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Autoimmune hepatitis is an independent risk factor for allograft loss and morbidity after liver transplantation: evidence from the Swiss Transplant Cohort Study

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Summary

BACKGROUND: Liver transplantation is the only curative option for end-stage autoimmune hepatitis (AIH). Although overall outcomes are favourable, previous studies suggest an increased risk of allograft loss and mortality in this population. We assessed post-liver transplantation outcomes in AIH recipients using data from the nationwide multicentre Swiss Transplant Cohort Study (STCS).

METHODS: We included all adult patients undergoing a first liver transplantation between May 2008 and December 2020. Indications were categorised as AIH, alcohol-related liver disease, viral liver disease, primary biliary cholangitis, primary sclerosing cholangitis, metabolic dysfunction-associated liver disease or other/mixed aetiologies. Survival analyses and multivariable competing risk Cox models were applied. Clinical Registration number (CER-VD): 2021-01982.

RESULTS: Among 880 liver transplantation recipients, 23 (2.6%) underwent liver transplantation for AIH. The 1-, 5- and 10-year post-liver transplantation survival rates in the AIH group were 87.0%, 76.5% and 76.5%, respectively – comparable to other indications. Allograft failure occurred in 34.8% of AIH recipients, significantly more often than in alcohol-related liver disease (8.3%, p <0.001), viral liver disease (13.5%, p = 0.003), primary sclerosing cholangitis (12.2%, p = 0.029) and metabolic dysfunction-associated liver disease (8.2%, p = 0.005). On multivariable analysis, AIH remained independently associated with increased allograft loss (HR 4.23, 95% CI 1.85–9.64; p = 0.003). Hepatic artery thrombosis occurred in 4/23 AIH patients and caused early allograft failure in all cases.

CONCLUSION: Despite comparable long-term survival, AIH recipients had a higher risk of early allograft loss, with vascular complications contributing to several early failures. Although the small AIH sample limited the precision of the estimated effects, these findings suggest the need for tailored pre- and post-transplant surveillance with particular attention to vascular complications.

Introduction

Autoimmune hepatitis is a chronic inflammatory liver disease characterised by elevated serum transaminase levels, increased immunoglobulin G levels, typical findings on histology and the presence of autoantibodies. The disease disproportionately affects women and typically presents either during childhood or between the fourth and sixth decades of life [1]. Mortality is highest in the first year after diagnosis and remains 6-fold higher than in the general population [2]. A recent meta-analysis showed an increase both in prevalence and incidence of autoimmune hepatitis in the last 50 years [3].

Although autoimmune hepatitis generally responds well to immunosuppressive therapy (most commonly corticosteroids and azathioprine) [4], about 10% of patients experience disease progression despite treatment, leading to decompensated cirrhosis. In these cases, as well as in case of acute liver failure, liver transplantation remains the only curative option [5].

According to data from the European Liver Transplantation Registry (ELTR), autoimmune hepatitis accounted for approximately 2% of all indications for liver transplantation between 1988 and 2016 [6, 7]. Although long-term outcomes are generally favourable, with reported survival rates of 79.4%, 70.8% and 60.3% at 5-, 10- and 15-years post-liver transplantation, respectively, emerging evidence suggests that recipients with autoimmune hepatitis may be at higher risk for post-transplant complications compared to those transplanted for other indications [8-10]. Several large European and North American registry studies have reported increased rates of allograft loss and mortality in this population, frequently attributed to infections and immune-mediated mechanisms such as acute or chronic rejection [8, 11, 12].

Recurrence of autoimmune hepatitis is another recognised contributor to allograft dysfunction, with prevalence estimates ranging from 17% to 42% [7]. Young age at transplantation (<42 years) has been associated with higher recurrence risk [13]. Unlike T cell-mediated rejection, recurrent autoimmune hepatitis displays distinct histological features and may lead to allograft failure in up to half of cases [14]. Importantly, experts have emphasised the need to prioritise re-transplantation for patients with recurrent autoimmune hepatitis-related cirrhosis even in the presence of a low Model for End-stage Liver Disease (MELD) score, due to high waiting list mortality [14].

Despite these insights, the underlying mechanisms contributing to adverse allograft outcomes in autoimmune hepatitis remain poorly understood, and most available data originate from single-centre or registry-based studies lacking detailed granularity. Furthermore, the role of vascular complications, which are rarely explored in this context, may be underrecognised.

In this study, we aimed to assess post-liver transplantation outcomes in patients transplanted for autoimmune hepatitis using national, multicentre, prospectively collected data. We specifically evaluated patient and allograft survival, rates and types of complications and aetiologies of allograft loss in autoimmune hepatitis recipients compared to those transplanted for other liver diseases.

Methods

Trial design and study population

We conducted a multicentric and retrospective study including all adult patients (≥18 years) who underwent liver transplantation between 1 May 2008 and 31 December 2020 in Switzerland and provided consent to participate in the Swiss Transplant Cohort Study. The Swiss Transplant Cohort Study is a countrywide cohort study with prospective data collection that has been enrolling all solid-organ transplant recipients since 2008. This project was approved by the Swiss Transplant Cohort Study Scientific Committee (reference: FUP181) and the local ethics committees (Commission cantonale d'éthique de la recherche sur l'être humain, CER-VD; reference: 2021-01982).

Patient classification and variables of interest

Patients were categorised into seven groups based on the primary indication for liver transplantation: (1) autoimmune hepatitis, (2) alcohol-related liver disease, (3) viral liver disease, (4) primary biliary cholangitis, (5) primary sclerosing cholangitis, (6) metabolic dysfunction-associated liver disease, and (7) other or mixed aetiologies.

Patients were assigned to the autoimmune hepatitis group (group 1) based on stringent clinical, immunological and histopathological criteria. Patients with variant syndrome were included in group 1 only if autoimmune hepatitis was the predominant component. All autoimmune hepatitis cases were reviewed individually, and in cases of diagnostic uncertainty, the treating physician was contacted for the diagnosis adjudication. The terms "autoimmune hepatitis group" and "patients with autoimmune hepatitis" will be used interchangeably throughout the manuscript.

Patients with unequivocal aetiologies were classified in the respective categories (groups 2 to 6). Patients with ambiguous, multiple (including MetALD) or rare aetiologies were classified in group 7 and excluded from comparative multivariable statistical analyses (see below).

Demographic, clinical and transplant-related data were collected through the Swiss Transplant Cohort Study database and supplemented via a dedicated case report form for patients with autoimmune hepatitis. Variables included autoimmune comorbidities, anticoagulation and antiplatelet therapy. Post-liver transplantation immunosuppressive regimens were categorised as follows: 1) "tacrolimus-based": combination of tacrolimus, mycophenolate mofetil and/or prednisone; 2) "ciclosporin-based": combination of ciclosporin, mycophenolate mofetil and/or prednisone/prednisolone; 3) "mTOR inhibitor-based immunosuppression": combination of everolimus or sirolimus, with any calcineurin inhibitor and/or prednisone/prednisolone, optionally with mycophenolate mofetil or azathioprine; 4) "other regimens": combinations outside the above definitions.

Outcomes

The primary outcomes were patient and allograft survival. Allograft loss was defined as allograft failure leading to death or retransplantation. Death without prior allograft dysfunction was not considered allograft loss. Secondary outcomes included post-transplant complications:

- Vascular: arterial and venous thrombosis, vascular stenosis, haemorhagic events;
- Biliary: biliary stenosis and strictures, biliary leak, biliary dilation;
- Infectious: any systemic or localised infection;
- Rejection episodes: all biopsy-proven or clinically diagnosed T cell-mediated rejections, irrespective of severity.

A specific case report form was used to document complications and outcomes for all autoimmune hepatitis recipients, including causes of death and allograft failure (see Appendix).

