matthias Moor[3]

*1. Department of Nephrology, Inselspital, Bern University Hospital, University of Bern, Bern, Switzerland, 2. Inselspital, Department of Nephrology, Bern, Switzerland, 3. Inselspital Bern*

**Introduction:** Fetuin-A (FA) is an endogenous circulating calcification inhibitor. Here, we hypothesized that the binding of recombinant FA to calcium phosphate microcrystals can be used to estimate vascular calcification propensity in formalin-fixed paraffin-embedded biobanked aortic tissues from an experimental model of chronic kidney disease (CKD).

**Methods:** Biobanked aorta tissue from n = 63 mice, 5 mouse strains without or with adenine nephropathy (Groups 1, 2) or adenine nephropathy plus bisphosphonate [zoledronate] treatment (Group 3) was sectioned, rehydrated and incubated with mRuby-coupled purified Fetuin-A. After DAPI counterstain and mounting, an IMIC digital microscope was used for image acquisition. Microcalcifications were first quantified using the deep learning method StarDist2D after a model was trained on a set of images annotated using the SAMJ-tiny annotator plugin in FIJI. Alternatively, microcalcification-spots were evaluated via the deep learning method Spotiflow. Spotiflow was used as a Python library and a napari plugin was used for ease of interface. Macrocalcifications were calculated as von Kossa-positive tissue area fractions. Spearman's correlation was calculated across methods. Linear models estimated CKD and bisphosphonate effects on log-transformed microcalcification count.

**Results:** Both Spotiflow and Stardist quantification revealed tendencies of aortic microcalcification in CKD versus control aortae of 48 males, but only with Stardist this reached significance (beta: 0.53, 95% confidence interval 0.06-1.0, p = 0.03). Zoledronate showed no significant effect on overall aortic microcalcifications, but strain differences existed. For correlation of aortic microcalcifications with macrocalcifications, Spotiflow revealed rho = -0.12 (p = 0.38) and Stardist rho = -0.14 (p = 0.31) across all 63 specimen.

**Discussion:** This study sheds light on strain differences in CKD-related microcalcifications. Microcalcifications poorly correlate with macrocalcifications. This observation warrants further investigation, as micro- vs. macrocalcifications may have different kinetics and pathogenicity.

Figure 1: 60X magnified aortic microcalcifications (top), quantified by Stardist across 5 mouse strains (middle), and log-transformed correlation between the two methods Stardist and Spotiflow (bottom).

## SO-33

### Upfront Combination Therapy With Nefecon and Sparsentan in IgA Nephropathy: Early Real-World Experience

Dr. Stephanie Zappi[1], Dr. Raffaela Flury[1], Mrs. Beatrice Paul[1], Dr. Davide Spica[1], Dr. Min Jeong Kim[1]

1. *Kantonsspital Aarau*

**Background:** With the rapid evolution of the therapeutic landscape in IgA nephropathy (IgAN), an increasing number of treatment options are becoming available. However, the optimal selection, sequencing, and combination of these therapies remain unclear. Here, we report our initial real-world experience with upfront combination therapy with nefecon and sparsentan in patients with IgAN at high risk of progression.

**Methods:** Patients with biopsy-proven IgAN and a high risk of disease progression were initiated on nefecon and sparsentan concomitantly. Treatment with an ACE inhibitor and an SGLT-2 inhibitor was initiated prior to or shortly after kidney biopsy. Proteinuria and albuminuria (uPCR and uACR; mg/mmol), as well as estimated glomerular filtration rate (eGFR; CKD-EPI, mL/min/1.73 m²) and hematuria (erythrocytes/µL), were regularly monitored during follow-up.

**Results:** Three male patients with IgAN were initiated on upfront combination therapy with Nefecon and sparsentan. Clinical and laboratory parameters at the time of kidney biopsy are summarized in Table 1. In the first patient, the sparsentan dose was reduced to 200 mg/day due to low blood pressure, while the Nefecon dose was reduced to 8 mg/day because of muscle cramps. The remaining patients tolerated the full doses of both therapies well. Follow-up data are summarized in Table 2. Overall, treatment response was favorable, with a rapid reduction in proteinuria and albuminuria. In patients 2 and 3, a decline in eGFR was observed, considered to be hemodynamic in nature. Continued treatment and further follow-up are planned.

**Conclusions:** In this initial real-world experience, patients with IgAN at high risk of progression showed a favorable response to upfront combination therapy with nefecon and sparsentan, in addition to RAS inhibition and SGLT-2 inhibitor therapy, with a rapid reduction in proteinuria. Further follow-up will be required to assess the longer-term effects of this treatment strategy on kidney function.

**Table 1. Clinical and Laboratory Parameters at Kidney Biopsy**

| Age | Sex | Race | BMI kg/m² | BP mmHg | uPCR mg/ mmol | uACR mg/mmol | Hematuria /µl | eGFR ml/min/1.73m² | RASi | SGLT2i | M 0/1 | E 0/1 | S 0/1 | T 0/1/2 | C 0/1/2 | Risk of progression (%)* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 32 | M | white | 20 | 106/69 | 156 | 109 | 1212 | 75 | + | - | 1 | 0 | 1 | 0 | 0 | 9.5 |
| 64 | M | white | 35 | 138/82 | 768 | 534 | 78 | 46.8 | + | + | 1 | 1 | 0 | 0 | 1 | 19.3 |
| 50 | M | white | 25.5 | 128/87 | 103 | 74.6 | 23 | 40 | + | + | 1 | 0 | 1 | 0 | 0 | 78 |

* International IgAN Prediction Tool at biopsy - Risk of a 50% eGFR decline or progression to end-stage renal disease 5.0 years after renal biopsy

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**Table 2. Follow-up Parameters During Upfront Combination Therapy With Nefecon and Sparsentan in Addition to RAS Inhibition and SGLT-2 Inhibitor Therapy**

| | | Kidney biopsy | Start Nefecon und Sparsentan | 3 months | 5 months |
|---|---|---|---|---|---|
| Patient 1 (32 yrs) | uPCR, mg/mmol | 156 | 114 | 43.2 | 39.5 |
| | uACR, mg/mmol | 109 | 76.8 | 22.8 | 18.4 |
| | eGFR, ml/min/1.73m² | 75 | 75 | 75 | 76 |
| | Hematuria, /µl | 1212 | 136 | 20 | 33 |
| Patient 2 (65 yrs) | uPCR, mg/mmol | 768 | 270 | 32.5 | |
| | uACR, mg/mmol | 534 | 192 | 9.74 | |
| | eGFR, ml/min/1.73m² | 46.8 | 38 | 35 | |
| | Hematuria, /µl | 78 | 392 | 10 | |
| Patient 3 (50 yrs) | uPCR, mg/mmol | 103 | 112 | 50.8 | |
| | uACR, mg/mmol | 74.6 | 72.3 | 22.6 | |
| | eGFR, ml/min/1.73m² | 40 | 40 | 36 | |
| | Hematuria, /µl | 23 | 102 | 15 | |

## SO-34

### Urinary Dickkopf-3 as a Potential Biomarker for Long-Term Renal Stability

Dr. Androniki Papachristou[1], Dr. Ivo Strebel[2], Dr. Markus Mutke[3], Mr. Raphael Kaufmann[2], Dr. Gabrielle Hure[2], Dr. Stefan Neuhaus[4], Prof. Stefano Bassetti[3], Prof. Christian Mueller[2], Prof. Danilo Fliser[4], Prof. Tobias Breidthardt[5]

*1. Division of Internal Medicine, University Hospital Basel, Basel and Department of Cardiology, Cardiovascular Research Institute of Basel, University Hospital Basel, Basel, 2. Department of Cardiology, Cardiovascular Research Institute of Basel, University Hospital Basel, Basel, 3. Division of Internal Medicine, University Hospital Basel, Basel, 4. Department of Internal Medicine IV-Nephrology and Hypertension, Saarland University Medical Center, Homburg/Saar, 5. Division of Internal Medicine, University Hospital Basel, Basel and Department of Internal Medicine, Luzerner Kantonsspital (LUKS), Lucerne*

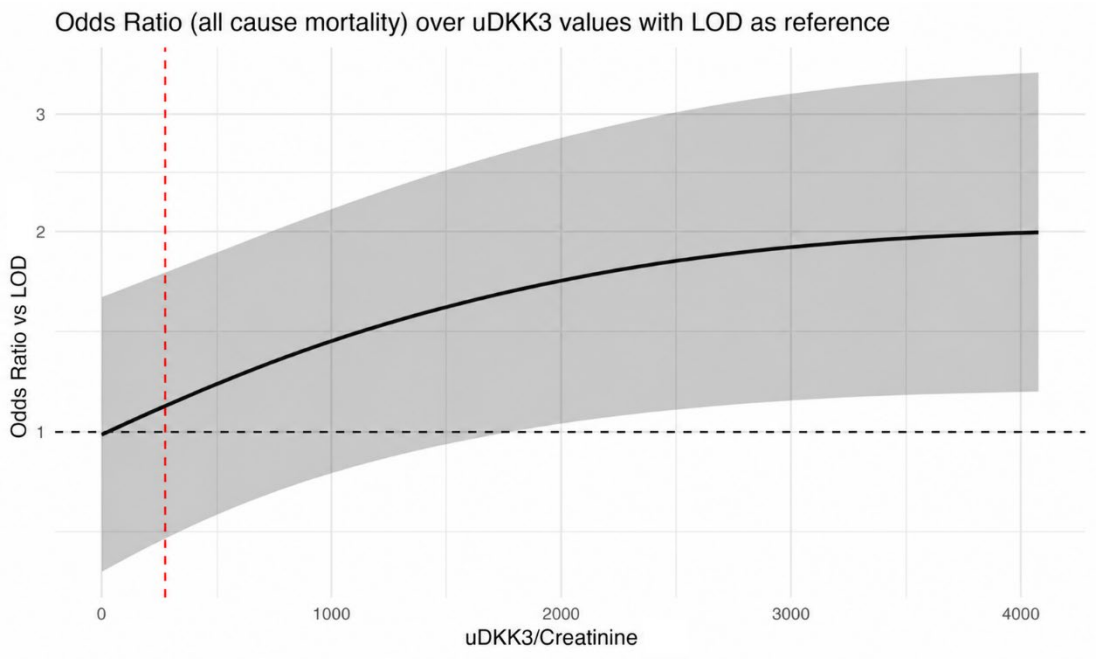
**Background:** Chronic kidney disease (CKD) is a leading cause of death and impairs quality of life. Predicting individual kidney function course remains an unmet clinical need. We explored the association of urinary Dickkopf-3 (uDKK3), a tubular epithelial-derived glycoprotein, with all-cause mortality and long-term glomerular filtration rate (GFR) trajectories.

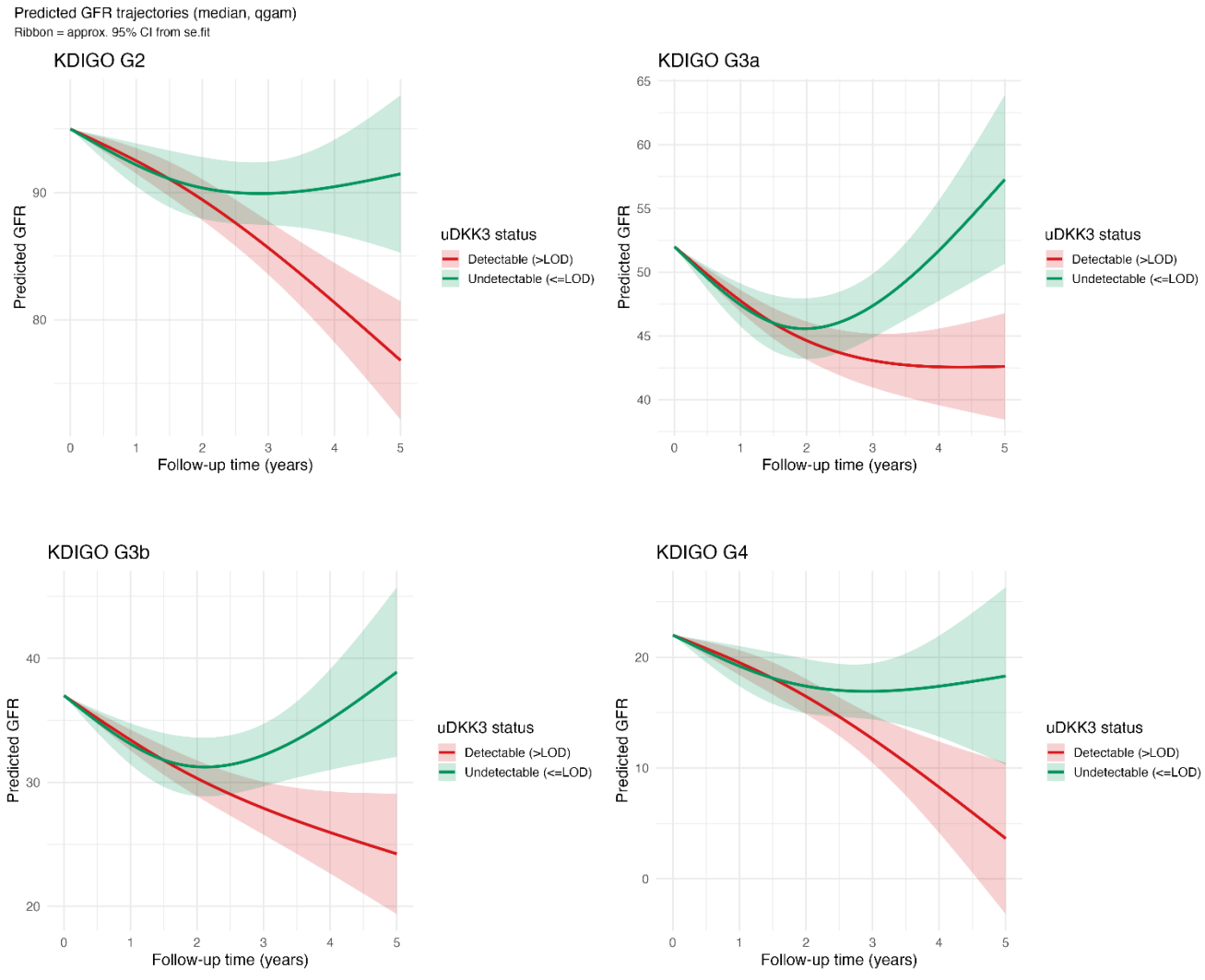
**Methods:** In an international multicenter observational cohort of adults presenting to emergency departments with dyspnea of any cause, we measured uDKK3 at discharge. uDKK3 was indexed to urinary creatinine; the detection limit was 30 pg/mg. We assessed all-cause mortality in 917 patients using adjusted logistic regression. We analyzed five-year GFR trajectories in 521 patients with serial GFR measurements using adjusted median quantile generalized additive mixed models.

