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Characteristics, predictors and outcomes among 99 patients hospitalised with COVID-19 in a tertiary care centre in Switzerland: an observational analysis

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Summary

AIMS OF THE STUDY: To describe admission characteristics, risk factors and outcomes of patients with coronavirus disease 2019 (COVID-19) hospitalised in a tertiary care hospital in Switzerland during the early phase of the pandemic.

METHODS: This retrospective cohort study included adult patients with a severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection confirmed by polymerase chain reaction (PCR) testing and hospitalised at the cantonal hospital Aarau (Switzerland) between 26 February 2020 and 30 April 2020. Our primary endpoint was severe COVID-19 progression defined as a composite of transfer to the intensive care unit (ICU) and in-hospital mortality.

RESULTS: A total of 99 patients (median age 67 years [interquartile range 56–76], 37% females) were included and 35% developed severe COVID-19 progression (24% needed ICU treatment, 19% died). Patients had a high burden of comorbidities with a median Charlson comorbidity index of 3 points and a high prevalence of hypertension (57%), chronic kidney disease (28%) and obesity (27%). Baseline characteristics with the highest prognostic value for the primary endpoint by means of area under the receiver operating characteristic curve were male gender (0.63) and initial laboratory values including shock markers (lactate on ambient air 0.67; lactate with O_2 supply 0.70), markers of inflammation (C-reactive protein 0.72, procalcitonin 0.80) and markers of compromised oxygenation (pO_2 0.75 on ambient air), whereas age and comorbidities provided little prognostic information.

CONCLUSION: This analysis provides insights into the first consecutively hospitalised patients with confirmed COVID-19 at a Swiss tertiary care hospital during the initial period of the pandemic. Markers of disease progression such as inflammatory markers, markers for shock and impaired respiratory function provided the most prognostic information regarding severe COVID-19 progression in our sample.

Keywords: *baseline characteristics, COVID-19, in-hospital mortality, intensive care, prognostic markers, SARS-CoV-2, Switzerland*

Introduction

Since its first description in China [1, 2], severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), a novel coronavirus, has led to a global pandemic with severe respiratory disease. The number of affected patients suffering from coronavirus disease 2019 (COVID-19) continues to rise, at the time of writing with more than eight million confirmed cases and large numbers of deaths worldwide [3].

Several smaller studies and case reports in Switzerland have already examined different aspects of COVID-19, such as the impact of nonpharmaceutical interventions on disease transmission [4], the utility of diagnostic testing [5], early off-label treatments [6] and the impact of digital health aspects [7, 8]. Also, information regarding the reproductive number of COVID-19 with focus on specific cantons [9], as well as estimates of case fatality rates [10] have been published. These reports have provided important information to better understand this novel disease.

However, local information describing clinical presentation, patient characteristics, risk factors for deterioration and outcomes of hospitalised COVID-19 patients in Switzerland is still lacking. This information is crucial for estimating patient morbidity and mortality and need for hospital resource allocation, and hence supports strategic decisions in Switzerland. Therefore, the purpose of this study was to describe the presenting characteristics, risk

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factors and outcomes of the first 99 COVID-19 patients admitted during the initial 2 months of the pandemic to the Cantonal Hospital Aarau, a tertiary care centre in the northern part of Switzerland.

Methods

This retrospective cohort study involved all consecutively hospitalised adult patients (≥18 years) with a confirmed SARS-CoV-2 infection at the Cantonal Hospital Aarau (Switzerland), a tertiary care hospital with 120 medical ward beds, between 26 February 2020 and 30 April 2020. The study was approved by the ethics committee (EKZN, 2020-01306). Four patients were excluded from the analysis as they did not provide general informed consent. We included only hospitalised patients and excluded outpatients. In our centre, indications for hospitalisation were respiratory distress, high fever, or relevant clinical deterioration.

Definition of a confirmed COVID-19 infection was typical clinical symptoms (e.g., respiratory symptoms with or without fever, and/or pulmonary infiltrates and/or anosmia/dysgeusia) together with a positive real-time reverse transcription polymerase chain reaction test (RT-PCR) taken from nasopharyngeal swabs or lower respiratory tract specimens, according to the World Health Organization (WHO) guidance [11]. All analysed data were assessed as part of the clinical routine during the hospitalisation (from admission to discharge/death).