Statistical analysis

Descriptive statistics were reported as median (interquartile range, IQR) or mean (standard deviation, SD) for continuous variables, as appropriate, and counts (percentage) for categorical variables. Comparisons between groups were performed using the chi-squared or Fisher's exact test for categorical variables, and the t-test or nonparametric testing (Mann–Whitney U or Kruskal–Wallis rank-sum test) for continuous variables, as appropriate.

Baseline characteristics were described across all seven aetiological groups. Each group was compared with the autoimmune hepatitis group, and an additional comparison was performed between the autoimmune hepatitis group and all non-autoimmune hepatitis recipients combined. Outcomes were reported for groups 1 to 6; group 7 was excluded because of its heterogeneous composition. Given the limited number of outcome events, univariable and multivariable analyses compared the autoimmune hepatitis group with all non-autoimmune hepatitis recipients combined.

Patient survival was assessed using the Kaplan–Meier analysis. For allograft failure and death, cumulative incidence functions were calculated to account for competing risks (death without an allograft failure as the competing event), and differences between groups were tested using Gray's test. We used the Bonferroni method to adjust for multiple comparisons.

To investigate associations of autoimmune hepatitis on transplant outcomes, we performed cause-specific Cox proportional hazards models. These models were fitted for the endpoint allograft loss correcting for known predictors (autoimmune hepatitis vs non-autoimmune hepatitis aetiology, recipient sex and age, MELD score, type of donation (living vs deceased), cold ischaemia time, rejection episodes, induction immunosuppressive therapy and mycophenolate mofetil use) and competing event death [15-17]. Variables for inclusion in multivariable models were selected based on clinical relevance, evidence from prior literature and parsimony guided by the number of events. Ethnicity was not included as a covariate because of the highly imbalanced distribution and

the limited number of outcome events, despite evidence showing a potential impact of ethnicity on the outcome [18]. Cause-specific Cox proportional hazards models were fitted for the endpoint overall complication and specific complications (vascular, biliary, infectious complications, as well as rejection), correcting for known predictors [19], with rejection complication coded as a 90-day exposure. Hazard ratios (HR) are reported relative to a reference category with an HR of 1.0, while absolute event probabilities are presented using Kaplan–Meier estimates or cumulative incidence functions, as appropriate.

A p-value <0.05 was considered statistically significant. All statistical analyses and figures were performed using R version 4.4.3.

This study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement; the completed checklist is provided in table S5 [20].

Results

Study population and baseline characteristics

A total of 880 adult liver transplantation recipients were included in our analysis. Among them, 23 (2.6%) were transplanted for autoimmune hepatitis, 254 (28.9%) for alcohol-related liver disease, 303 (34.4%) for viral liver disease, 38 (4.3%) for primary biliary cholangitis, 49 (5.6%) for primary sclerosing cholangitis, 61 (6.9%) for metabolic dysfunction-associated liver disease and 152 (17.3%) for other or mixed aetiologies (figure 1). Patient characteristics are presented in table 1.

Table 1: Baseline characteristics of liver transplant recipients and donors according to the liver disease aetiology leading to transplantation.

	AIH (n = 23)	ARLD (n = 254)	VLD (n = 303)	PBC (n = 38)	PSC (n = 49)	MASLD (n = 61)	Other (n = 152)	Total (n = 880)	p- value†
Patient sex: male, n (%)	8 (34.8%)	211 (83.1%)	233 (76.9%)	11 (28.9%)	35 (71.4%)	41 (67.2%)	118 (77.6%)	657 (74.7%)	<0.001
Donor sex: male, n (%)	14 (60.9%)	141 (55.5%)	185 (61.3%)	19 (50.0%)	29 (61.7%)	35 (57.4%)	93 (61.2%)	516 (58.8%)	0.841
Recipient age, median (IQR)	49.0 (39.0; 57.5)	60.0 (55.0; 64.0)	56.0 (51.0; 61.0)	55.5 (48.3; 64.8)	49.0 (42.0; 59.0)	62.0 (57.0; 66.0)	56.0 (51.0; 61.0)	57.0 (51.0; 62.0)	0.002
Recipient ethnicity, n (%)									<0.001
… Caucasian	20 (87.0%)	248 (97.6%)	253 (83.5%)	38 (100.0%)	48 (98.0%)	60 (98.4%)	141 (92.8%)	808 (91.8%)	
… African	1 (4.3%)	1 (0.4%)	23 (7.6%)	0	1 (2.0%)	0	7 (4.6%)	33 (3.8%)	
… Asian	2 (8.7%)	3 (1.2%)	20 (6.6%)	0	0	0	2 (1.3%)	27 (3.1%)	
… Other	0	2 (0.8%)	7 (2.3%)	0	0	1 (1.6%)	2 (1.3%)	12 (1.4%)	
Donor age, median (IQR)	54.0 (37.0; 73.5)	57.0 (45.0; 69.0)	56.0 (40.0; 66.5)	54.0 (36.0; 70.8)	57.0 (48.0; 65.0)	63.0 (54.0; 71.0)	59.0 (44.8; 68.3)	57.0 (44.0; 68.0)	0.823
Type of donation, n (%)									0.268
… Living	2 (8.7%)	8 (3.1%)	13 (4.3%)	2 (5.3%)	7 (14.3%)	0	5 (3.3%)	37 (4.2%)	
… DCD	5 (21.7%)	37 (14.6%)	42 (13.9%)	3 (7.9%)	6 (12.2%)	14 (23.0%)	15 (9.9%)	122 (13.9%)	
… DBD	16 (69.6%)	209 (82.3%)	248 (81.8%)	33 (86.8%)	36 (73.5%)	47 (77.0%)	132 (86.8%)	721 (81.9%)	
MELD score, median (IQR)	30.0 (25.8; 35.0)	29.0 (23.0; 34.0)	28.0 (22.0; 34.3)	28.0 (20.0; 32.5)	28.0 (24.0; 35.5)	30.0 (24.0; 35.0)	30.0 (25.0; 35.0)	29.0 (23.0; 35.0)	0.463
Type of allograft, n (%)									0.254
… Domino graft	1 (4.3%)	4 (1.6%)	2 (0.7%)	1 (2.6%)	0	0	2 (1.3%)	10 (1.1%)	
… Reduced liver	0	0	1 (0.3%)	0	0	0	0	1 (0.1%)	
… Split left	1 (4.3%)	2 (0.8%)	1 (0.3%)	0	2 (4.1%)	0	2 (1.3%)	8 (0.9%)	
… Split right	1 (4.3%)	8 (3.1%)	19 (6.3%)	3 (7.9%)	5 (10.2%)	0	3 (2.0%)	39 (4.4%)	
… Whole liver	20 (87.0%)	240 (94.5%)	279 (92.4%)	34 (89.5%)	42 (85.7%)	61 (100.0%)	145 (95.4%)	821 (93.4%)	
Recipient BMI, median (IQR)	24.4 (22.1; 28.6)	26.9 (24.1; 30.4)	25.7 (23.10; 29.0)	23.3 (21.8; 25.3)	22.3 (21.0; 24.5)	29.3 (27.3; 32.1)	27.3 (24.2; 31.6)	26.3 (23.4; 29.8)	0.311
Recipient positive history of diabetes mellitus, n (%)	3 (13.0%)	85 (33.5%)	60 (19.8%)	5 (13.2%)	4 (8.2%)	44 (72.1%)	53 (34.9%)	254 (28.9%)	0.090
Recipient positive history of hyperlipidaemia, n (%)	2 (8.7%)	15 (5.9%)	13 (4.3%)	4 (10.5%)	2 (4.1%)	14 (23.0%)	24 (15.8%)	74 (8.4%)	0.960

† p-value calculated as comparison between AIH group and all the other patients grouped together. Abbreviations: AIH: autoimmune hepatitis; ARLD: alcohol-related liver disease; BMI: body mass index; DBD: donor brain death; DCD: donor circulatory death; IQR: interquartile range; MASLD: metabolic dysfunction-associated liver disease; MELD: model for end-stage liver disease; PBC: primary biliary cholangitis; PSC: primary sclerosing cholangitis; VLD: viral liver disease.