**Results:** Of 917 patients, 369 (40%) died. Higher uDKK3/creatinine concentrations were independently associated with all-cause mortality (OR 1.43, 95% CI 1.18–1.74), with increasing odds ratio across the concentration range (Figure 1). Discrimination was modest (AUC 0.605, 95% CI 0.567–0.642). Among 521 patients, 20% had uDKK3/creatinine below the limit of detection (LOD). Across discharge KDIGO categories, median GFR trajectories significantly differed between patients with detectable and undetectable uDKK3/creatinine (Figure 2). At 5 years, detectable uDKK3 was significantly associated with renal progression (delta-GFR −9.08 mL/min/1.73 m²; 95% CI, −13.14 to −5.02), whereas undetectable uDKK3 identified stable GFR trajectories (delta-GFR +5.59 mL/min/1.73 m²; 95% CI, −0.97 to 12.15). Compared with undetectable uDKK3, the predicted median GFR change for a typical patient with detectable uDKK3 was 4.29 (95% CI, −7.56 to −1.03) and 14.67 mL/min/1.73 m² (95% CI, −22.38 to −6.95) lower at 3 and 5 years, respectively.

**Conclusions:** Discharge uDKK3 concentrations were independently associated with all-cause mortality and long-term GFR trajectories. Importantly, uDKK3 below the limit of detection identified a low-risk population without renal disease progression, while detectability identified patients at risk for kidney function decline across KDIGO categories.

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## SO-35

### Variation in CKD-induced aortic calcification across five inbred mouse strains

Dr. Sandra Stefanikova[1], Dr. Yunsu Jang[1], Dr. Stefan Rudloff[1], Dr. Matthias Moor[1]

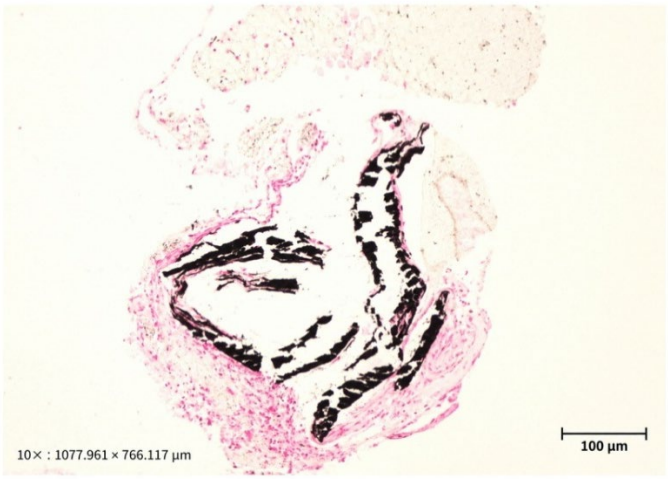
*Inselspital, Department of Nephrology, Bern, Switzerland*

**Background:** Vascular calcification is a driver of cardiovascular complications in chronic kidney disease (CKD). Here, we hypothesized that the BXD consortium of recombinant inbred (RI) mouse strains presents an opportunity for investigating the contribution of genetic background to human-relevant CKD-induced disease phenotypes.

**Methods:** We assessed biobanked aortic specimen that were collected during a population genetics pilot study on renal osteodystrophy. Total 100 mice including both sexes of 5 BXD strains were randomized to one of three experimental groups: control diet, 0.2% adenine diet for six weeks to induce CKD, or adenine diet plus a single zoledronate (ZOL) injection. Serum blood urea nitrogen was used to verify CKD severity. Abdominal aorta calcification was assessed in 91 mice by von Kossa staining, followed by an automated greyscale threshold-based quantification in Image J. Linear regression was used to estimate effects on calcified tissue area, with experimental group, sex, and strain as exposures of interest.

**Results:** Adenine treatment increased aortic calcification compared to control diet (β = 0.06, 95% CI 0.02-0.11, p = 0.005). Similarly, adenine + ZOL was associated with increased calcification compared with controls (β = 0.05, 95% CI 0.01-0.09, p = 0.019). Male sex showed a non-significant trend to increased aortic calcification (β = 0.03, 95% CI 0.00-0.07, p = 0.07). Substantial variation in aortic calcification was observed across strains.

**Figure 1.** Representative von Kossa-stained aorta crosssection of a male mouse from a calcification-prone BXD strain.




**Conclusion:** Adenine-induced CKD was associated with increased aortic calcification across the 5 studied inbred mouse strains, with substantial strain-dependent variation. The present data allow the formal calculation of heritable fraction of the variation in CKD-induced vascular calcification and will thus serve for a power analysis of a future genomic screen in BXDs.

## SO-36

### What do fish scales have to do with end stage kidney disease and colitis?

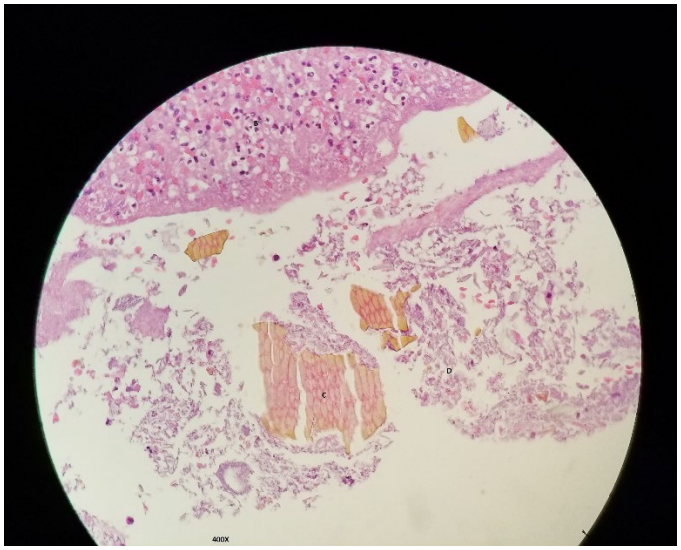
Dr. Catarina Mata[1], Dr. Anja Korntheuer[2], Dr. Stephan Grouls[1], Dr. Agnes Kneubühl[1]

1. *Hospital Lachen, 2. City Hospital Triemli Zürich*

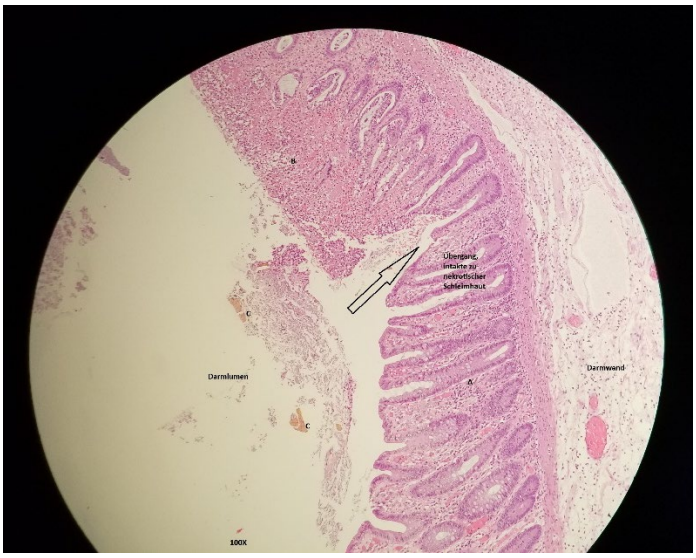
**Background:** Hyperphosphatemia is a major driver of vascular and bone complications in end stage renal disease. Phosphate binders are a usual treatment option. While gastrointestinal side effects such as nausea, vomiting, abdominal pain and constipation are well recognized, severe mucosal injury, including ischemic colitis, has rarely been reported.

**Methods:** We report the case of a 67-year-old man with end stage kidney disease due to diabetes mellitus type 2 and severe obesity, on dialysis for five years now, currently taking 2.4 g sevelamer carbonate daily. He was admitted to hospital for evaluation of weight loss of almost 20 kg and newly irregular bowel inhabits. Colonoscopy was planned but could not be completed due to inadequate bowel preparation. Mistakenly phosphate containing laxative was given by a nurse. One day later the patient developped sudden clinical deterioration with severe abdominal pain. CT scan showed a dilated colon filled with stool and gas, consistent with stercoral colitis, likely reflecting the inadequate bowel preparation, probably contributing to mucosal injury. He developed septic shock with ischemic colitis the follwing day, an emerging colonoscopy revealed colonic necrosis, prompting hemicolectomy with formation of stoma.

**Results:** Histology showed necrotic colon, mucosal changes with luminal fish-scale crystalline structures (picture 1, 2), typically described in sevelamer carbonate-associated colitis and not by intake of phosphate containing laxatives. After surgical treatment and stabilisation in the intensive care unit and cessation of sevelamer-carbonat, the patient improved and recovered.




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**Conclusion:** In end stage kidney disease patients with fecal stasis and ischemic bowel disease sevelamercarbonat-associated colitis has to be considered. Histology is one of the cornerstones for diagnosis and typically shows luminal fish-scale crystalline structures. Predisposing risk factors are severe renal insufficiency, hypotension, fecal stasis. In literature even gastrointestinal bleeding and colon rupture are reported. Pathophysiologically crystal formation is explained by the non absorbable anion-exchange resin.

## ELEVATOR PITCH PRESENTATION

### EP-01

### An 82-Year-Old Woman with Glomerulonephritis and Acute Kidney Injury: the Therapeutic Dilemma

Dr. Heidi Sarrasin[1], Dr. Nicolas Cornière[1], Dr. Anne-Hélène Reboux[1], Ms. Soukaina Abdul[1], Dr. Laura Moi[2], Prof. Daniel Teta[3]

*1. Service of nephrology, Valais Hospital, Sion, Switzerland, 2. Service of immunology and allergology, Institut Central des Hôpitaux, Valais Hospital, Sion, Switzerland, 3. Hôpital du Valais – Centre Hospitalier du Valais Romand (CHVR), Hôpitaux de Martigny, Sierre et Sion, Service of Nephrology, Sion*

**Background:** Anti-glomerular basement membrane (GBM) disease is a rare and potentially life-threatening disorder characterized by rapidly progressive glomerulonephritis. Early diagnosis and prompt treatment with plasma exchange, corticosteroids, and cyclophosphamide are essential to reduce pathogenic antibody levels and prevent irreversible kidney damage. However, management remains uncertain in patients with severe renal involvement and persistent anti-GBM antibodies despite prolonged therapy.

**Case presentation:** A healthy 82-year-old woman with fever and myalgia was admitted with acute kidney injury (serum creatinine 380 µmol/L), preserved urine output, glomerular hematuria, and proteinuria. Anti-GBM antibodies were strongly positive (2600 U), while ANCA was negative and there was no pulmonary involvement. Kidney biopsy showed severe acute crescentic glomerulonephritis with cellular crescents, fibrinoid necrosis, and Bowman's capsule rupture, with limited chronic damage. Treatment with corticosteroids, daily plasma exchange, and intravenous cyclophosphamide was promptly initiated. After 9 weeks, 52 plasma exchanges and 2 g cyclophosphamide, anti-GBM antibody levels had fallen to 35 U, although remained weakly positive (negative <20 U). Following a brief interruption of plasma exchange, anti-body levels rebounded, prompting the addition of mycophenolate mofetil and resumption of plasma exchange three times weekly until seronegativity. A repeat kidney biopsy showed predominantly chronic lesions with minimal residual activity. Despite severe renal impairment, kidney replacement therapy was never required. The patient remains dialysis-free, with serum creatinine of 388 µmol/L and anti-GBM antibody levels close to the negative threshold. Further renal recovery is anticipated.

**Conclusion:** This case highlights the challenge of severe anti-GBM disease with persistent antibody positivity despite prolonged intensive treatment. The discrepancy between serological activity and predominantly chronic renal lesions raises questions about the optimal duration of plasma exchange and the role of antibody titers in guiding therapy. Prolonged plasma exchange may help preserve kidney function despite poor prognostic features. Mycophenolate mofetil may be considered in cases of persistent antibody positivity.