Data collection

We collected clinical data by chart abstraction and automatic export from internal medical data. Specifically, vital signs and clinical characteristics upon admission were recorded. Clinical information, including sociodemographics and comorbidities, home medications and COVID-19-specific inpatient medication were assessed until hospital discharge or death and exported from the hospital electronic clinical information system. Experimental treatment was offered to all patients and included, for hospital ward patients, hydroxychloroquine only (first line) and lopinavir/ritonavir, and for intensive care unit (ICU) patients, tocilizumab. Azithromycin was also used in patients transferred from France. For all patients the age-adjusted Charlson comorbidity index [12] and the Clinical Frailty Score (up to 9 points) [13] were calculated as part of the clinical routine. Comorbidities were also assessed through chart review and based on the International classification of Diseases, 10th edition (ICD10) code. Further, patient outcomes including in-hospital mortality, admission to the ICU, length of hospital stay (LOS) and length of ICU stay were collected by chart review. Laboratory test results were available according to clinical routine. Laboratory values correspond to the first blood draw obtained within 24 hours after admission.

Outcomes

Our primary endpoint was severe COVID-19 progression defined as a composite of transfer to ICU during the index hospital stay and all-cause in-hospital mortality, both verified by chart review. This composite outcome was chosen since the aim was to show the severity of patients with COVID-19 either by ICU admission or by mortality.

Statistical analysis

Discrete variables are expressed as frequency (percentage) and continuous variables as medians with interquartile ranges (IQRs) or means with standard deviations (SDs). In addition to descriptive statistics, we also investigated the association of baseline risk factors with the primary endpoint by logistic regression analysis reporting odds ratios (ORs) with corresponding 95% confidence intervals (CIs) and p-values as a measure of association. The p-values were derived from the regression models based on likelihood ratio chi-square tests. Areas under the receiver operating characteristic curve (AUC) were calculated as a measure of discrimination. A two-sided p-value of <0.05 was considered significant. Statistical analysis was performed using Stata 15.1 (StataCorp, College Station, TX, USA).

Results

Figure 1 provides an overview of the study flow. A total of 99 patients hospitalised with a confirmed SARS-CoV-2 infection were included in this analysis (median age 67 years [IQR 56–76]; 37% females). A total of 28 patients were transferred from other hospitals (two cases from France, one case from the Canton Ticino, 25 cases from regional hospitals not accepting COVID-19 admissions or when treatment at a tertiary care hospital was indicated). Median time from symptom onset prior to presentation to the emergency department was 8 days (IQR 5–11).

Table 1 shows patient demographics, comorbidities based on pre-existing ICD-10 diagnoses according to electronic medical records, as well as vital signs and laboratory findings at admission in the overall cohort and stratified according to the primary endpoint. The evaluation of home medication revealed that 22% of patients were taking angiotensin converting-enzyme inhibitors, 18% angiotensin II receptor blockers and few patients were taking corticosteroids or other immunosuppressive treatments. Patients had a high burden of comorbidities, with a median Charlson comorbidity index of 3 points and a high median frailty score of 3 points. Most common comorbidities included hypertension (57%, n = 56), chronic kidney disease (28%, n = 28) and obesity (27%, n = 27). A majority of patients presented with high clinical severity, particularly regarding

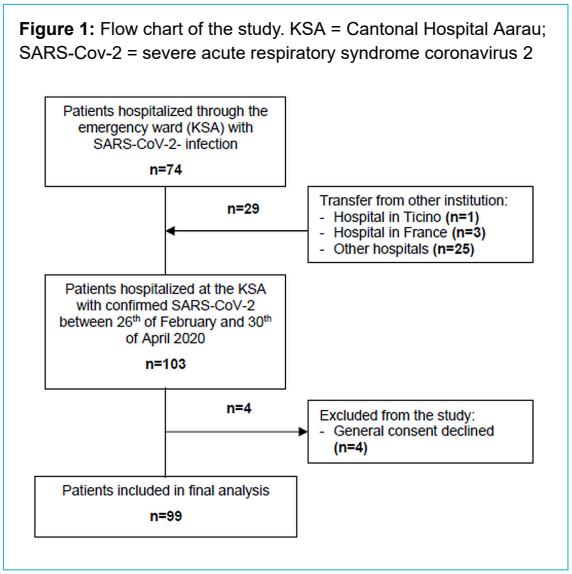

Figure 1: Flow chart of the study. KSA = Cantonal Hospital Aarau; SARS-Cov-2 = severe acute respiratory syndrome coronavirus 2

the respiratory system with a high respiratory rate and evidence of compromised oxygenation in blood gas analysis. There was also a modest increase in the inflammation marker C-reactive protein (CRP) (mean level 78.5 mg/l) and a slight increase in procalcitonin (PCT) levels (0.11 µg/l).