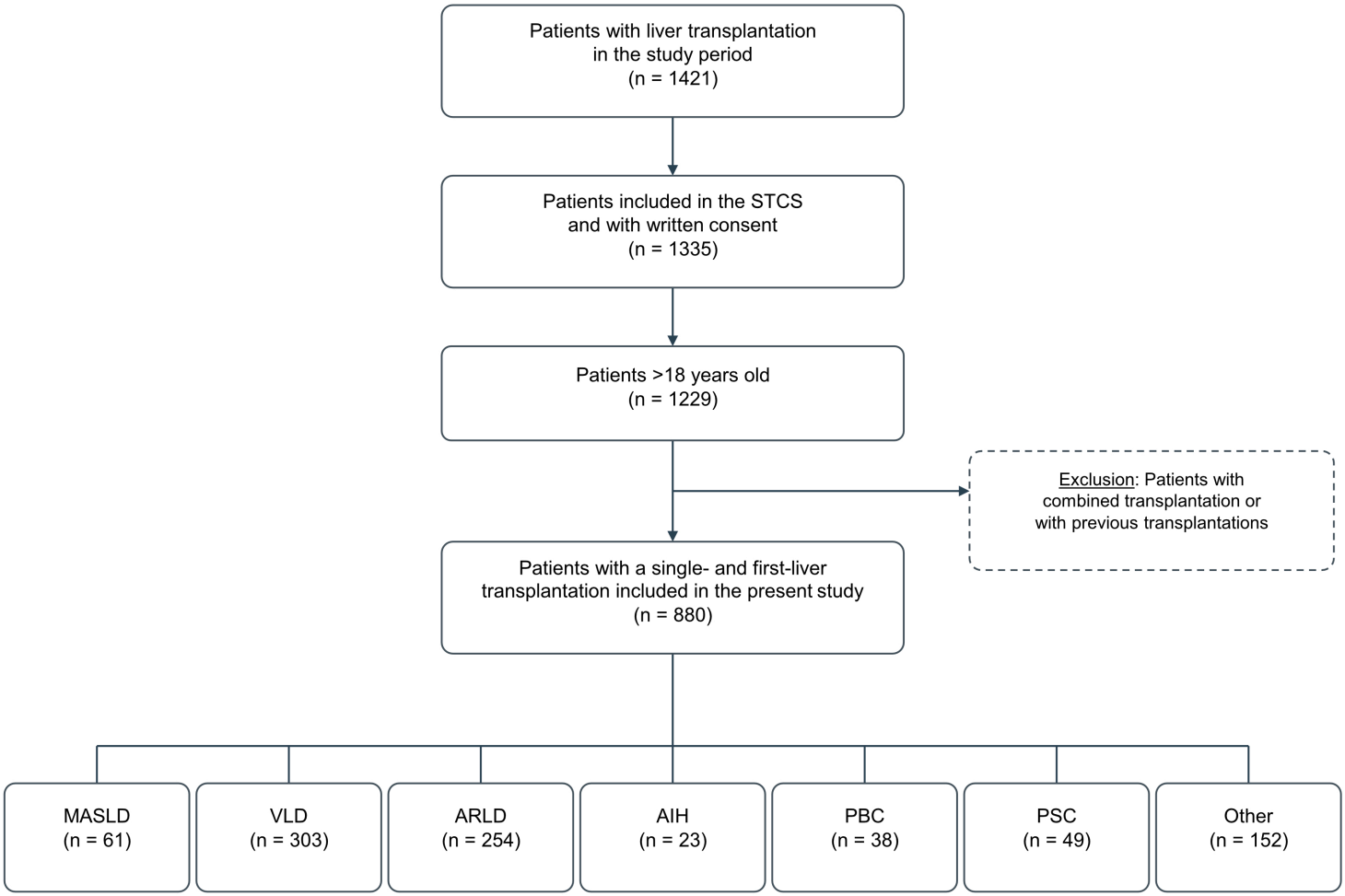


Figure 1: Study flowchart.

General description of patients with autoimmune hepatitis

Among the 23 autoimmune hepatitis recipients, 14 (60.9%) had a classic chronic presentation (type 1 in 13 cases and type 2 in 1 case). Seven (30.4%) had variant syndrome with primary biliary cholangitis or primary sclerosing cholangitis, categorised under autoimmune hepatitis due to predominance of autoimmune hepatitis features, and two patients (8.7%) had fulminant presentations requiring urgent liver transplantation. The cohort was predominantly female (65.2%) and of Caucasian origin (87.0%), with a median age of 49 years (IQR 39–58) at the time of transplantation. The median MELD score at liver transplantation was 30 (IQR 26–35). Most patients (91.3%) received allografts from deceased donors; 69.9% of donors were males, with a median age of 54 years (IQR 38–74) at donation and a median cold ischaemia time of 6.6 hours (IQR 5.6–8.3). No patient experienced delayed allograft function. Ten patients (43.5%) had a concomitant autoimmune condition including Hashimoto thyroiditis (n = 5), mixed connective tissue disease (n = 2), coeliac disease (n = 2), systemic lupus erythematosus (n = 1) and ulcerative colitis (n = 1).

At the time of transplantation, 69.6% of patients with autoimmune hepatitis were receiving immunosuppression (IS), primarily prednisone (52.2%) and/or azathioprine (43.5%). Others had received mycophenolate mofetil (21.7%), ciclosporin (8.7%) or infliximab (4.3%). Seven patients (30.4%) were not immunosuppressed, including two patients with fulminant autoimmune hepatitis, four patients with variant syndrome and one patient with an initially unclear liver disease. In the perioperative period, 95.7% received corticosteroids and 87.0% received tacrolimus. No patient received an mTOR inhibitor-based regimen. Early post-transplant IS was tacrolimus-based in 87.0%, ciclosporin-based in 4.3% and classified as “other” in 8.7%. In the latter group, one patient received a combination of mycophenolate mofetil and corticosteroid and one received corticosteroid

monotherapy. All patients except one received induction treatment with the anti-IL-2 receptor antibody basiliximab. Eleven patients received antiplatelet or anticoagulation therapy (eight and two patients, respectively, with one patient having to replace antiplatelet therapy with an anticoagulation treatment after a thromboembolic event occurred). Anticoagulation was systematically initiated following a documented thromboembolic event.

Compared to other groups, autoimmune hepatitis recipients were significantly younger at the time of liver transplantation, with a median age of 49 years (IQR 39–57.5) vs 60 years (IQR 55–64) for alcohol-related liver disease, 56 years (51–61) for viral liver disease and 62 years (57–66) for metabolic dysfunction-associated liver disease (p <0.001). In contrast, primary biliary cholangitis and primary sclerosing cholangitis recipients had similar age profiles to autoimmune hepatitis recipients. Patients with autoimmune hepatitis were more often female than those with alcohol-related liver disease, viral liver disease, primary sclerosing cholangitis or metabolic dysfunction-associated liver disease. No significant differences were observed across groups in MELD score, BMI, cardiovascular comorbidities, donor age or donation type. Details are shown in table 1.

Patient survival was comparable between liver transplantation recipients with autoimmune hepatitis and the other defined groups

Five autoimmune hepatitis recipients (21.7%) died during the follow-up period (maximum 11.5 years). The median follow-up time in patients with autoimmune hepatitis was 3.4 years (95% confidence interval (CI) 1.7–5.2). The 1-year, 5-year and 10-year post-transplant survival rates in the autoimmune hepatitis group were 87.0% (95% CI 74.2–100), 76.5% (95% CI 60.2–97.2) and 76.5% (95% CI 60.2–97.2), respectively. The Kaplan–Meier analysis demonstrated no significant differences in overall survival between autoimmune hepatitis and other groups: alcohol-related liver disease (p = 0.503), viral liver disease (p = 0.969), primary biliary cholangitis (p = 0.562), primary sclerosing cholangitis (p = 0.936) or metabolic dysfunction-associated liver disease (p = 0.521) (figure 2).