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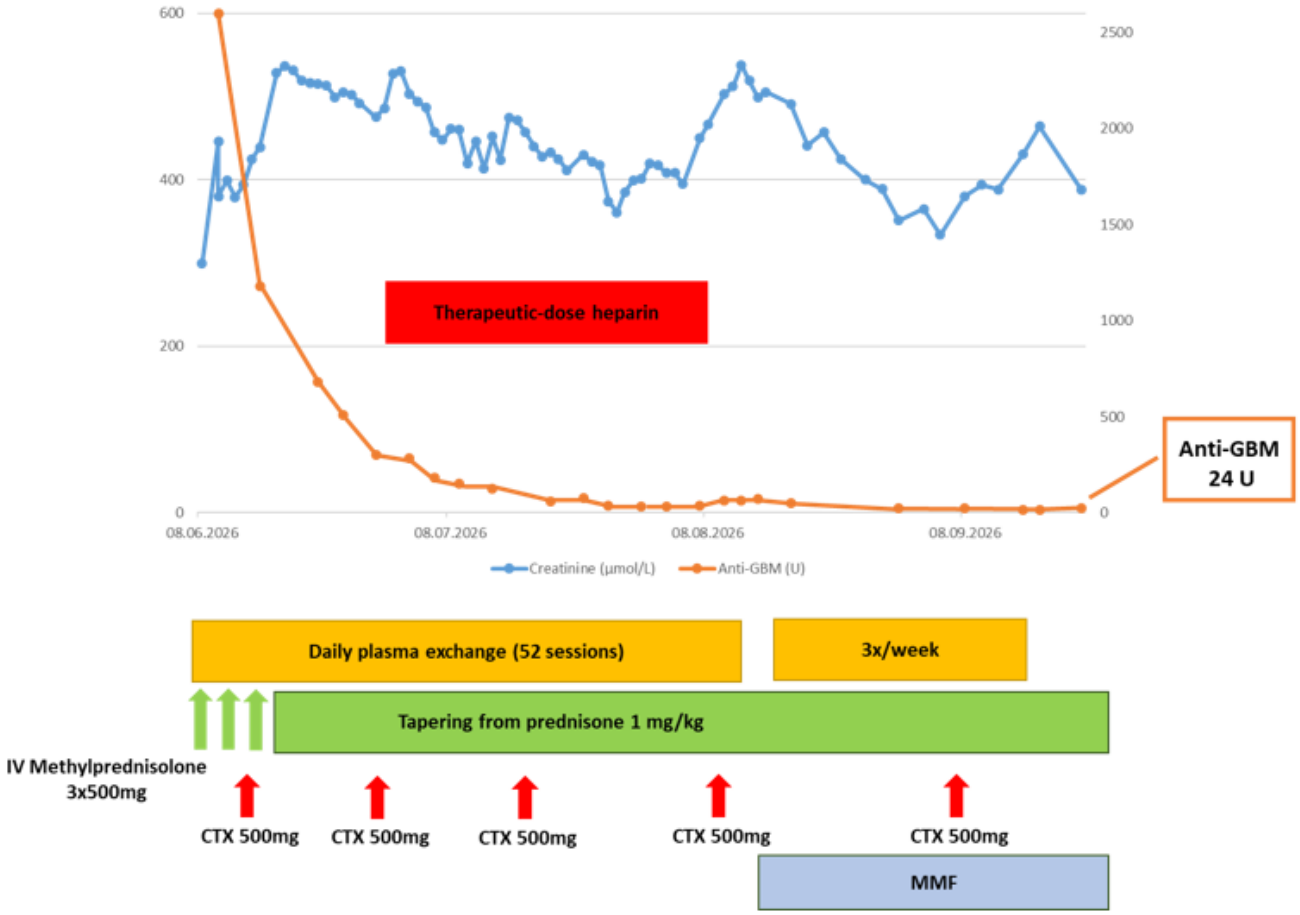



Figure 1
Evolution of renal function and anti-GBM antibody levels over 4 months of treatment. Corticosteroid therapy was initiated with three 500 mg pulses of methylprednisolone, followed by prednisone at 1 mg/kg with subsequent tapering. Daily plasma exchange was started immediately. Cyclophosphamide (CTX) was initiated on day 4 according to a 0-, 2-, 4-, 7-, and 10-week regimen, with plasma exchange withheld on the day following each administration. Therapeutic anticoagulation with heparin was initiated for catheter-related jugular vein thrombosis. After 10 weeks, in the setting of persistent anti-GBM antibodies, plasma exchange was temporarily discontinued to assess the subsequent antibody kinetics. As antibody levels increased, mycophenolate mofetil (MMF) was added, and plasma exchange was resumed three times weekly until anti-GBM antibody negativity was achieved.
*CTX: cyclophosphamide; MMF: mycophenolate mofetil.*

## EP-02

### Beyond thrombocytopenia: an atypical biopsy-proven gemcitabine-induced thrombotic microangiopathy with cellular crescents

Dr. Charlotte Storione[1], Dr. Nadine Ngatchou[1], Dr. Gregoire Arnoux[2], Dr. James TATAW ASHU[3]

*1. Hôpital de Moutier, Department of Internal Medicine and Nephrology, Moutier, 2. University of Geneva / Geneva University Hospitals, Department of Pathology, Geneva, 3. Faculty of Medicine, University of Geneva / Geneva University Hospitals, Geneva and Hôpital de Moutier, Department of Internal Medicine and Nephrology, Moutier*

**Background:** Gemcitabine-induced thrombotic microangiopathy (GiTMA) is a rare but severe complication of chemotherapy, usually associated with microangiopathic hemolytic anemia, thrombocytopenia, hypertension, and kidney injury. Renal-predominant presentations without thrombocytopenia may be diagnostically challenging.

**Case report:** A 68-year-old man with metastatic pancreatic adenocarcinoma received gemcitabine and nab-paclitaxel for eight months, reaching a cumulative gemcitabine dose of 37,440 mg. After approximately six months, serum creatinine progressively increased from 80–100 µmol/L to >250 µmol/L, accompanied by new-onset hypertension and severe anemia. Laboratory testing showed Coombs-negative microangiopathic hemolytic anemia, numerous schistocytes, and markedly decreased haptoglobin, but platelet counts remained normal. Interestingly, urinalysis demonstrated glomerular hematuria and proteinuria of approximately 1 g/day. Kidney biopsy revealed severe glomerular and arteriolar thrombotic microangiopathy (TMA), with mesangiolysis, double contours of the glomerular basement membranes, and fibrin thrombi (figure 1 Panels A,B

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and C). The tubulointerstitial compartment showed moderately severe and relatively diffuse acute tubular necrosis with red-cell casts. Two glomeruli also showed segmental cellular crescents, initially raising concern for concomitant crescentic glomerulonephritis (figure 1, panel D). Anti-neutrophil cytoplasmic antibodies (ANCA) and anti-glomerular basement membrane (anti-GBM) antibodies were negative. A disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13 (ADAMTS13) activity was preserved, and complement investigations were unremarkable. Gemcitabine was permanently discontinued. After a short corticosteroid course given for initially suspected pauci-immune glomerulonephritis, supportive management was continued. Serum creatinine subsequently improved from approximately 350 to 201 µmol/L.

**Conclusion:** GiTMA should be suspected in gemcitabine-treated patients with kidney dysfunction, hypertension, and microangiopathic hemolysis even when thrombocytopenia is absent. Cellular crescents may occur as part of severe TMA and should not automatically imply crescentic glomerulonephritis. Kidney biopsy can be decisive in atypical renal-predominant presentations.

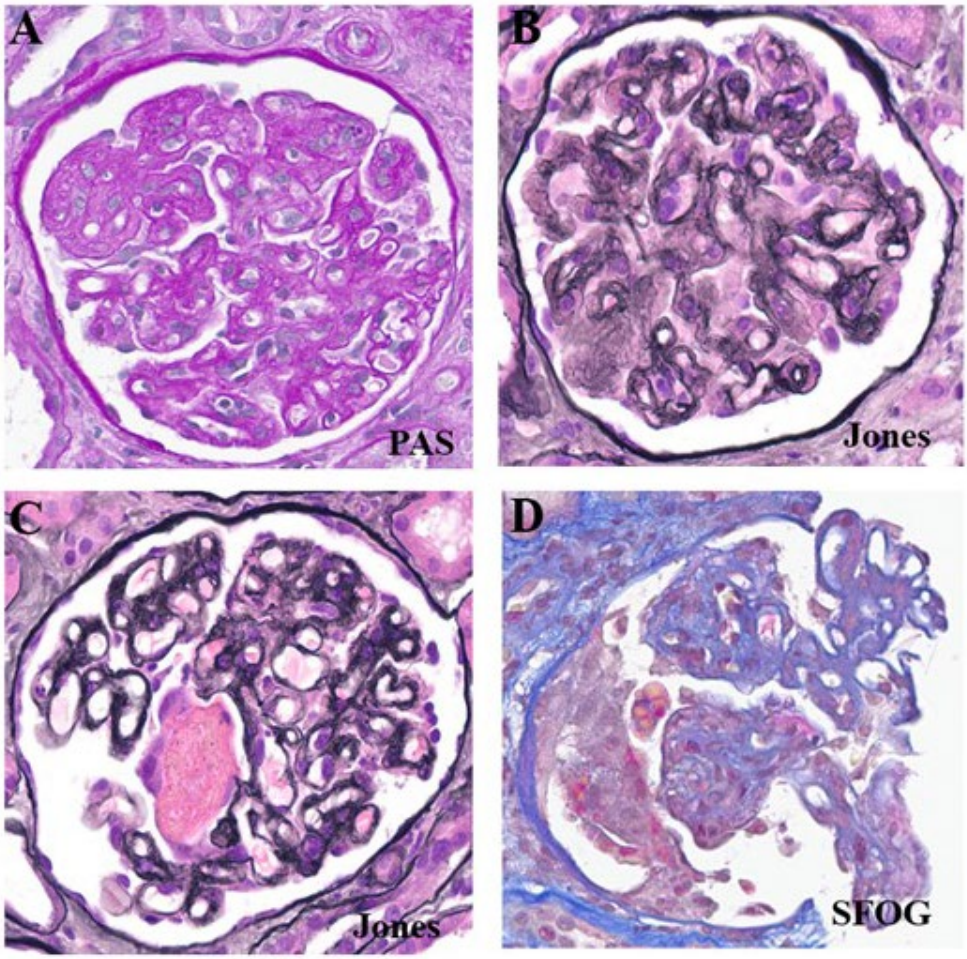



**Figure 1: Formalin-Fixed Paraffin-Embedded kidney biopsy on light microscopy with routine staining panel.** *Panel A and B show glomerular thrombotic microangiopathy with classic mesangiolysis, glomerular basal membrane duplication (A) and apparent thickening of the capillary wall due to subendothelial expansion (B). Panel C shows numerous thrombi along juxtaglomerular arterioles near the vascular pole of affected glomeruli. Occasional cellular crescents are observed, suggesting severe glomerular capillary wall damage (Panel D). PAS: Periodic Acid-Schiff; SFOG: Sour Fuchsin-Orange G, Jones: Methenamine Silver-Periodic-Acid-Schiff.*


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## EP-03

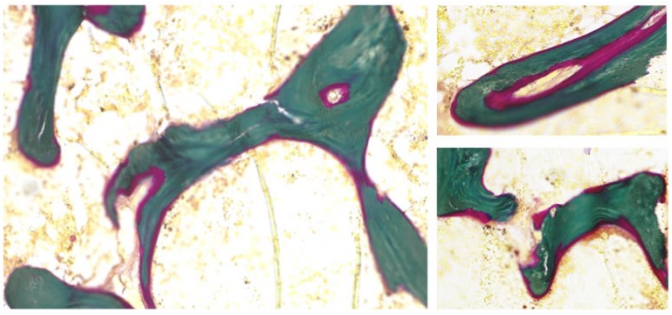
### Denosumab-associated osteomalacia in a patient undergoing hemodialysis

Dr. Alexander Orth[1], Dr. Maude Gerbaix[2], Prof. Marie-Hélène Lafage-Proust[3], Prof. Andrea Trombetti[2], Dr. Matthias Moor[1]

*1. Inselspital, Department of Nephrology, Bern, Switzerland, 2. Division of Bone Diseases, Department of Medicine, Geneva University Hospitals and Faculty of Medicine, Geneva, 3. INSERM UMR 1059 SAINBIOSE, Saint-Etienne*

A 67-year-old postmenopausal woman with systemic sarcoidosis leading to advanced kidney disease on regular hemodialysis, presented a challenging clinical case of recurrent fractures. Despite ongoing long term antiresorptive therapy with denosumab since September 2020, the patient experienced multiple fragility fractures including vertebral, rib, acetabular and a femoral neck fracture. Her medical history was notable for tertiary hyperparathyroidism treated with partial parathyroidectomy in 2016, chronic glucocorticoid exposure complicated by the development of type 2 diabetes mellitus in 2018 and intestinal malabsorption. Biomarkers showed recurrent low calcemia and phosphatemia, denosumab-associated spikes of parathyroid hormone, and elevated bone-specific alkaline phosphatase. Thus, differential diagnoses included severe osteoporosis, adynamic bone disease, inadequate treatment response and persistent disturbances of bone mineral metabolism with osteomalacia. CT-guided iliac crest biopsy revealed severe osteoporosis with osteomalacia (Figure). This case illustrates the complexity of fracture risk assessment, management of osteoporosis in advanced kidney disease patients and highlights the importance of bone biopsy to guide diagnosis and therapeutic approaches.