In-hospital treatments and outcomes of the hospitalised patients are presented in table 2. Overall, 47% of patients received an experimental antiviral treatment (mostly hydroxychloroquine, rarely ritonavir-boosted lopinavir). A total of 35% (n = 35) of patients developed severe COVID-19 progression characterised by a need for ICU treatment (24%, n = 24, including mechanical ventilation in 19 patients) and/or death (19%, n = 18). Overall, patients hospitalised because of COVID-19 had a median length of stay of 8.5 days (IQR 4.0–14.5).

We also investigated which baseline factors were associated with our primary endpoint by means of association and discrimination. Several baseline factors were associated with severe COVID-19 progression with, however, only modest prognostic value regarding differentiation between severe and non-severe COVID-19 infection. These included male gender (OR for severe COVID-19 progression of female vs male patients 0.28, 95% CI 0.11–0.74; p = 0.010; AUC 0.63) and markers for disease progression including shock markers (lactate on ambient air OR per unit increase 2.41, 95% CI 0.85–6.83; p = 0.097; AUC 0.67; lactate with O_2 supply OR per unit increase 2.77, 95% CI 0.63–12.10; p = 0.176; AUC 0.70), markers of inflammation (for CRP, OR per unit increase 1.01, 95% CI 1.00–1.02; AUC 0.72 and for PCT, OR per µg/l increase 5.16, 95% CI 4.18–713.45; AUC 0.80) and respiratory failure (pO_2 OR per mm Hg increase 0.93, 95% CI 0.88–0.99; AUC 0.75 in patients on ambient air; FiO_2 OR per unit increase 1.04, 95% CI 1.00–1.08; AUC 0.76 in those requiring oxygen substitution). Receiver operating characteristic curves for the two most predictive variables, namely FiO_2 in patients with O_2 supply and PCT, are presented in figures 2 and 3. Patient age (AUC 0.53), frailty score (AUC 0.50) and the Charlson comorbidity index (AUC 0.54), as well as single comorbidities were not associated with severe COVID-19 progression and thus provided little prognostic information.

Discussion

Several smaller studies and case reports in Switzerland have examined different aspects of care for patients infected with SARS-CoV-2 in Switzerland. However, to our knowledge, this current study represents one of the first descriptions of consecutively hospitalised COVID-19 patients during the initial pandemic in Switzerland. In this population of hospitalised patients, older males with pre-existing hypertension, chronic kidney disease and obesity were highly prevalent. These data are consistent with the data reported from New York, US [14], as well as from Wuhan, China [15] and Lombardy, Italy [16], but has some important differences, which may have implications for patient care in the future and thus deserve further commenting.

First, median LOS in our sample of COVID-19 patients was 8.5 days (mean 10.7 days) and therefore significantly longer than for prior patients with pneumonia hospitalised in the same institution, who had an average LOS of 5 to 6 days [17, 18]. Reports from Wuhan described a LOS of 11 days [15], whereas patients in New York had a markedly shorter LOS of only 4 days [14]. The longer LOS in COVID-19 patients found in our and the Wuhan cohorts may indicate that these patients needed more time to recover and longer monitoring periods compared with patients with other types of respiratory tract infection, but the short LOS in patients from New York is surprising [14]. We speculate that either the US health system has a very efficient logistic system with early discharge of patients to other healthcare facilities after initial stabilisation, or milder cases of COVID-19 infection were hospitalised with early discharge, thereby reducing the average LOS in the overall cohort. Also, the timing of admission may vary within different healthcare systems, which depends on available resources and the number of infected patients and strongly influences LOS and mortality. In our institution, we mainly hospitalised COVID-19 patients with severe distress and clinical deterioration, but not clinically stable patients for the purpose of monitoring. This may explain the high severity, the long average LOS and also the high frequency of patients transferred to ICU. Also, in our clinical experience, it was challenging to discharge patients to nursing facilities because of the presumed risk that af-

Figure 2: Receiver operating characteristic (ROC) curve of FiO_2 in patients with O_2-supply as strong predictor of the composite outcome (ICU admission and/or mortality) with an area under the curve (AUC) of 0.76 (95% CI 0.56–0.95).