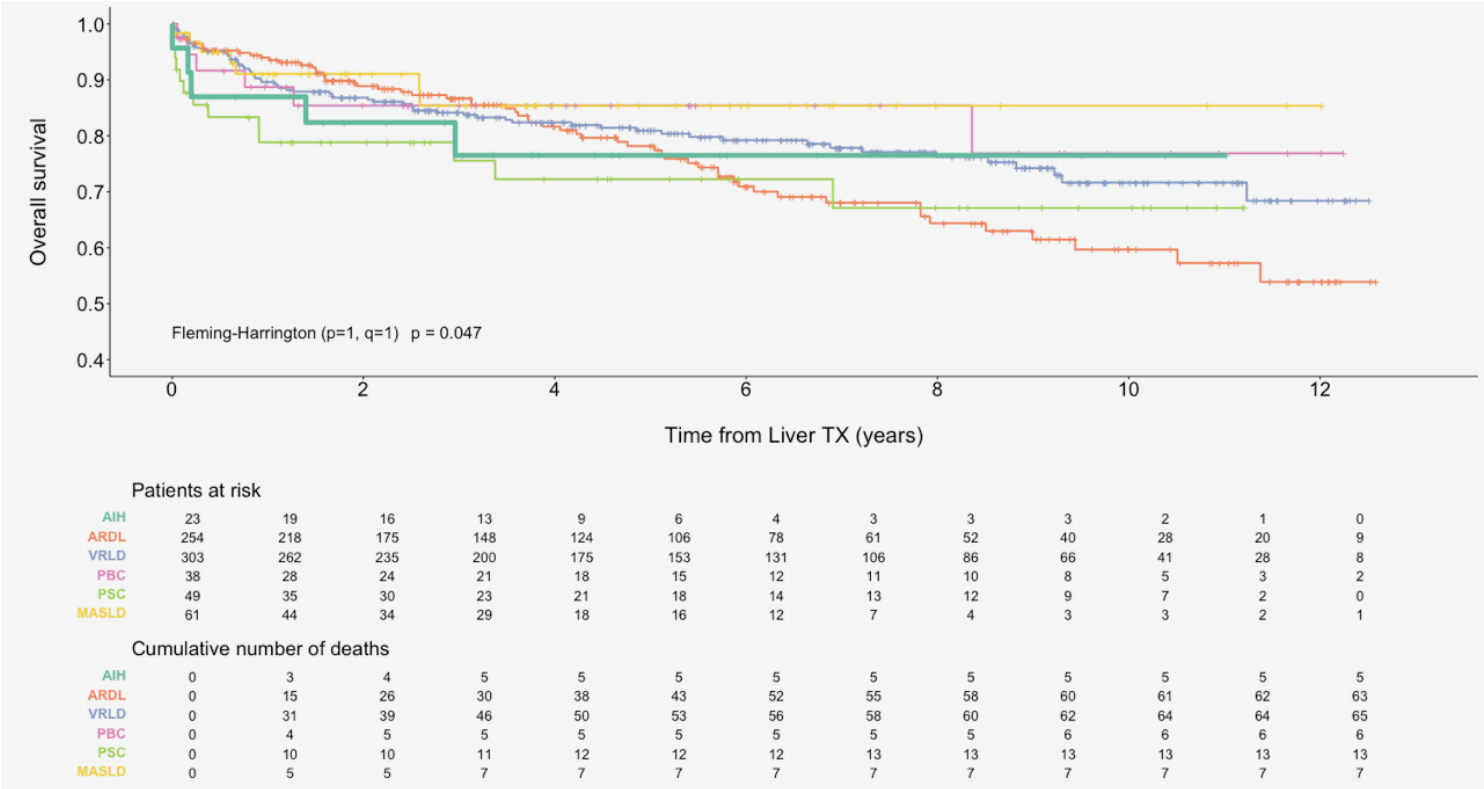


Figure 2: Overall survival. ABBREVIATIONS: AIH: autoimmune hepatitis; ARLD: alcohol-related liver disease; MASLD: metabolic dysfunction-associated liver disease; PBC: primary biliary cholangitis; PSC: primary sclerosing cholangitis; VLD: viral liver disease.

Patients with autoimmune hepatitis are exposed to an increased risk of allograft failure

In contrast to patient survival, early allograft failure was significantly more frequent in patients with autoimmune hepatitis, with 8 of 23 (34.8%) experiencing allograft loss. The 1-, 5- and 10-year allograft survival rates in this group were 73.9% (95% CI 54.8–89.7), 64.0% (95% CI 44.0–83.4) and 64.0% (95% CI 44.0–83.4), respectively. The median time to allograft loss was 41.0 days (IQR 1.8–172.0). Competing risk analysis revealed a significantly higher cumulative incidence of allograft loss in autoimmune hepatitis compared with other aetiologies (p = 0.002) (figure 3). Differences were particularly notable when comparing autoimmune hepatitis to alcohol-related liver disease (p <0.001), viral liver disease (p = 0.003), primary sclerosing cholangitis (p = 0.029) and metabolic dysfunction-associated liver disease (p = 0.005). There was no significant difference versus primary biliary cholangitis (p = 0.178). All patients with autoimmune hepatitis who died had previously suffered an allograft failure. The cause of allograft loss was ischaemic lesions of the liver in five patients, massive haemorrhage in one patient, primary non-function in one patient and chronic rejection in one patient.