**Figure:** Modified Goldner Trichrome-stained non-decalcified bone biopsy.



## EP-04

### Long-term renal effects of palopegteriparatide for adults with hypoparathyroidism: pooled final data from the path forward and pathway trials

Dr. Karin Senn[1]

1. *Ascendis Pharma Switzerland GmbH*

**Background:** Palopegteriparatide is a prodrug of PTH (1-34) designed to provide active PTH within physiological range for 24 hours/day for the treatment of hypoparathyroidism. This post-hoc analysis assesses long-term impact of palopegteriparatide on renal function in the phase 2 PaTH Forward and phase 3 PaTHway trials.

**Methods:** Data from PaTH Forward and PaTHway, comprising randomized, double-blind, placebo-controlled periods through Weeks 4 and 26 and open-label extension periods through Weeks 266 and 182, respectively, were pooled by palopegteriparatide exposure duration. Renal function was assessed by eGFR. Safety assessments included 24-hour urine calcium excretion and treatment-emergent adverse events (TEAEs).

**Results:** Of 129 participants with complete data at final trial visits, 95% were independent from therapeutic doses of calcium (required ≤600mg/day elemental calcium), 98% independent from active vitamin D, and 88% normocalcemic (8.3-10.6mg/dL). Mean (SE) 24-hour urine calcium levels normalized (≤250mg/day) from baseline to Week 26, decreasing from 396.2mg/day (15.8mg/day) to 205.5mg/day (10.0mg/day) (P<0.0001) and stabilizing through Week 266. Mean (SE) eGFR increased from baseline to Week 26 by 7.2mL/min/1.73 m2 (0.9mL/min/1.73 m2) (P<0.0001), remaining stable with a baseline change of 9.4mL/min/1.73 m2 (1.9mL/min/1.73 m2) at Week 266 (P<0.0001). Annualized mean (SE) eGFR slope stabilized from Week 26 to Week 266 (0.6 [0.2]mL/min/1.73 m2/year; P = 0.0033). Most TEAEs were grade 1 or 2, with no new safety signals or new nephrolithiasis clinical episodes.

**Conclusions:** Palopegteriparatide treatment of adults with chronic hypoparathyroidism showed sustained normalization of 24-hour urine calcium excretion, improved renal function, and continued safety and efficacy through OLE completion of PaTH Forward and PaTHway.

## EP-05

### Not All Hemolysis Is TMA: A Hidden Sickle Cell Disease Presenting with Severe AKI

Dr. Rolf Dieterich[1], Dr. Federica Bocchi[2]

1. *Inselspital Bern, 2. Inselspital, Department of Nephrology, Bern, Switzerland*

**Background:** Thrombotic microangiopathy (TMA) is an important differential diagnosis in patients presenting with thrombocytopenia, hemolysis and acute kidney injury (AKI). However, other causes of hemolysis may mimic this constellation and require a fundamentally different diagnostic approach.

**Case presentation:** A 28-year-old man from the United Kingdom was transferred to our centre with suspected TMA after several days of deteriorating general condition, lower abdominal pain and one episode of diarrhoea with mild haematemesis. Laboratory testing revealed severe anaemia (Hb 57 g/L), thrombocytopenia (86 G/L), laboratory findings consistent with hemolysis, and severe AKI (creatinine 1249 µmol/L, urea >50 mmol/L). With a PLASMIC score of 4, TTP was considered less likely, while STEC-HUS and complement-mediated HUS remained differential diagnoses. ADAMTS13 testing was performed, and the patient received antibiotics and red blood cell transfusions. Haemodialysis was initiated the following day. A subsequent review of the peripheral blood smear revealed numerous sickle cells, target cells and Howell–Jolly bodies, providing a crucial diagnostic clue. ADAMTS13 activity was 55%, and stool testing for Shiga toxin-producing Escherichia coli was negative. Haemoglobin electrophoresis showed HbS 33.5%, HbF 1.9% and HbA2 3.0%; interpretation was limited by prior red blood cell transfusions. Nevertheless, the positive sickling test, characteristic blood smear and an atrophic spleen supported the diagnosis of previously unrecognized sickle cell disease. A vaso-occlusive crisis was presumed based on the clinical and sonographic findings. The patient remained dialysis-dependent at discharge for continued treatment in his home country.

**Conclusion:** This case highlights the importance of maintaining a broad differential diagnosis in patients presenting with hemolysis, thrombocytopenia and severe AKI. Careful examination of the peripheral blood smear provided the key diagnostic clue and redirected the diagnostic work-up away from TMA. Previously unrecognized sickle cell disease should be considered in appropriate patients presenting with severe AKI and hemolysis.


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## EP-06

### Novel potassium binders for enabling mineralocorticoid receptor antagonist therapy in adults with chronic kidney disease and eGFR below 45 mL/min/1.73 m²: a systematic review and structured quantitative synthesis

Ms. Stephanie Bull-Larsen[1], Prof. Jörg Latus[2], Dr. Jan Brügger[3]

*1. Universität Zürich, 2. Robert-Bosch-Krankenhaus, 3. nephro.swiss AG, Centre for nephrology and dialysis, Horgen*

**Background:** Hyperkalaemia frequently limits initiation, up-titration, or maintenance of indicated mineralocorticoid receptor antagonists (MRAs) as kidney function declines. We evaluated whether patiromer or sodium zirconium cyclosilicate (SZC) enables or sustains MRA therapy in adults with chronic kidney disease and estimated glomerular filtration rate (eGFR) <45 mL/min/1.73 m².

**Methods:** MEDLINE, Embase, CENTRAL, registries, conference sources, and citation networks were searched through 3 June 2026. Randomised trials required an MRA-specific result for the target population. The first and last authors screened independently. Cochrane RoB 2 and GRADE informed risk of bias and certainty. Native effects were retained because trial estimands were non-exchangeable. No pooled effect was calculated. PROSPERO CRD420261362223.

**Results:** Four reports from three trials contributed 561 eligible participants. In AMBER, patiromer increased spironolactone persistence at week 12 (126/147 versus 98/148), with a risk ratio of 1.29 (95% CI 1.13 to 1.48) and a number needed to treat for benefit of 6 (95% CI 4 to 10). In the prespecified descriptive DIAMOND eGFR <45 subgroup, patiromer reduced the rate of sustained reduction below the protocol-defined MRA target (adjusted cause-specific hazard ratio 0.48, 95% CI 0.28 to 0.81). In the prespecified exploratory REALIZE-K eGFR <45 subgroup, SZC increased the odds of repeated optimal response (odds ratio 11.27, 95% CI 4.36 to 29.18). Certainty was moderate for AMBER and low for both subgroup results. In exploratory full-trial REALIZE-K data, cardiovascular death or worsening heart failure occurred in 11/102 with SZC and 3/101 with placebo, but causality was not established.

**Conclusions:** Across three complementary clinical scenarios, binder therapy favoured each trial-defined MRA outcome. Potassium binders may help selected patients initiate or sustain an otherwise indicated steroidal MRA when hyperkalaemia is the main modifiable barrier. The evidence does not establish binder superiority or downstream patient-important benefit. Larger trials using comparable outcomes are needed.

## EP-07

### Obinutuzumab in multidrug-resistant FSGS: a single-centre case series

Dr. Tabea Vogel[1], Dr. Philipp Grosse[1], Dr. Kathrin Fausch[1], Dr. Reto Venzin[1], Dr. Argyrios Georgalis[1]

1. *Department of Nephrology, Cantonal Hospital Graubunden, Chur*

**Background:** Resistant FSGS has a poor prognosis; prolonged high-dose glucocorticoids are poorly tolerated. Obinutuzumab, a type II anti-CD20 antibody, induces deeper B-cell depletion than rituximab.

**Patients:** Three patients with biopsy-proven FSGS with diffuse foot process effacement and negative genetic testing received obinutuzumab (1 g/dose) after failure of multiple immunosuppressive regimens.

**Results:** Case 1 (61-year-old woman, steroid-dependent course): no partial remission after rituximab (2 × 1 g) plus tacrolimus. Two months after an additional rituximab dose, severe relapse (proteinuria 11 g/d, albumin 17 g/L) despite complete CD19+ depletion and tacrolimus troughs 5–10 ng/mL. After one obinutuzumab dose with prednisone 60 mg/d (then 15 mg/d), proteinuria decreased to 3.5 g/d (−68%) and albumin rose to 32 g/L by month 2, with stable eGFR (67 mL/min/1.73 m²). The patient was subsequently transferred to another centre. Case 2 (39-year-old woman, 7-year steroid-dependent course): relapse (proteinuria 8 g/d, albumin 25 g/L) under tacrolimus and low-dose prednisone. Obinutuzumab given at weeks 0, 2, 26 and 52 with prednisone ≤10 mg/d. At month 15: proteinuria 2.6 g/d (−68%), albumin 38 g/L, stable eGFR (70 mL/min/1.73 m²), tacrolimus troughs reduced to 3–5 ng/mL by month 5. Case 3 (61-year-old man, sarcoidosis with hilar/mediastinal lymphadenopathy, no granulomatous interstitial nephritis): after glucocorticoids and tacrolimus, proteinuria fell from 10 to 4.5 g/d (albumin 18 to 33 g/L), but eGFR declined from 60 to 35 mL/min/1.73 m². Obinutuzumab (weeks 0, 2, 26, 28) without glucocorticoids allowed tacrolimus withdrawal by month 6 after previous reduction. At month 12: proteinuria 3 g/d, albumin 38 g/L, eGFR 30 mL/min/1.73 m². No adverse events occurred. Updated data will be presented.

**Conclusion:** Obinutuzumab reduced proteinuria, improved serum albumin and allowed CNI and glucocorticoid reduction or withdrawal, even after rituximab failure and with low-dose or no glucocorticoids. Obinutuzumab appears promising; larger series are needed.

## EP-08

### Off-label Tolvaptan for persistent marked hyperoxaluria after kidney transplantation in primary hyperoxaluria type 2

Dr. Thomas Reich[1], Dr. Martin Scoglio[2], Dr. Matteo Bargagli[2], Prof. Daniel Sidler[3], Prof. Daniel Fuster[4]

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**Background:** Primary hyperoxaluria (PH) is a rare cause of end-stage kidney disease. After kidney transplantation, mobilization of systemic oxalate stores may result in substantial oxalate deposition within the allograft, thereby contributing to graft dysfunction. For PH type 1, RNA interference (RNAi) therapies and combined liver–kidney transplantation effectively reduce hepatic oxalate production. In contrast, no effective therapies exist for PH type 2. Tolvaptan, a vasopressin V2-receptor antagonist approved for the treatment of ADPKD, increases urine volume and may thereby reduce urinary supersaturation, stone formation and renal oxalate deposition. To our knowledge, only one case of tolvaptan use after kidney transplantation has been reported in a patient with PH type 1 receiving concomitant RNA interference therapy. We report the successful off-label use of tolvaptan in a kidney transplant recipient with severe PH type 2.

**Case description:** A 55-year-old man had recurrent calcium oxalate nephrolithiasis requiring multiple urologic interventions since 2000. In 2015, primary hyperoxaluria type 2 was confirmed by identification of a homozygous pathogenic *GRHPR* variant. Hemodialysis was initiated in 2018 and successful kidney transplantation was performed in November 2024. Soon after transplantation, allograft function deteriorated rapidly and a biopsy showed extensive calcium oxalate crystal deposition. Tolvaptan was initiated in March 2025. Urine volume increased from 2.3 to 4.6 liters per day despite persistent severe hyperoxaluria, with urinary oxalate excretion of up to 2.3 mmol per 24 hours. Three subsequent allograft biopsies after treatment start with Tolvaptan showed only minimal oxalate deposition. Two

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years after transplantation, kidney allograft function remains stable and well preserved.

**Conclusion:** Tolvaptan induced sustained aquaresis and was associated with a marked reduction in calcium oxalate deposition in the kidney allograft despite persistent, severe hyperoxaluria. To our knowledge, this is the first report of successful tolvaptan use after kidney transplantation in a patient with primary hyperoxaluria type 2.