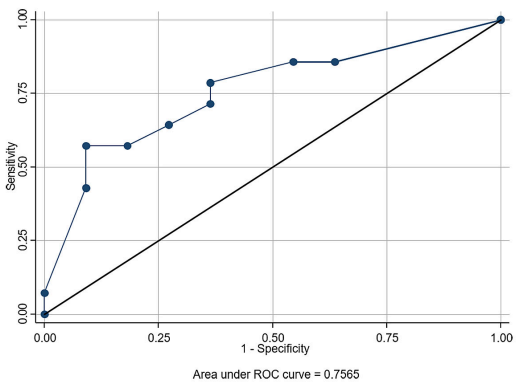

Figure 3: Receiver operating characteristic (ROC) curve of procalcitonin as strong predictor of the composite outcome (ICU admission and/or mortality) with an area under the curve (AUC) of 0.80 (95% CI 0.71–0.90).

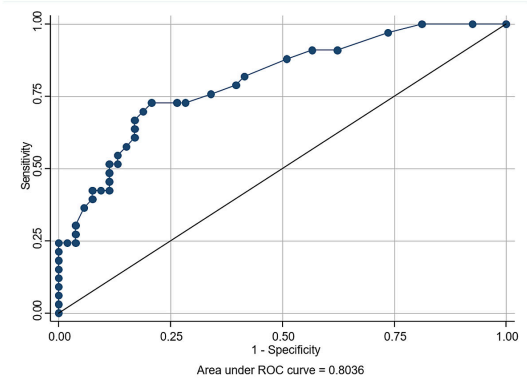

Table 1: Baseline characteristics, vital signs and laboratory results of patients hospitalised owing to COVID-19 at initial presentation in the emergency department.