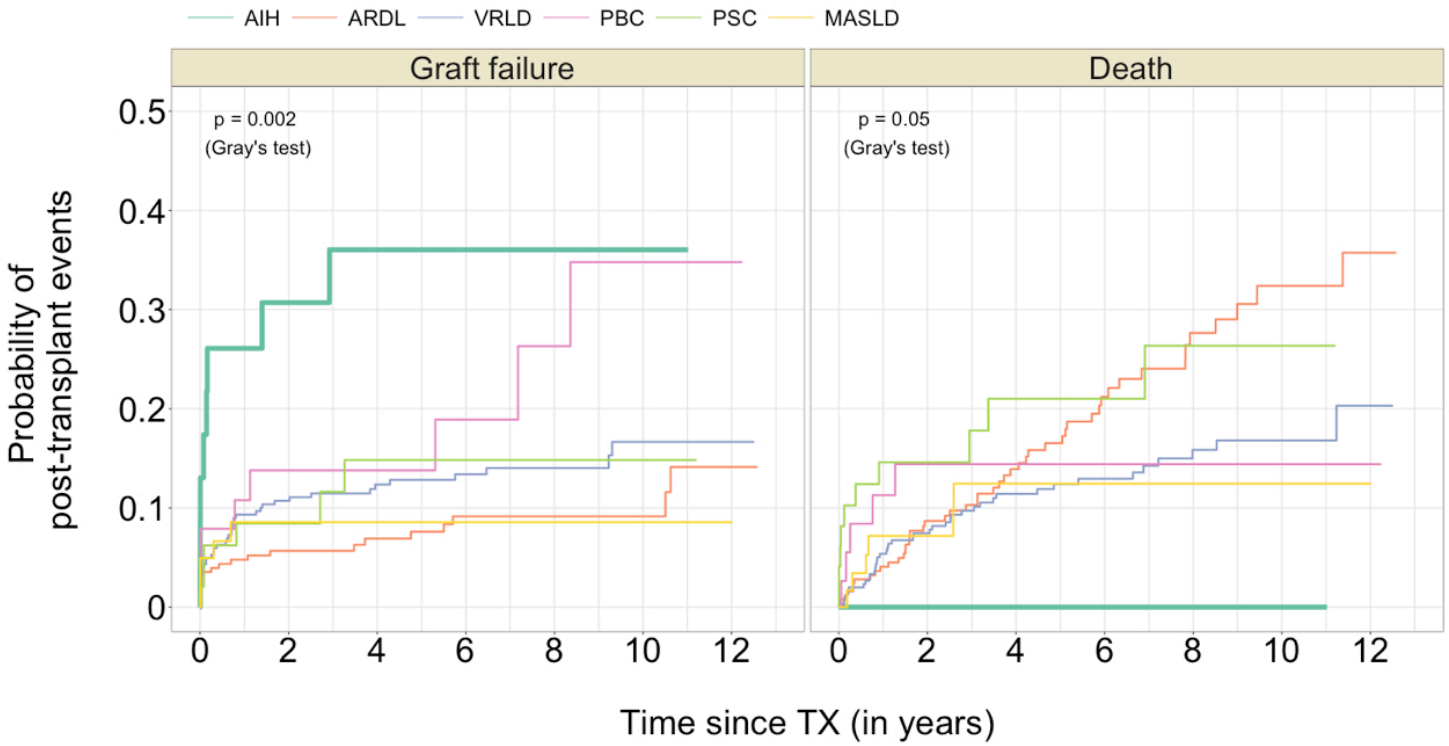


Figure 3: Allograft failure and death without previous allograft failure. ABBREVIATIONS: AIH: autoimmune hepatitis; ARLD: alcohol-related liver disease; MASLD: metabolic dysfunction-associated liver disease; PBC: primary biliary cholangitis; PSC: primary sclerosing cholangitis.

Burden of complications in autoimmune hepatitis liver transplantation recipients

Eighteen patients with autoimmune hepatitis (78.3%) experienced at least one post-transplant complication. The median time to first complication was 17 days (IQR 2.8–29.2). The most frequent complications were vascular (n = 14, 60.9%), followed by biliary (n = 7, 30.4%) and infectious (n = 5, 21.7%). Regarding vascular complications, hepatic artery thrombosis occurred in 4 patients (17.4%), leading to allograft failure within 90 days in all cases. One of the patients developed hepatic artery thrombosis in the context of pancreatobiliary adenocarcinoma of the allograft. Biopsy-proven rejection episodes were observed in eight autoimmune hepatitis recipients (34.8%), with a median onset at 23.5 days (IQR 15.8–158). No biopsy-proven recurrence of autoimmune hepatitis was documented. Comparative rates of complications and comorbidities are detailed in tables 2 and 3.

Table 2: Description of the 23 patients with autoimmune hepatitis (AIH).

Patient	Sex	Type of AIH	Cause of death	Cause of allograft loss	Complication on hepatic artery	Autoimmune comorbidity	Antiplatelet, anticoagulation	Immunosuppression before liver transplantation
1	Male	AIH/PSC	N/A	N/A	Stenosis	Ulcerative colitis	Aspirin	Azathioprine, prednisone, infliximab
2	Female	Type 1	Multiorgan failure after allograft dysfunction	Massive haemorrhage	Stenosis	Mixed collagenosis	No	Azathioprine, prednisone, ciclosporin
3	Female	Fulminant/Type 1	N/A	N/A	No	No	Aspirin	Azathioprine, prednisone
4	Female	AIH/PBC	N/A	N/A	No	Hashimoto disease	No	Azathioprine, prednisone
5	Female	Type 1	Probable acute rejection of second allograft	Probable acute vascular rejection	No	No	No	Azathioprine
6	Female	Type 1	N/A	Primary nonfunction	No	Hashimoto disease	No	Azathioprine
7	Female	AIH/PBC	N/A	Ischaemic lesions after HAT	Thrombosis	No	Rivaroxaban	Mycophenolate mofetil
8	Female	Type 2	N/A	N/A	No	No	Aspirin	History unknown
9	Male	Fulminant	Pancreatobiliary adenocarcinoma of the allograft leading to multiorgan failure	HAT in the context of the pancreatobiliray adenocarcinoma of the allograft	Thrombosis	No	Aspirin	No
10	Male	Type 1	N/A	N/A	No	Mixed collagenosis	Aspirin then DOAC	Prednisone
11	Female	Type 1	N/A	N/A	No	Hashimoto disease	No	Mycophenolate mofetil, prednisone
12	Female	Type 1	N/A	N/A	Stenosis	No	Aspirin	Azathioprine, prednisone, mycophenolate mofetil
13	Female	Type unknown	N/A	N/A	No	Systemic lupus erythematous, coeliac disease	No	Prednisone
14	Female	AIH/PBC	N/A	N/A	No	No	No	No
15	Female	Type 1	N/A	HAT	Thrombosis	Hashimoto disease	No	Mycophenolate mofetil
16	Female	AIH/PBC	N/A	N/A	Kinking	No	No	No
17	Male	Type 1	N/A	N/A	No	No	No	Azathioprine, prednisone
18	Male	AIH/unspecified cholangitis	Septic shock after retransplantation	Ischaemic lesions after thrombosis	Thrombosis	No	No	No
19	Male	Type 1	N/A	N/A	No	No	Aspirin	Prednisone
20	Male	AIH/PBC	Graft dysfunction	Chronic rejection	No	No	Aspirin	No
21	Female	Type 1	N/A	N/A	Asymptomatic stenosis	Coeliac disease	Aspirin	Azathioprine, prednisone, mycophenolate mofetil
22	Male	Type 1	N/A	N/A	No	No	Rivaroxaban	No
23	Female	Fulminant	N/A	N/A	No	No	No	No

Abbreviations: AIH: autoimmune hepatitis; DOAC: direct oral anticoagulant; HAT: hepatic artery thrombosis; N/A: not available; PBC: primary biliary cholangitis.

Table 3: Post-transplant outcomes and complications according to the indication for liver transplantation.

	AIH (n = 23)	ARLD (n = 254)	VLD (n = 303)	PBC (n = 38)	PSC (n = 49)	MASLD (n = 61)	p-value[†]
Deaths, n (%)	5 (21.7%)	63 (24.8%)	65 (21.5%)	6 (15.8%)	13 (26.5%)	7 (11.5%)	0.250
Allograft loss, n (%)	8 (34.8%)	21 (8.3%)	41 (13.5%)	8 (21.1%)	6 (12.2%)	5 (8.2%)	<0.001
Patients with any rejections, n (%)	8 (34.8%)	69 (27.2%)	106 (35%)	14 (36.8%)	15 (30.6%)	11 (18%)	0.014
Time to 1st rejection (days), median (IQR)	23.5 (15.8; 158)	124 (15; 377)	62.5 (9.2; 270.5)	184 (21; 537)	53 (16.5; 221)	69 (12; 91)	0.596
Patients with at least one complication, n (%)	18 (78.3%)	120 (47.2%)	136 (44.9%)	21 (55.3%)	28 (57.11%)	23 (37.7%)	0.002
Time to first complication (days), median (IQR)	17.00 (2.8; 29.3)	24.5 (2.0; 123.5.0)	12.5 (2.7; 75.0)	20.0 (2.0; 367.0)	91.0 (3.0; 116.0)	25 (4.0; 95.0)	0.424
Patients with at least one vascular complication, n (%)	14 (60.9%)	68 (26.8%)	79 (26.1%)	10 (26.3%)	17 (34.7%)	12 (19.7%)	<0.066
Time to first vascular complication (days), median (IQR)	15.5 (0.2; 25.7)	5.0 (1.0; 244.0)	8.0 (1.0; 90.5)	1.5 (0.2; 4.5)	18.0 (3.0; 116.0)	4.5 (1.0; 117.75)	0.543
Patients with at least one HAT, n (%)	4 (17.4%)	9 (3.5%)	15 (5.0%)	1 (2.6%)	2 (4.1%)	1 (1.6%)	0.001
Patients by number of HATs, n (%)							<0.001
… 0	19 (82.6%)	245 (90.9%)	288 (95.0%)	37 (97.4%)	47 (95.9%)	60 (98.4%)	
… 1	2 (8.7%)	8 (3.1%)	13 (4.3%)	1 (2.6%)	2 (4.1%)	1 (1.6%)	
… 2	2 (8.7%)	1 (0.4%)	2 (0.7%)	0	0	0	
Patients with allograft loss in the 90 days following HAT, n (%)	4 (100%)	3 (33.3%)	8 (53.3%)	1 (100.0%)	2 (100.0%)	0	0.250
Time to HAT (days), median (IQR)	268.0 (18.7; 586.0)	1450.0 (28.0; 2518.0)	10.0 (2.5; 94.0)	2.0 (2.0; 2.0)	21.5 (16.7; 26.2)	2983.0 (2983.0; 2983.0)	0.138
Patients with at least one infectious complication, n (%)	5 (21.7%)	38 (15.0%)	38 (12.5%)	6 (15.8%)	13 (26.5%)	6 (9.8%)	0.182
Time to first infectious complication (days), median (IQR)	182.0 (19.0; 332.0)	91.0 (3.5; 1831.5)	91.5 (17.2; 475.7)	99.5 (56.2; 181.7)	558.0 (95.0; 1984.0)	25.0 (4.0; 116.0)	0.301
Patients with at least one biliary complication, n (%)	7 (30.4%)	71 (28.0%)	98 (32.3%)	13 (34.2%)	17 (34.7%)	10 (16.4%)	0.749
Time to first biliary complication (days), median (IQR)	30.0 (21.5; 222.0)	64.0 (18.0; 254.5)	56.0 (13.0; 233.0)	368.0 (55.0; 724.0)	106.0 (8.5; 251.2)	85.0 (30.7; 97.0)	0.549