## EP-09

### Rare types of kidney stones: a case of a 1-methyluric acid kidney stone nephropathy

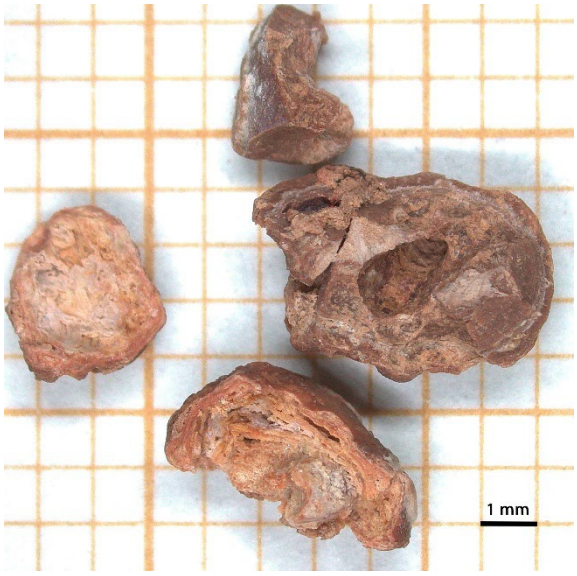
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1. *Service of Nephrology and Hypertension. Lausanne University Hospital and University of Lausanne, Lausanne, Switzerland.*, 2. *Diagnostic Laboratory, University Hospitals of Geneva, Geneva, Switzerland*

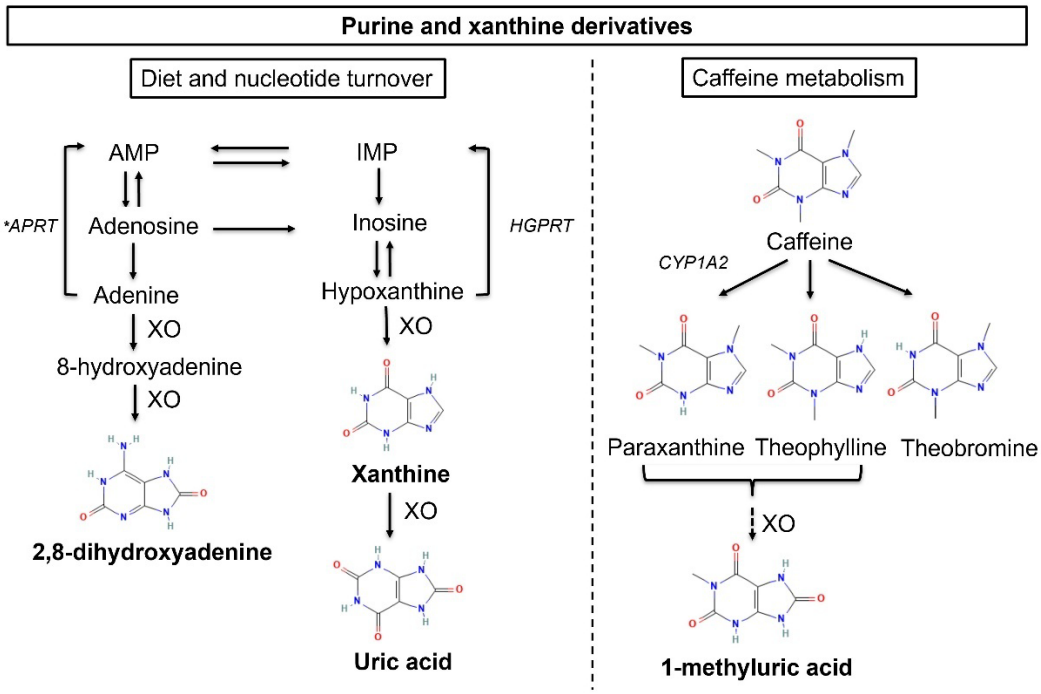
**Background:** Coffee, one of the most widely consumed beverages worldwide, contains caffeine, chemically known as 1,3,7-trimethylxanthine. Caffeine is metabolized by CYP1A2 in the liver to paraxanthine, theophylline and theobromine and then subsequently converted by xanthine oxidase into 1-methyluric acid (1-MUA), the main caffeine derivate found in urine (Figure 1).

**Case presentation:** A 60-year-old Caucasian woman, active smoker, with a history of rheumatoid arthritis, heart disease and esophageal leiomyoma, who presented with flank pain due to multiple kidney stones. She reported long-standing, heavy coffee intake (10 ristretto daily) and low water intake. Stone infrared spectroscopic analysis revealed an uncommon composition of 100% 1-MUA, an orange-brown stone with a smooth surface and concentric layers cross-section (Figure 2). The episode was complicated by acute kidney injury (AKI) and kidney function did not fully recover (eGFR 50ml/min/1.73m$^2$).

**Discussion:** Most kidney stones consist of calcium oxalate (85%), calcium phosphates (10%) or uric acid (5%) but rare forms are related to purine and xanthine metabolism include 2,8-dihydroxyadenine, xanthine stones and 1-MUA (Table 1). 1-MUA lithiasis is a very rare cause linked to heavy coffee consumption. A large French cohort identified only 20 such cases over the past 40 years. Few reported cases included 3 cases of 1-MUA nephropathy with AKI and tubular crystal deposition on kidney biopsy, and 1 case had mixed stone-composition of 50% 1-MUA in a heavy coffee drinker. The increased risk of AKI/chronic kidney disease suggests a mechanism beyond obstruction, involving direct nephrotoxicity from tubular 1-MUA crystals precipitation in susceptible individuals. Moreover, half-life of caffein metabolite may be prolonged by CYP1A2 inhibition due to genetic polymorphism or common drugs (contraceptives, antidepressants, opioids) ore shortened in case of smoking. Management consists of hydration, caffeine withdrawal and allopurinol. This case highlights the value of stone composition analysis in atypical nephrolithiasis, as identifying directly informs management.



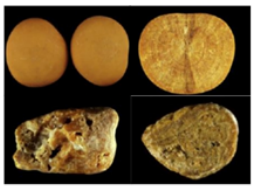
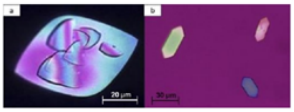
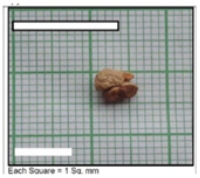
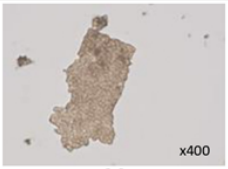
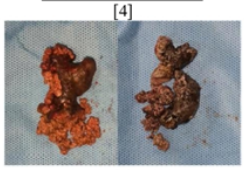
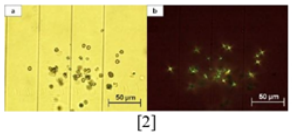
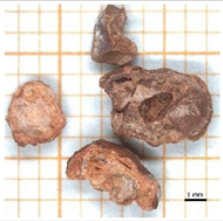
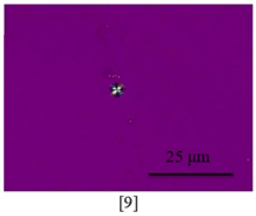

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Table 1 : Purine and xanthine derived kidney stones

| Kidney stone and epidemiology | Macroscopic aspect | Microscopic aspect | Causes | Management |
|---|---|---|---|---|
| **Uric acid (anhydrous and/or dihydrate)**<br>5-10% | <br>[1] | <br>Uric acid dihydrate crystals<br>[2] | **Low urine pH:**<br>• Metabolic syndrome<br>• Type 2 diabetes mellitus<br>• Intestinal alkali loss in patients having chronic diarrhea<br>**High excretion of uric acid:**<br>• Diabetes mellitus<br>• Myelo or lymphoproliferative syndromes<br>• Vaquez disease<br>• Tubular dysfunction inducing a defect in urate reabsorption<br>**Stasis prostate hypertrophy**<br>**Ammoniagenesis defect**<br>**High production and excretion of uric acid from diet origin :**<br>• High fructose intake<br>• Nucleo-protein rich food<br>• Genetic diseases on the nucleotide pathways (Lesh–Nyhan syndrome, phosphor-ribosyl-pyrophosphate synthetase hyperactivity) | • High fluid intake<br>• Urine alcalinization (pH> 6.5)<br>• Dietary purine restriction<br>• Allopurinol or febuxostat |
| **Xanthine**<br>rare | <br>[6] | <br>[7]<br>Specificity : refringent granulations or sticks without a characteristic aspect and Fourier transform infrared analysis is required for accurate identification [2] | • Homozygous xanthine deshydrogenase deficiency (familial xanthinuria) [8]<br>• Treatment of Lesh-Nyhan syndrome by high doses of allopurinol | • High fluid intake<br>• Dietary purine restriction |
| **2,8-dihydroxyadenine**<br>rare<br>[3] | <br>[4]<br><br>[5]<br>(65% 2.8 DHA)<br>Specificity: Do not react with uricase | <br>[2] | Defect of APTR<br>(adenine phosphoribosyltransferase) | • High fluid intake<br>• Hypothesis: dietary purine restriction |
| **1-methyluric acid**<br>rare |  | <br>[9] | • High caffeine consumption<br>• Potentialized by drugs, genetic polymorphism of CYP1A2, CKD<br>• hypothesis: aluminium containing drugs | • High fluid intake<br>• Coffee and tea restriction (n=2)<br>• Allopurinol (n=1)<br>• Hypothesis : urine alkalinization, eviction of aluminium-containing drug |

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## EP-10

### Resilience in patients with Fabry Disease and its association with disease course, psychosocial factors and quality of life: a multicentre cross-sectional study

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**Background:** Fabry caFabry disease (FD) is an X-linked lysosomal storage disorder originating from mutations in the GLA-gene causing deficiency in alpha-Galactosidase A (GLA) which leads to the accumulation of glycosphingolipids, particularly globotriaosylceramide (Gb3) and globotriaosylsphingosine (Lyso-Gb3). The classic phenotype is characterized by neuropathic pains and pain crises, anhidrosis, abdominal cramping. With advancing age, the disease leads to life-threatening complications such as renal dysfunction, left ventricular hypertrophy, hypertrophic cardiomyopathy or cryptogenic stroke. These debilitating symptoms are associated with depression and impaired quality of life. Resilience is known as an individual's ability of coping with stressors and is characterized as high self-esteem, optimism and self-efficacy. Resilience enhancement programmes were shown to have a benefit on resilience, depression, anxiety and quality of life.

**Methods:** 122 genetically confirmed FD patients from the university hospitals of Zurich Switzerland and Mainz Germany completed self-report questionnaires Resilience Scale 11 (RS-11), Beck Depression Inventory (BDI), Pain Disability Index (PDI), Pittsburgh Sleep Quality Index (PSQI), Trierer Inventory for Chronic (TICS-SSCS).

**Results:** Women indicated higher resilience scores than men (59 ± 14.4 vs 54.4 ± 19.2) and patients without enzyme replacement therapy (ERT) higher than on ERT (64.5 ± 15.9 vs 55.8 ± 16.5). Among females, resilience was higher if the FD diagnose was made at younger age (r = -0.40, p = 0.02), while resilience declined with increasing age (r = -0.26, p = 0.04). More symptomatic patients showed lower resilience scores (r = -0.24, p = 0.01). The RS-11 showed a significant adverse correlation with BDI (r = -0.59; p = 0.001), PDI (r = -0.31, p = 0.006), PSQI (r = -0.53, p = 0.001) and TICS-SSCS (r = -0.42, p = 0.001).

**Conclusions:** Our analyses show that impaired resilience is associated with depression, pains, poor sleep and chronic stress. All these factors are modifiable and treatable. Importantly, resilience enhancement programs should be offered to Fabry patients.

## EP-11

### Secondary oxalate nephropathy in a kidney allograft revealing previously unrecognised enteric hyperoxaluria

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**Background:** Oxalate nephropathy is an uncommon and often underrecognised cause of kidney injury. Enteric hyperoxaluria may result from fat malabsorption, including exocrine pancreatic insufficiency. We report a case of secondary oxalate nephropathy first diagnosed in the kidney allograft during evaluation of dialysis-requiring acute kidney injury initially attributed to repeated iodinated contrast exposure.

**Case description:** A male patient underwent deceased-donor kidney transplantation in March 2026 for kidney failure of uncertain aetiology, clinically attributed to diabetic and hypertensive nephropathy. A native kidney biopsy had never been performed. Initial allograft function was satisfactory and a 1-month protocol biopsy showed no rejection. The patient subsequently underwent seven iodinated contrast exposures for severe peripheral arterial disease. Allograft function subsequently deteriorated with creatinine reaching 516 µmol/L and intermittent haemodialysis becoming necessary. Contrast-associated acute tubular injury and rejection were the leading differential diagnoses. Allograft biopsy demonstrated no signs of rejection but acute tubular injury with numerous intratubular oxalate crystals. Plasma oxalate was 38.83 µmol/L and 24-hour urinary oxalate was markedly elevated at 1,318 µmol/24 h. Genetic testing prompted by values compatible with primary hyperoxaluria identified no pathogenic variants. Faecal elastase of <6 µg/g revealed severe exocrine pancreatic insufficiency, supporting secondary enteric hyperoxaluria. Pancreatic enzyme replacement was initiated.

**Conclusion:** Repeated contrast exposure provided a plausible explanation for acute allograft dysfunction but did not account for the prominent oxalate crystal deposition. The diagnosis of secondary enteric hyperoxaluria due to severe exocrine pancreatic insufficiency revealed a potentially treatable contributor to kidney injury. In the absence of native kidney histology, a contribution of previously unrecognised oxalate nephropathy to the original kidney disease cannot be excluded.

## EP-12

### Simultaneous appearance of IgA nephropathy and tubulointerstitial nephritis and uveitis (TINU) syndrome: a case report

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**Background:** IgA nephropathy is the most common primary glomerulonephritis, typically following upper respiratory tract infection. TINU syndrome is a rare condition of suspected autoimmune origin, combining tubulointerstitial nephritis and uveitis. Concurrent manifestation of both entities is extremely rare, with only a few cases reported in the literature.

**Case Presentation;** A 53-year-old woman presented with 1 month of postprandial nausea, vomiting, occipital headache, 5 kg weight loss, and night sweats. Laboratory findings revealed normocytic anemia, eosinophilia, hypoalbuminemia, and moderately impaired renal function (creatinine 166 µmol/l, eGFR 30 ml/min/1.73 m²) with glomerular hematuria and mixed proteinuria. During hospitalization, she developed left-sided anterior uveitis. Kidney biopsy demonstrated focal segmental necrotizing glomerulonephritis with crescents (3 of 13 glomeruli) and severe acute tubulointerstitial nephritis. Immunohistochemistry revealed granular mesangial IgA deposits. Secondary causes including ANCA-associated vasculitis, lupus nephritis, sarcoidosis, and Behçet's disease were systematically excluded. *Helicobacter pylori* gastritis and *Strongyloides* infection were additionally identified. Based on clinical, laboratory, and histopathological findings, concurrent IgA nephropathy and TINU syndrome were diagnosed. Treatment with intravenous methylprednisolone (250 mg/day for 2 days) followed by oral prednisolone (1 mg/kg/day) resulted in rapid improvement of renal function (creatinine 84 µmol/l, eGFR 69 ml/min/1.73 m² after 3 weeks). Nephroprotective therapy with a RAS blocker and an SGLT-2 inhibitor was initiated. Renal function remained stable at 26 months of follow-up (eGFR 69 ml/min/1.73 m²), with significant reduction of proteinuria.

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**Conclusion:** This case highlights the rare simultaneous occurrence of IgA nephropathy and TINU syndrome, potentially triggered by *Helicobacter pylori* and *Strongyloides* infection. Glucocorticoid therapy led to rapid and sustained renal recovery. Further research is warranted to elucidate the shared pathogenesis and the role of infectious triggers in both conditions.

## EP-13

### Total versus Ionized Magnesium in Chronic Kidney Disease and Dialysis

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**Background:** Total magnesium (tMg) is routinely used to assess magnesium status, whereas ionized magnesium (iMg) represents the biologically active fraction. Whether CKD and dialysis alter the relationship between tMg and iMg and whether iMg provides additional value beyond tMg remains uncertain.

**Methods:** We enrolled 334 participants: 185 with CKD, 84 receiving hemodialysis (HD), 12 receiving peritoneal dialysis, and 53 controls. Reference ranges were 0.66-0.91 mmol/L for tMg and 0.42-0.68 mmol/L for iMg. Dialysate Mg concentrations were 0.5 or 0.75 mmol/L. tMg, iMg, and iMg/tMg ratio were compared using robust linear regression. Adjusted models included demographic, clinical, and biochemical covariates.

**Results:** Among participants with CKD, hypomagnesemia was identified in 7.6% (14/185) by tMg and 6.5% (12/185) by iMg. Classification of hypomagnesemia was discordant in 37.5% of participants: 28.6% of those with low tMg had normal iMg, whereas 16.7% of those with low iMg had normal tMg. Among patients receiving HD, hypermagnesemia was identified in 86.9% (73/84) by tMg and 51.2% (43/84) by iMg (exact McNemar $P<.001$). Classification of hypermagnesemia was discordant in 41.1% of participants with all discordant cases had elevated tMg with normal iMg, but not the opposite pattern. Compared with controls, adjusted models showed lower tMg (β = -0.076, 95%CI -0.123 to -0.029) and iMg (β = -0.067, -0.101 to -0.033) in CKD with an attenuated iMg/tMg ratio difference (β = -0.023, p = 0.059), but higher tMg (β = 0.303, 0.125–0.480) and iMg (β = 0.138, 0.030 - 0.246) in HD with a lower ratio (β = -0.055, -0.096 to -0.015).

**Conclusions:** tMg and iMg showed broadly parallel associations. However, in CKD, although hypomagnesemia was uncommon, discordance was substantial when either measure was low. In HD, iMg classified substantially fewer participants as hypermagnesemic than tMg, suggesting that iMg may provide complementary information not captured by tMg alone.

## EP-14

### What does it even mean today? - The diagnosis of the syndrome of inappropriate antidiuresis (SIAD) in a contemporary, real-world cohort

Dr. Florian Buchkremer[1]

1. *NephroCare, Dialysepraxis A. Staub AG, Luzern*

**Background:** The syndrome of inappropriate antidiuresis (SIAD) is often considered the most common cause of hypotonic hyponatremia. Since its first clinical description in 1957, its definition has evolved considerably. The current meaning and purpose of SIAD as a clinical diagnosis is ill-defined. This study provides a fresh look at SIAD in a large, contemporary cohort of hospitalized patients.

**Methods:** Data were extracted from MIMIC-IV, an electronic health record dataset containing hospital admissions from more than 350’000 patients to the Beth Israel Deaconess Medical Center, a teaching hospital of Harvard Medical School in Boston. All patients with an ICD-10 diagnosis of SIAD (E22.2) were included.

**Results:** Overall, MIMIC-IV contained 2’039 patients and 2’523 hospital admissions with an ICD-10 diagnosis of SIAD. 317 patients had more than one hospital admission with SIAD. Among the 2’039 first time SIAD admissions, SIAD was the main hospital diagnosis in 16%. Hyponatremia was mild in 18%, moderate in 45%, and severe in 37%. In-hospital mortality was 5% and 40% of patients were treated in the intensive care unit. At discharge, 52% of patients were still hyponatremic and recurring hyponatremia within a year was demonstrated in an additional 18%. Antidiuresis had been confirmed in only 83% of patients. Applying guideline recommended criteria would have in fact excluded SIAD in at least 14%. Other risk factors of hyponatremia, unrelated to inappropriate antidiuresis, were present in almost two thirds of patients (65%).

**Conclusions:** In this large, contemporary, real-world cohort, patients with a diagnosis of SIAD required significant hospital resources and had a very high rate of hyponatremia during follow-up. Notably, many patients did not meet formal criteria for making a diagnosis of SIAD and unrelated risk factors of hyponatremia were present in the majority of patients.

## EP-15

### When BCG Goes Beyond the Bladder: Suspected Disseminated BCG Infection With Severe AKI

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**Background:** Intravesical Bacillus Calmette–Guérin (BCG) therapy is an established treatment for non-muscle-invasive bladder cancer. Although uncommon, systemic BCG-related complications can be severe and may involve the kidneys. Recognizing BCG-associated renal disease can be challenging, particularly in the absence of microbiological confirmation.

**Case presentation:** A 72-year-old man with newly diagnosed urothelial carcinoma of the bladder received two intravesical BCG instillations. 5 days after the second instillation, he presented with systemic symptoms and was initially treated with ciprofloxacin for suspected left-sided pyelonephritis based on computed tomography (CT) findings. Despite antibiotic therapy, he developed sepsis requiring escalation to meropenem and intensive care treatment, accompanied by severe acute kidney injury (AKI KDIGO stage 3). Given the temporal association with BCG therapy, BCG-associated granulomatous nephritis was suspected. Following transfer to our tertiary care centre, repeat CT imaging demonstrated progressive bilateral renal abnormalities suggestive of granulomatous nephritis. Concomitant hepatitis further raised suspicion of disseminated BCG infection. Anti-mycobacterial treatment with rifampicin, ethambutol and moxifloxacin was initiated. Microbiological cultures have remained negative to date. The patient has remained dialysis-dependent since 8 August 2026, and the clinical course is ongoing.

**Conclusion:** This case highlights the diagnostic challenge of suspected disseminated BCG infection presenting with severe AKI and progressive renal imaging abnormalities. In the absence of microbiological confirmation, the diagnosis relies on the integration of the temporal relationship to BCG therapy, systemic manifestations and characteristic radiological findings.

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Awareness of this rare complication is essential, as early recognition of BCG-associated renal involvement may allow timely treatment and potentially prevent irreversible kidney injury.

## EP-16

### When typical infection-associated glomerulonephritis unmasks C3 glomerulonephritis: Targeting the alternative complement pathway with iptacopan – a case report

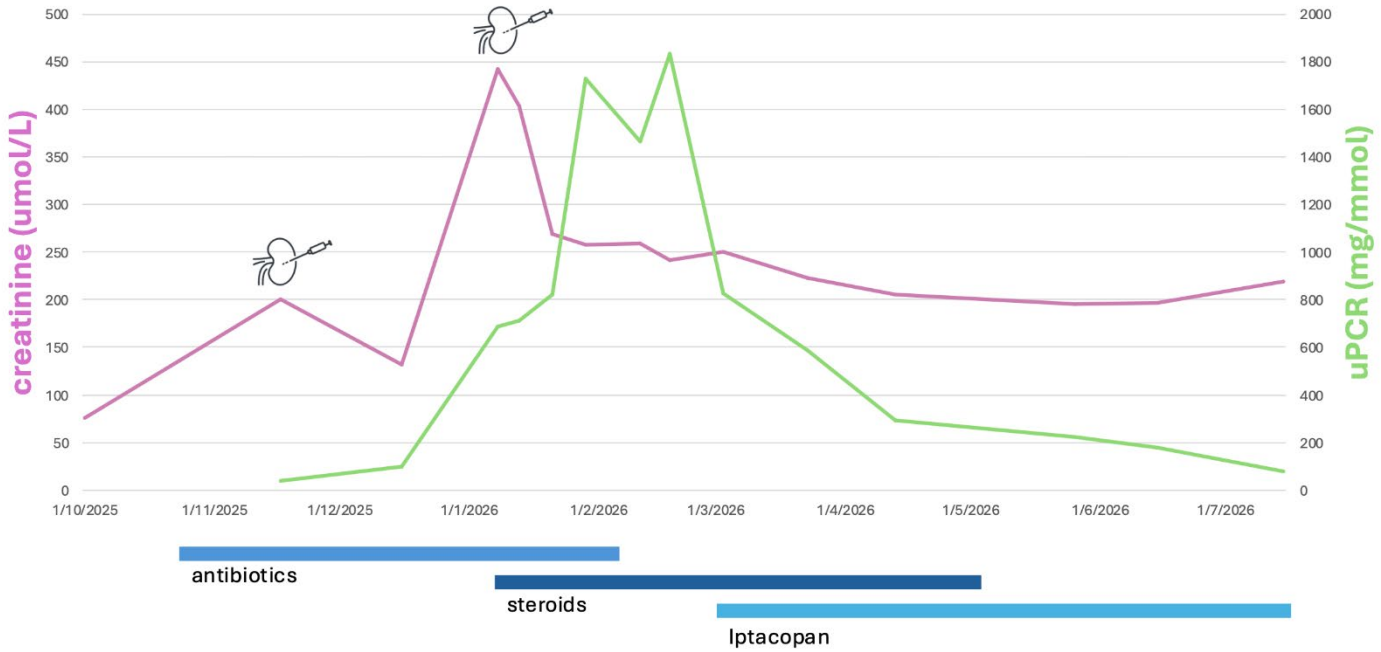
Ms. Mateja Kraljevic[1], Dr. Thomas Menter[2], Dr. Helmut Hopfer[2], Prof. Patricia Hirt-Minkowski[1]

1. *Clinic for Transplantation Immunology and Nephrology, University Hospital Basel, Basel, 2. Institute of pathology, University of Basel, Basel*

**Background:** Infection-associated glomerulonephritis is typically characterized by immune complex deposition in the setting of an active or recent infection. C3 glomerulonephritis (C3GN) is defined by glomerular C3 deposition and represents a complement-mediated glomerulopathy resulting from dysregulation of the alternative complement pathway (AP). Substantial clinicopathological overlap exists between so-called "atypical" infection-associated glomerulonephritis – characterized by persistent or worsening glomerular disease despite resolution of the infection – and C3GN. Persistent or progressive disease after resolution of the infection may indicate dysregulation of the AP. While treatment of classical infection-associated glomerulonephritis primarily focuses on eradication of the underlying infection, persistent complement-mediated disease may warrant consideration of targeted inhibition of the AP.

**Case Description:** A 79-year-old patient developed acute kidney injury, hematuria, and proteinuria in the context of *Staphylococcus aureus* bacteremia after spinal surgery. The initial kidney biopsy showed infection-associated glomerulonephritis with endocapillary proliferation and subepithelial deposits ("humps"). Despite adequate treatment of the infection, two months later, the patient progressed to kidney failure and nephrotic-range proteinuria up to 18 g/day. A second kidney biopsy showed dominant C3 staining with minimal immunoglobulin deposition on immunofluorescence microscopy, suggestive of C3GN. Complement analysis showed a slightly reduced serum C3 concentration and low factor B levels. Despite three months of high-dose steroids, nephrotic-range proteinuria persisted. Iptacopan, a factor B inhibitor of the AP, resulted in a reduction in proteinuria and stabilization of kidney function (figure 1).

**Conclusion:** This case illustrates a rapid shift from infection-associated glomerulonephritis to a C3-dominant glomerulopathy. The preceding infection may have unmasked AP dysregulation. The response to iptacopan is consistent with a pathogenic role of AP activation. These findings suggest that infection-associated glomerulonephritis may represent the initial manifestation of an underlying complement-mediated disease. Persistent or progressive kidney abnormalities despite resolution of infection should prompt consideration of repeat kidney biopsy and complement evaluation.