[†] p-value calculated as comparison between AIH group and all the other patients grouped together.

ABBREVIATIONS: AIH: autoimmune hepatitis; ARLD: alcohol-related liver disease; BMI: body mass index; HAT: hepatic artery thrombosis; IQR: interquartile range; MASLD: metabolic dysfunction-associated liver disease; MELD: model for end-stage liver disease; PBC: primary biliary cholangitis; PSC: primary sclerosing cholangitis; VLD: viral liver disease.

Autoimmune hepatitis is an independent risk factor for allograft loss

On univariable competing risk analysis for allograft loss, significant risk factors included diagnosis of autoimmune hepatitis (hazard ratio (HR) 3.56, 95% CI 1.72–7.37; p = 0.001) and increasing donor age (HR 1.01 per year, 95% CI 1.00–1.02; p = 0.048). The patient's sex, age, body mass index (BMI), history of diabetes mellitus, MELD score and cold ischaemia time were not identified as significant risk factors. On multivariable analysis adjusting for the variables described above, autoimmune hepatitis remained independently associated with increased risk of allograft loss (HR 4.23, 95% CI 1.85–9.64, p = 0.003) (table S1 in the Appendix). Sensitivity analyses after exclusion of patients with acute liver failure and variant syndromes showed that the increased risk of allograft loss remained present in these subgroups, although this was not statistically significant in the latter (figure S1).

Autoimmune hepatitis is an independent risk factor for post-transplant complications, in particular vascular complications

On univariable analysis, autoimmune hepatitis was significantly associated with the risk of any post-transplant complications (HR 2.34, 95% CI 1.55–3.54; p <0.001), along with a higher MELD score (HR 1.02 per point, 95% CI 1.01–1.03; p <0.001), living donation (HR 2.64, 95% CI 1.86–3.74; p <0.001) and the occurrence of acute T cell-mediated rejection (HR 4.62, 95% CI 3.48–6.14; p <0.001). The patient's age was a protective factor (HR 0.99, 95% CI 0.98–1.00, p = 0.03). Multivariable analysis confirmed the higher risk of complications for patients with autoimmune hepatitis (HR 1.81, 95% CI 1.14–2.89; p = 0.021) (table S2). The full multivariable model accounted for patient demographics, MELD score, donor factors, pre-liver transplantation IS and rejection events. MELD score (HR 1.02 per point, 95% CI 1.01–1.03; p <0.001), living donation (HR 2.86, 95% CI 1.87–4.37; p <0.001) and rejection episode (HR 4.80, 95% CI 3.59–6.42; p <0.001) were also identified as risk factors. When considering only vascular and thrombotic complications, autoim-

mune hepatitis remained associated with these specific complications (HR 2.86, 95% CI 1.60–5.11; p = 0.002 and HR 3.29, 95% CI 1.28–8.48; p = 0.032) (tables S3 and S4). Sensitivity analyses after exclusion of patients with acute liver failure and variant syndromes showed that the increased risk of post-transplant overall complications and vascular complications remained present in these subgroups, although this was not statistically significant in the latter (figures S2, S3 and S4).

Discussion

In this nationwide cohort study using data from the Swiss Transplant Cohort Study, we observed a similar long-term survival but a higher proportion of allograft loss among autoimmune hepatitis recipients, largely occurring early, compared to patients transplanted for other common indications. Despite comparable patient survival, the 1- and 5-year allograft survival rates in autoimmune hepatitis recipients (73.9% and 64.0%, respectively) were lower than those observed in non-autoimmune hepatitis groups and slightly inferior to outcomes reported in previous European studies. Importantly, the elevated risk of allograft failure did not translate into higher mortality, likely owing to timely access to retransplantation. Patient survival at 1 and 5 years (87.0% and 76.5%, respectively) was consistent with earlier studies, underscoring that long-term survival remains achievable in autoimmune hepatitis despite a high burden of allograft complications [8, 21].

An important finding of our study was the high incidence of vascular complications in the autoimmune hepatitis group, and in particular hepatic artery thrombosis, which occurred in 17.4% of patients and contributed to early allograft loss in all cases. This observation aligns with previous reports from the UK and France, where hepatic artery thrombosis and ischaemic cholangiopathy were among the causes of allograft loss, alongside recurrent autoimmune hepatitis and chronic rejection [7, 22]. Hepatic artery thrombosis and primary graft dysfunction were also identified as the main causes of allograft loss for all liver transplantation in a recent study [16].

The mechanisms underlying the increased vascular risk in autoimmune hepatitis remain uncertain. Several known risk factors for hepatic artery thrombosis (including younger recipient age, lower body weight, complex arterial anatomy, cytomegalovirus mismatch and graft type) have been described [23, 24]. In our cohort, autoimmune hepatitis recipients were significantly younger but did not differ in cardiovascular risk profile or allograft characteristics compared to the other groups. Of note, nearly half of the patients with autoimmune hepatitis had a concomitant autoimmune condition such as connective tissue disease, which may be associated with prothrombotic states, including antiphospholipid antibodies. However, our study was not designed to formally test this pathway, and we did not observe a clear patient-level correlation between autoimmune comorbidity and thrombotic events. Another possible explanation is altered vascular integrity or endothelial dysfunction in autoimmune hepatitis. Khalaf et al. previously reported friable arterial walls and reduced flow during transplantation in autoimmune hepatitis recipients, attributing these findings to long-term corticosteroid exposure [25]. While some patients in our cohort had prolonged immunosuppression prior to liver transplantation, this was not universal. One patient who developed hepatic artery thrombosis had fulminant autoimmune hepatitis without prior steroid treatment, suggesting that additional mechanisms, possibly intrinsic to autoimmune hepatitis or related immune dysregulation, may be involved. The potential role of antiphospholipid antibodies could also be considered. Autoimmune hepatitis has been associated with the presence of antiphospholipid antibodies, which may contribute to a prothrombotic state even in the absence of full-blown antiphospholipid syndrome [26]. Although we did not systematically screen for anticardiolipin antibodies in our cohort, this warrants further investigation in future studies as their presence could explain a higher incidence of thrombotic events.

Unlike previous studies, we did not observe a significantly higher incidence of rejection or infections in the autoimmune hepatitis group [8, 9, 12, 13]. However, this finding should be interpreted with caution. Protocol liver biopsies are not routinely performed in all Swiss centres, and the limited sample size may have underpowered the analysis for detecting differences in these outcomes. The absence of documented disease recurrence may also reflect the limitations of surveillance practices rather than a true absence of recurrent autoimmune hepatitis.

One of the strengths of our study lies in the use of high-quality, prospectively collected data from the Swiss Transplant Cohort Study, which captures the vast majority of liver transplantation performed in Switzerland. The relatively modest number of autoimmune hepatitis recipients al-

lowed for meticulous individual chart review, ensuring diagnostic accuracy and granular characterisation of post-transplant events. This detailed assessment strengthened the internal validity of our findings.

Nevertheless, our study has limitations. *First,* the small sample size of autoimmune hepatitis recipients, reflecting both the rarity of the disease and the limited national population, reduces statistical power and limits the generalisability of our findings. To address this, we included patients with variant syndromes and fulminant autoimmune hepatitis, accepting a degree of heterogeneity to enhance analytical validity. While this approach may affect group homogeneity, we believe it reflects real-world clinical complexity and increases relevance. Despite our best efforts, our statistically significant results have wide confidence intervals, which may have led to an overestimation of the effect of autoimmune hepatitis on the measured outcomes. *Second,* ascertainment of complications may have differed across groups. autoimmune hepatitis recipients underwent detailed chart review and had a dedicated case report form, whereas non-autoimmune hepatitis recipients relied primarily on routine Swiss Transplant Cohort Study data capture, which may lead to differential detection of complications. In contrast, allograft loss represents a more robust endpoint and is less susceptible to bias. *Third,* residual confounding cannot be excluded, particularly for perioperative and surgical factors and unmeasured prothrombotic states, which were not systematically assessed. *Finally,* the absence of protocol biopsies and routine post-liver transplantation surveillance for recurrent autoimmune hepatitis likely limited our ability to capture subclinical disease recurrence or mild rejection episodes.

Conclusions

In conclusion, within a nationwide cohort, liver transplantation recipients with autoimmune hepatitis experienced significantly higher rates of allograft loss compared to those with other aetiologies, despite comparable long-term survival. Vascular complications, particularly thrombosis, emerged as a potential contributor to poor allograft outcomes, although the observational design and small sample size preclude causal inference. These results support further multicentre work with standardised adjudication of vascular outcomes and systematic assessment of prothrombotic and autoimmune profiles to clarify mechanisms and inform risk-adapted perioperative monitoring and preventive strategies. Despite the limitations, we believe that our study provides a different insight on the complications that can occur in patients with autoimmune hepatitis after liver transplantation. If confirmed in larger studies, our findings would underscore the need for further thromboembolic risk assessment in this group of patients.

Data sharing statement

The data analysed during the current study are available from the corresponding author upon reasonable request.

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Use of AI: The authors declare that generative artificial intelligence (AI) tools were used solely for language editing and refinement of the manuscript. No AI tools were used to generate scientific content, analyse or interpret data, or draw conclusions.

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Potential competing interests

All authors have completed and submitted the International Committee of Medical Journal Editors form for disclosure of potential conflicts of interest. No potential conflict of interest related to the content of this manuscript was disclosed.

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Table S1. Multivariable Cox model for allograft loss

Variable	Hazard ratio (HR)	95% confidence interval (CI)	p value
Liver disease etiology			0.003
Other	1.0 (reference)		
AIH	4.23	1.85-9.64	
Sex			0.56
Female	1.0 (reference)		
Male	1.17	0.69-1.98	
Recipient age (years)	0.98	0.96-1.01	0.17
Pre-LT MELD-score	0.99	0.97-1.01	0.31
Donor age (years)	1.01	1.00-1.02	0.03
Cold ischemia time (hours)	1.09	1.00-1.20	0.05
Body mass index	1.01	1.00-1.02	0.09
Diabetes history			0.23
No	1.0 (reference)		
Yes	1.36	0.83-2.20	

AIH, autoimmune hepatitis; LT, liver transplantation.

Table S2. Multivariable Cox model for overall post-transplant complications

Variable	Hazard ratio (HR)	95% confidence interval (CI)	p value
Liver disease etiology			0.02
Other	1.0 (reference)		
AIH	1.81	1.14-2.89	
Sex			0.46
Female	1.0 (reference)		
Male	1.09	0.87-1.35	
Recipient age (years)	0.99	0.98-1.00	0.24
Pre-LT MELD-score	1.02	1.01-1.03	<0.001
Donor type			<0.001
Deceased	1.0 (reference)		
Living	2.86	1.87-4.37	
Donor age (years)	1.00	1.00-1.01	0.81
Cold ischemia time (hours)	1.00	0.96-1.05	0.97
Rejection episode	4.80	3.59-6.42	<0.001
Induction treatment			0.02
Yes	1.0 (reference)		
No	0.74	0.58-0.96	
Mycophenolate			0.12
Yes	1.0 (reference)		
No	1.19	0.96-1.48	

AIH, autoimmune hepatitis; LT, liver transplantation.

Table S3. Multivariable Cox model for vascular post-transplant complications

Variable	Hazard ratio (HR)	95% confidence interval (CI)	p value
Liver disease etiology			0.002
Other	1.0 (reference)		
AIH	2.86	1.60-5.11	
Pre-LT MELD-score	1.01	1.00-1.03	0.11
Recipient age (years)	1.00	0.98-1.01	0.67
Sex			0.05
Female	1.0 (reference)		
Male	1.40	0.99-1.98	
Diabetes history			0.73
No	1.0 (reference)		
Yes	0.94	0.68-1.31	
Donor age (years)	1.00	0.99-1.01	0.86
Cold ischemia time (hours)	1.00	0.95-1.07	0.89
Platelet inhibitor	1.44	0.96-2.14	0.09

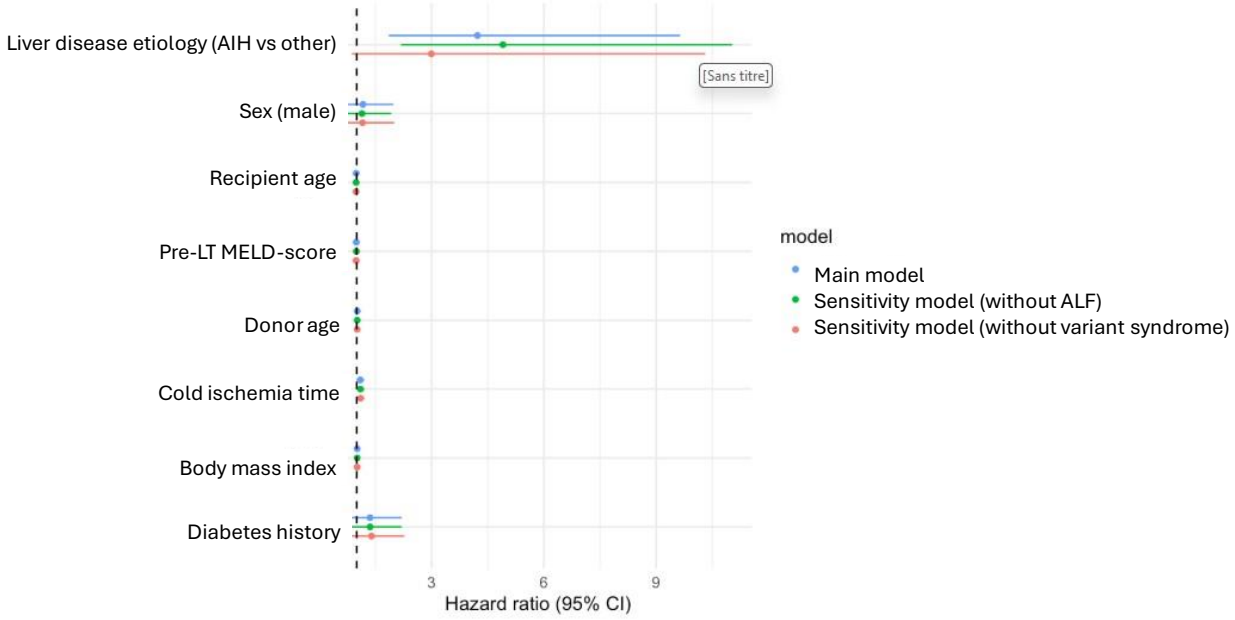
AIH, autoimmune hepatitis; LT, liver transplantation.