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## YSN AWARD (PECHA KUCHA)

### YSN-01

### Diagnostic algorithm based on urinary CXCL10 for detection of rejection and infection at one-year posttransplant

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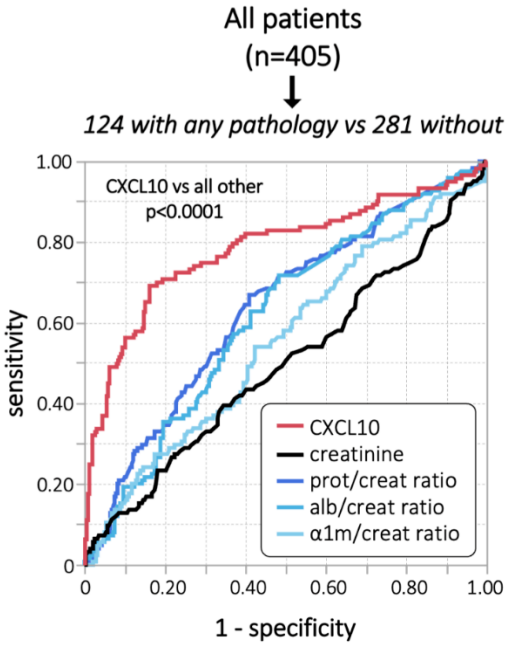
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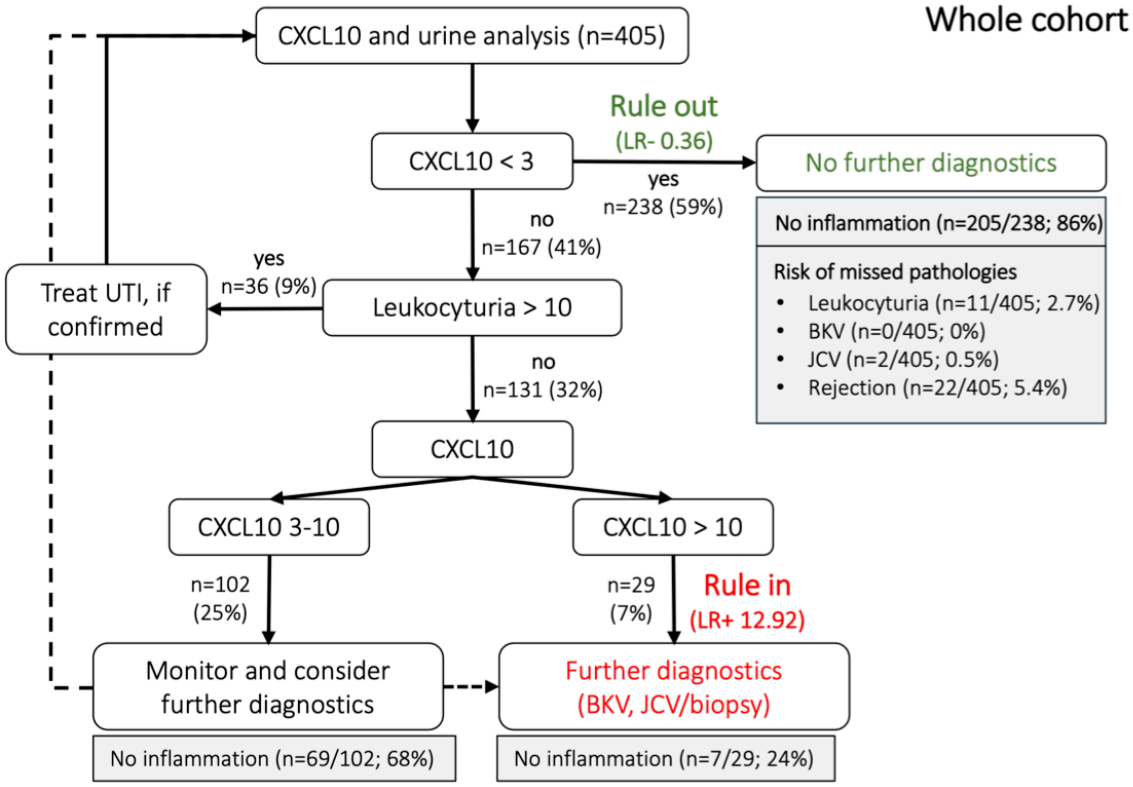
**Background:** Urinary CXCL10 chemokine levels correlate with inflammatory processes in the reno-urinary tract after kidney transplantation. With this study we wanted to investigate the clinical utility of urinary CXCL10 as a noninvasive biomarker for detection of major inflammatory processes (i.e. rejection, urinary tract infection, BKV- and JCV-DNAuria) in stable renal allografts and to develop a simple diagnostic algorithm to guide subse-quent analyses.

**Methods:** 405 stable renal allograft recipients underwent a surveillance biopsy one-year posttransplant. Additionally, leukocyturia, BKV- and JCV-DNAuria as well as urinary CXCL10 at the same time were measured.

**Results:** Urinary CXCL10 had a significantly higher AUC than serum creatinine and proteinuria (0.786 vs 0.499 vs 0.636; both p<0.0001) for detection of inflammatory processes. The highest AUC was observed for detection of BK polyomavirus DNAuria (0.936), but it was lower for detection of subclinical rejection (0.703). We defined a low, rule-out cutoff (<3ng/mmol; 59% of all patients; negative likelihood ratio 0.36) and a high, rule-in cutoff (>10ng/mmol; 12% of all patients; positive likelihood ratio 12.92). Next, we developed a stepwise diagnostic algorithm primarily based on urinary CXCL10 and leukocyturia. At the rule-out cutoff, 51% of surveillance biopsies could be safely deferred, and the risk of subclinical rejection decreased from 15.8% to 5.4%. At the rule-in cutoff, absence of any inflammatory pathologies was only 2%.

**Conclusions:** These data support urinary CXCL10 measurements at one-year posttransplant in stable allografts to guide further diagnostics.





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## YSN-02

### Impact of Using Different eGFR Equations on the Prevalence of Chronic Kidney Disease in the General Swiss Population (SSS2-CKD Prevalence Study)

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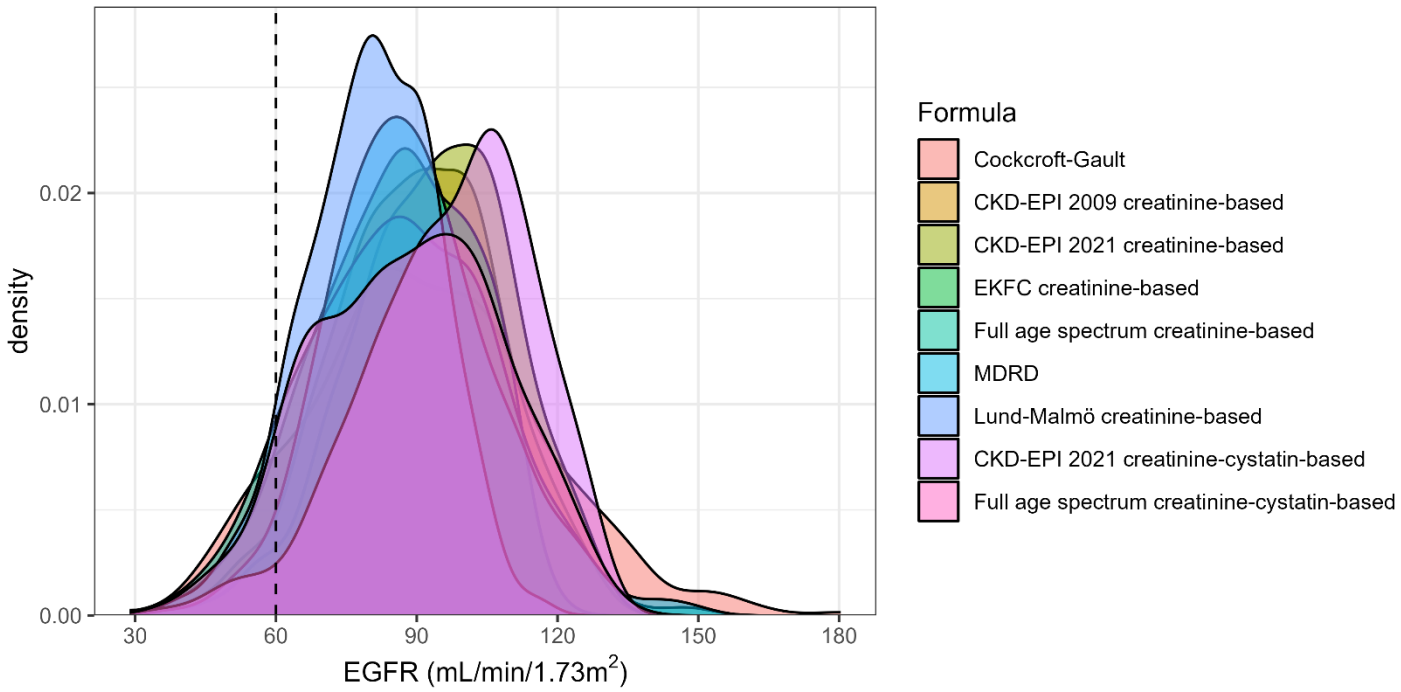
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**Introduction:** Regular national updates of chronic kidney disease (CKD) prevalence are essential to account for demographic changes, the evolving landscape of risk factors, and the needs of healthcare planning. The objective of this study was to evaluate the prevalence of CKD in the general Swiss population using different equations to estimate glomerular filtration rate (eGFR).

**Methods:** This study is an ancillary analysis of the Swiss Salt Study, which assessed salt consumption in the general Swiss population. Creatinine and cystatin C were measured in participants who consented to an optional blood draw. eGFR was then estimated using nine different equations. Stratified analyses by sex and age were performed for each equation. Albuminuria was also assessed.

**Results:** A total of 451 participants were included (231 men and 220 women). Using an eGFR threshold of 60 mL/min/1.73 m² to define CKD, prevalence ranged from to 3.1% (using Chronic Kidney Disease Epidemiology 2021 creatinin-cystatin C based) to 10.2% (using Cockcroft & Gault). Discrepancies between equations were consistent across age groups and between sexes. Albuminuria was infrequent.

**Conclusion:** The use of different eGFR equations resulted in substantial differences in estimated CKD prevalence. From a public health perspective, this may be of major importance with potential implications for healthcare costs and policies, and the risk of under or overdiagnosis.




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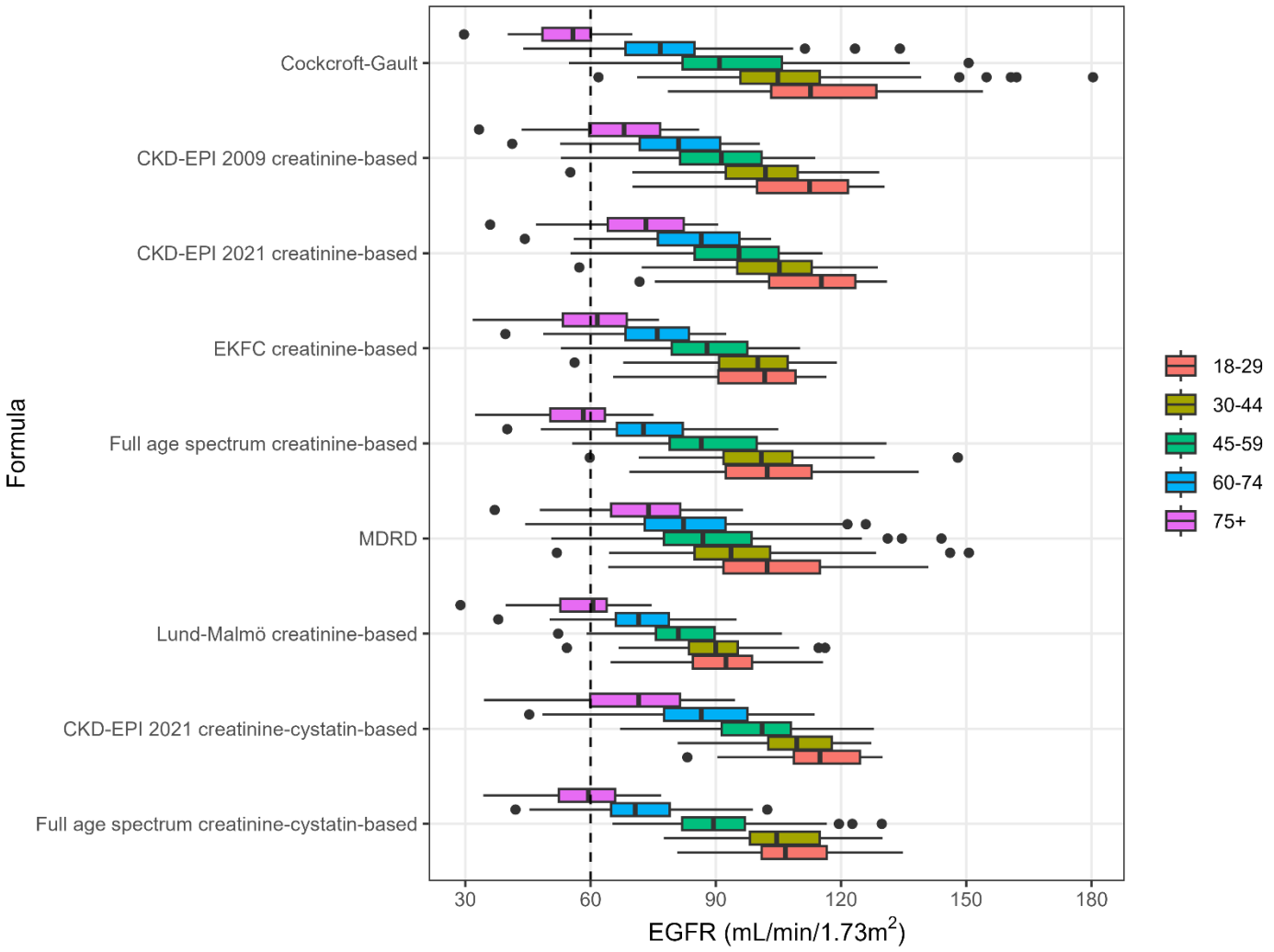