Table S4. Multivariable Cox model for thrombotic post-transplant complications

Variable	Hazard ratio (HR)	95% confidence interval (CI)	p value
Liver disease etiology			0.03
Other	1.0 (reference)		
AIH	3.29	1.28-8.48	
Pre-LT MELD-score	1.01	0.98-1.04	0.46
Recipient age (years)	1.01	0.98-1.04	0.51

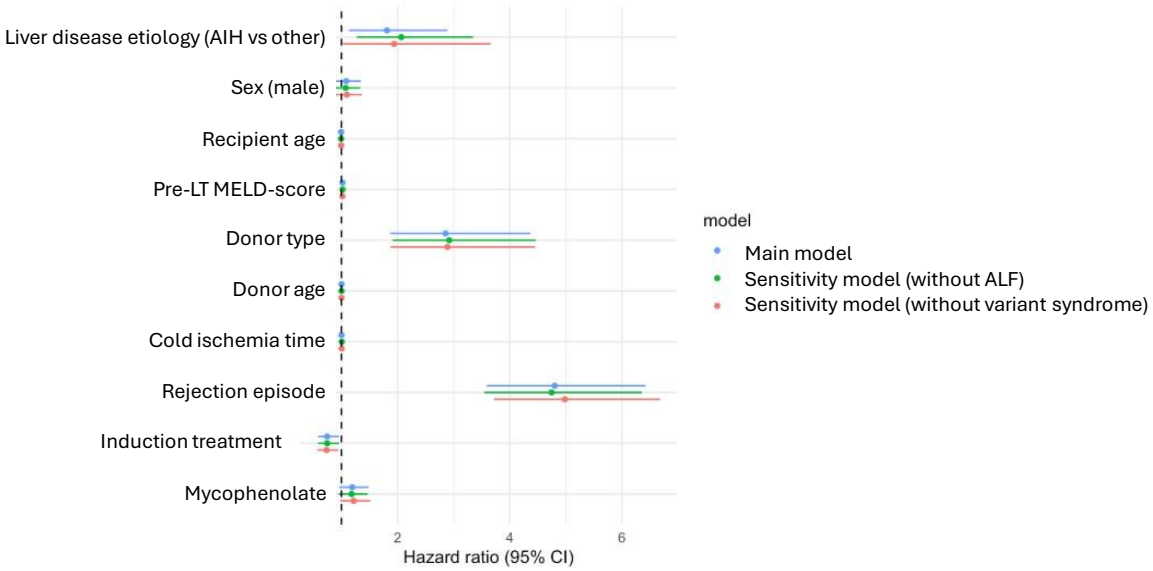
AIH, autoimmune hepatitis; LT, liver transplantation.

Figure S1. Comparison of multivariable Cox models for allograft loss



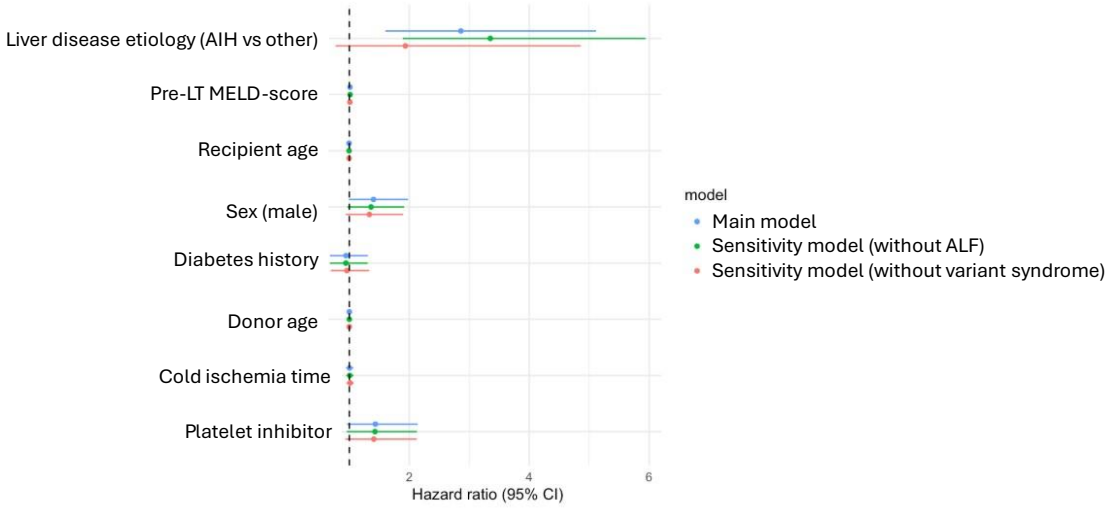
AIH, autoimmune hepatitis; CI, confidence interval ; LT, liver transplantation

Figure S2. Comparison of multivariable Cox models for overall post-transplant complications



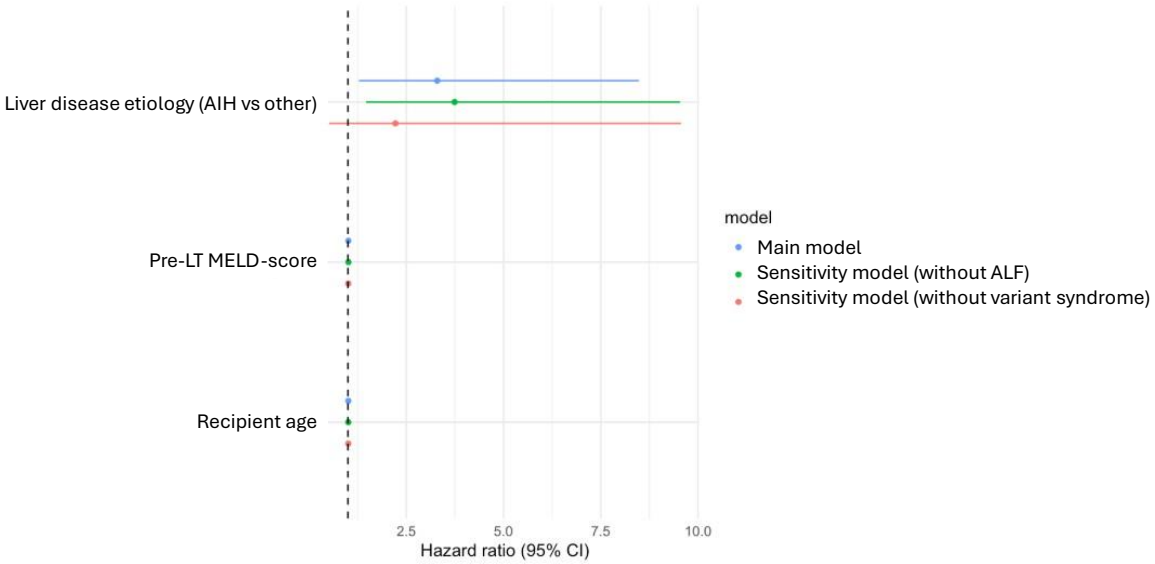
AIH, autoimmune hepatitis; CI, confidence interval ; LT, liver transplantation

Figure S3. Comparison of multivariable Cox models for vascular post-transplant complications



AIH, autoimmune hepatitis; CI, confidence interval ; LT, liver transplantation

Figure S4. Comparison of multivariable Cox models for thrombotic post-transplant complications



AIH, autoimmune hepatitis; CI, confidence interval ; LT, liver transplantation