Table 2: eGFR distribution of the participants depending on the formula used for estimation, and weighted estimates of prevalence of CKD in the source population with 95% confidence intervals.

| Formulas | N=451 | | | | | | Median eGFR, mL/min/1.73 m² (IQR) | Weighted prevalence of CKD, % (95%CI) |
|---|---|---|---|---|---|---|---|---|
| eGFR categories | ≥120 | 90-119 | 60-89 | 45-59 | 30-44 | 0-29 | | |
| Cockcroft-Gault, n (%) | 57 (13) | 173 (38) | 175 (39) | 40 (8.9) | 5 (1.1) | 1 (0.2) | 91 (76 – 107) | 12.8 (9.2 – 17.5) |
| CKD-EPI 2009 creatinine-based, n (%) | 16 (3.5) | 216 (48) | 200 (44) | 16 (3.5) | 3 (0.7) | 0 (0) | 91 (79 – 102) | 4.3 (2.5 – 7.3) |
| CKD-EPI 2021 creatinine-based, n (%) | 24 (5.3) | 248 (55) | 164 (36) | 13 (2.9) | 2 (0.4) | 0 (0) | 95 (83 – 106) | 3.3 (1.8 – 6.0) |
| EKFC creatinine-based, n (%) | 0 (0) | 189 (42) | 233 (52) | 25 (5.5) | 4 (0.9) | 0 (0) | 87 (74 – 99) | 7.4 (4.7 – 11.6) |
| Full age spectrum creatinine-based, n (%) | 20 (4.4) | 176 (39) | 216 (48) | 33 (7.3) | 6 (1.3) | 0 (0) | 86 (73 – 101) | 9.5 (6.5 – 13.7) |
| MDRD, n (%) | 22 (4.9) | 178 (39) | 235 (52) | 14 (3.1) | 2 (0.4) | 0 (0) | 87 (77 – 99) | 3.3 (1.8 – 5.9) |
| Lund-Malmö creatinine-based, n (%) | 0 (0) | 116 (26) | 301 (67) | 30 (6.7) | 3 (0.7) | 1 (0.2) | 81 (71 – 90) | 8.6 (5.6 – 13.0) |
| CKD-EPI 2021 creatinine-cystatin-based, n (%) | 41 (9.1) | 267 (59) | 129 (29) | 12 (2.7) | 2 (0.4) | 0 (0) | 100 (86 – 111) | 3.4 (1.8 – 6.4) |
| Full age spectrum creatinine-cystatin-based, n (%) | 21 (4.7) | 201 (45) | 199 (44) | 26 (5.8) | 4 (0.9) | 0 (0) | 89 (73 – 103) | 8.1 (5.2 – 12.2) |

CKD: Chronic kidney disease; eGFR: Estimated glomerular filtration rate; CKD-EPI: Chronic kidney disease epidemiology; EKFC: European kidney function consortium; MDRD: Modification of diet in renal disease; IQR: Interquartile range; CI: Confidence interval


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## YSN-03

### Perioperative Fetuin-A dynamics as a potential patient-specific biomarker of acute kidney injury after cardiovascular surgery: First findings from the PEAK study

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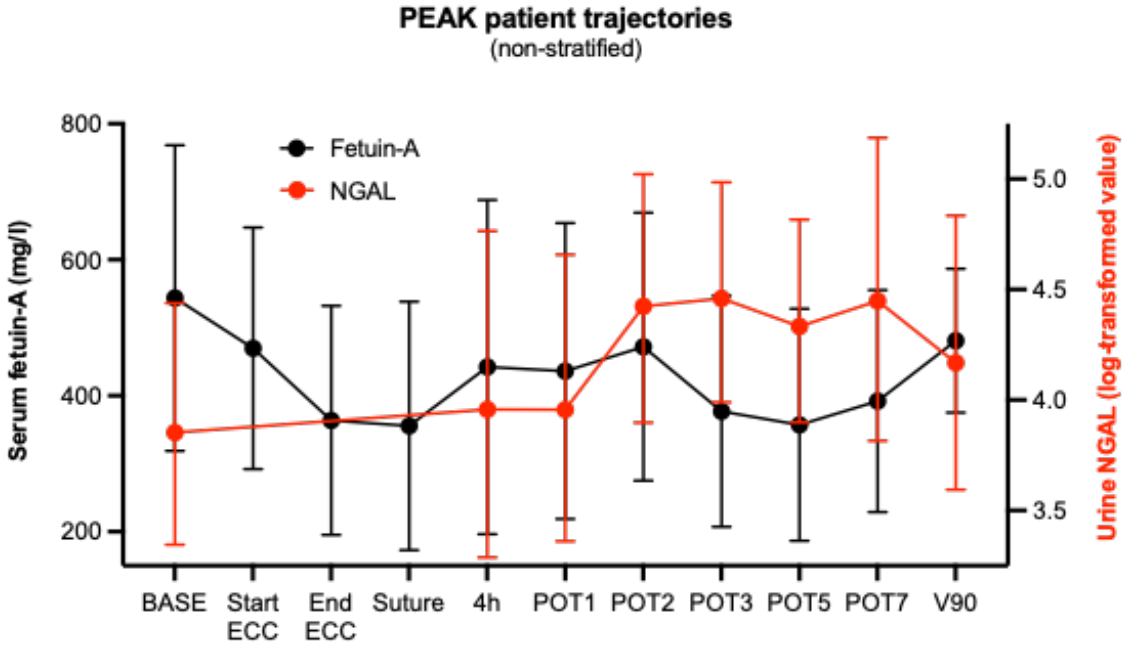
**Background.** Acute kidney injury (AKI) is a frequent complication of cardiovascular surgery (CVS) and is associated with increased morbidity and risk of chronic kidney disease (CKD). Fetuin-A (FA) is a liver-derived glycoprotein with mineral-chaperoning and tissue-protective properties. Experimental studies suggest that FA is depleted following tissue injury and ischemia–reperfusion injury (IRI), but its perioperative dynamics in humans remain poorly characterized. We hypothesized that serum FA dynamics reflect perioperative tissue injury and are associated with AKI.

**Methods.** PEAK is a prospective observational cohort study of patients undergoing elective CVS at the University Hospital Bern, Switzerland. Serum FA, urinary neutrophil gelatinase-associated lipocalin (uNGAL), and additional markers of renal injury and inflammation were measured serially by ELISA from the perioperative period through postoperative day 7 and again at day 90. Baseline clinical characteristics and frailty were recorded.

**Results.** Among 103 patients, mean age was 66 ± 9 years, 26.2% of participants were female, baseline creatinine was 84.5 ± 2.2 µmol/L, and 14.9% had preexisting CKD. FA showed a distinct bimodal postoperative depletion pattern, with two nadirs during the first postoperative week. In contrast, uNGAL followed an opposing trajectory, increasing during periods of greater FA depletion (Figure 1).

**Conclusion**. These findings suggest that perioperative FA depletion may reflect increased FA utilization during tissue injury, potentially reflecting sequential myocardial and renal ischemia-reperfusion injury following CVS. FA dynamics may therefore provide a patient-specific measure of perioperative injury burden and potentially identify a therapeutic window in which restoring depleted FA could limit IRI-associated kidney injury.

**Figure 1.** Perioperative dynamics of Fetuin-A and uNGAL following cardiovascular surgery.





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## YSN-04

### Tracking proteinuria-induced tubular injury by longitudinal intravital imaging

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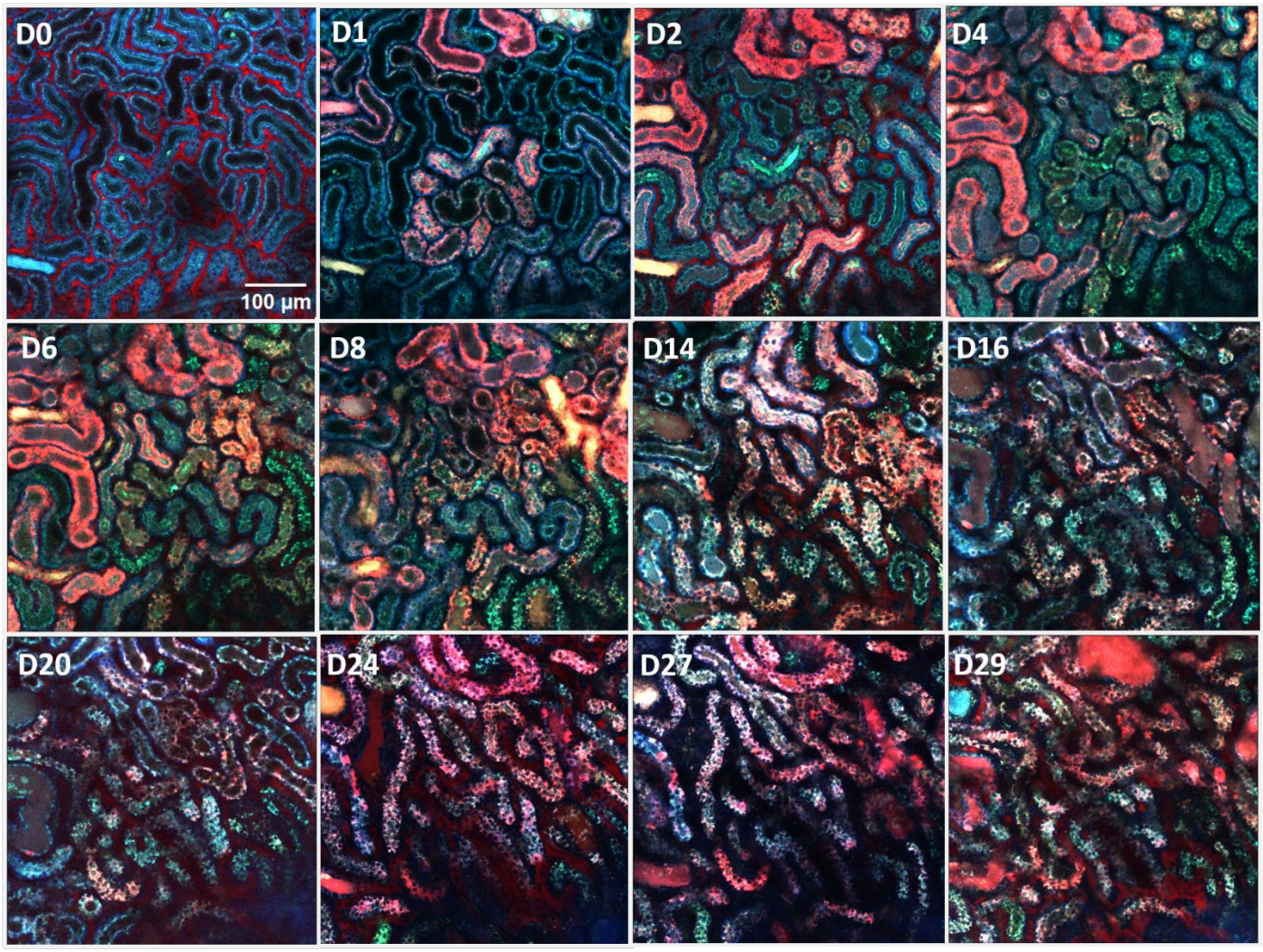
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Proteinuria is a driver of chronic kidney disease progression, yet the mechanisms by which excess filtered proteins induce segment-specific tubular dysfunction remain incompletely understood. Previous work showed that glomerular proteinuria induces transcriptional remodeling throughout the nephron, with extension of protein uptake from the S1 into the S2 proximal tubular segment, followed by injury, failed repair, and loss of canonical S2 functions. We hypothesize that the S2 segment is susceptible to protein overload because increased albumin handling disrupts lysosomal homeostasis and proteolytic function. We also hypothesize that the distal nephron is also affected by proteinuria, with changes in morphology, function and gene expression patterns.

To investigate this, we use the inducible POD-ATTAC mouse model of glomerular proteinuria, in which administration of dimerizer activates caspase-8-dependent apoptosis selectively in podocytes. We use a single-nucleus RNA-sequencing dataset from control and proteinuric POD-ATTAC kidneys to identify segment-specific alterations in lysosomal, endocytic, injury, and repair pathways. In parallel, renal tubular morphology and fate is investigated longitudinally in vivo using intravital two-photon microscopy. Lysosomal dysfunction is assessed using LysoTracker, while DQ-BSA, a self-quenched albumin substrate that becomes fluorescent following proteolytic degradation, is used to visualize albumin processing and lysosomal proteolytic activity in real time.

Preliminary intravital imaging demonstrates abnormal albumin uptake and persistent S2 albumin-derived material, with lysosomal dysfunction appearing earliest along failed repair axis. Consistent with these observations, analysis of the snRNA-seq dataset reveals changes in pathways associated with lysosomal function and cellular proteostasis in protein-exposed tubular populations. Ongoing experiments aim to determine whether altered lysosomal acidification and reduced proteolytic capacity precede structural injury and failed tubular repair.

Together, this study combines longitudinal functional imaging with transcriptomic analysis to define how proteinuria perturbs lysosomal function along the proximal tubule and to test whether lysosomal dysfunction represents a key mechanism underlying the vulnerability of the S2 segment.




A. Longitudinal imaging from D0 to D29 after POD-ATTAC induction. Blue and green = tubular autofluorescence, red = Albumin-594


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