	Overall n = 99	Patients without severe COVID-19 progression n = 64	Patients with severe COVID-19 progression n = 35	Univariate OR (95% CI) p-value	AUC (95% CI)
Sociodemographics					
Age (years), median (IQR)	67.0 (56.0–76.0)	63.5 (56.0–76.0)	69.0 (57.0–75.0)	1.19 (0.67–2.09) p = 0.553	0.53 (0.41–0.65)
Female sex, n (%)	37 (37%)	30 (47%)	7 (20%)	0.28 (0.11–0.74) p = 0.010	0.63 (0.54–0.73)
Nationality, n (%)					
– France	3 (3%)	0 (0%)	3 (9%)		
– Italy	6 (6%)	2 (3%)	4 (11%)		
– Switzerland	60 (61%)	43 (67%)	17 (48%)		
– Turkey	4 (4%)	4 (6%)	0 (0%)		
– Others	10 (10%)	8 (13%)	2 (6%)		
– Unknown	16 (16%)	7 (11%)	9 (26%)		
Pre-existing risk factors and medication					
Active smoker, n (%)	6/76 (8%)	4/52 (8%)	2/24 (8%)	1.09 (0.19–6.41) p = 0.923	0.50 (0.44–0.57)
Corticosteroid use, n (%)	2 (2%)	1 (2%)	1 (3%)	1.85 (0.11–30.56) p = 0.666	0.51 (0.47–0.54)
Immunosuppressant, n (%)	4 (4%)	2 (3%)	2 (6%)	1.88 (0.25–13.95) p = 0.538	0.51 (0.47–0.56)
Angiotensin converting-enzyme inhibitor, n (%)	22 (22%)	13 (20%)	9 (26%)	1.36 (0.51–3.59) p = 0.537	0.53 (0.44–0.62)
Angiotensin II receptor blocker, n (%)	18 (18%)	13 (20%)	5 (14%)	0.65 (0.21–2.02) p = 0.459	0.53 (0.45–0.61)
Pre-admission history					
Symptom onset before admission (days), median (IQR)	8.0 (5.0–11.0) n = 85	7.0 (4.0–10.0) n = 56	8.0 (5.0–12.0) n = 29		
Transfer from another hospital, n (%)	28 (28%)	11 (17%)	17 (49%)		
Comorbidities					
Charlson comorbidity index, median (IQR)	3.0 (2.0–6.0)	3.0 (2.0–7.0)	4.0 (2.0–6.0)	1.03 (0.90–1.17) p = 0.696	0.54 (0.42–0.66)
Clinical frailty score, median (IQR)	3.0 (2.0–4.0) n = 76	3.0 (2.0–4.5) n = 52	3.0 (2.0–4.0) n = 24	0.97 (0.72–1.32) p = 0.864	0.5 (0.36–0.63)
Cancer, n (%)	11 (11%)	4 (6%)	7 (20%)	3.75 (1.01–13.87) p = 0.048	0.57 (0.5–0.64)
Hypertension, n (%)	56 (57%)	37 (58%)	19 (54%)	0.87 (0.38–1.99) p = 0.735	0.52 (0.41–0.62)
Coronary artery disease, n (%)	25 (25%)	16 (25%)	9 (26%)	1.04 (0.40–2.67) p = 0.938	0.50 (0.41–0.59)
Chronic heart failure, n (%)	3 (3%)	3 (5%)	0 (0%)	NA	0.52 (0.49–0.55)
Asthma, n (%)	16 (16%)	11 (17%)	5 (14%)	0.80 (0.25–2.53) p = 0.708	0.51 (0.44–0.59)
COPD, n (%)	7 (7%)	3 (5%)	4 (11%)	2.62 (0.55–12.46) p = 0.225	0.53 (0.47–0.59)
Obstructive sleep apnoea, n (%)	14 (14%)	7 (11%)	7 (20%)	2.04 (0.65–6.37) p = 0.222	0.55 (0.47–0.62)
Solid organ transplant recipient, n (%)	1 (1%)	1 (2%)	0 (0%)	NA	0.51 (0.49–0.52)
Rheumatic disease, n (%)	2 (2%)	1 (2%)	1 (3%)	1.85 (0.11–30.56) p = 0.666	0.51 (0.47–0.54)
Chronic kidney disease, n (%)	28 (28%)	18 (28%)	10 (29%)	1.02 (0.41–2.55) p = 0.962	0.50 (0.41–0.60)
Obesity (BMI >30 kg/m^2), n (%)	27 (27%)	15 (23%)	12 (34%)	1.70 (0.69–4.22) p = 0.249	0.55 (0.46–0.65)
Diabetes, n (%)	22 (22%)	14 (22%)	8 (23%)	1.06 (0.39–2.84) p = 0.911	0.50 (0.42–0.59)
Vital signs					
SpO_2 (%), median (IQR)	93.2 (88.9–95.5) n = 72	94.3 (91.6–95.7) n = 49	89.8 (82.2–94.4) n = 23	0.89 (0.81–0.97) p = 0.007	0.71 (0.58–0.84)
Blood pressure, systolic (mm Hg), median (IQR)	139.0 (126.0–156.0) n = 71	133.0 (123.0–152.0) n = 49	143.5 (137.0–159.0) n = 22	1.02 (1.00–1.05) p = 0.064	0.63 (0.49–0.77)

	Overall n = 99	Patients without severe COVID-19 progression n = 64	Patients with severe COVID-19 progression n = 35	Univariate OR (95% CI) p-value	AUC (95% CI)
Blood pressure, diastolic (mm Hg), median (IQR)	81.0 (71.0–88.0) n = 71	81.0 (70.0–88.0) n = 49	83.0 (73.0–87.0) n = 22	1.01 (0.97–1.04) p = 0.731	0.53 (0.38–0.67)
Pulse (bpm), median (IQR)	85.6 (77.0–96.4) n = 69	84.2 (77.0–92.2) n = 47	87.0 (71.4–98.0) n = 22	1.01 (0.98–1.05) p = 0.419	0.52 (0.36–0.69)
Respiratory rate (breaths/min), median (IQR)	21.3 (17.0–27.0) n = 55	20.3 (17.3–24.3) n = 36	25.6 (16.0–30.1) n = 19	1.03 (0.96–1.12) p = 0.382	0.64 (0.46–0.82)
Temperature (°C), median (IQR)	37.7 (37.3–38.3) n = 71	37.7 (37.2–38.3) n = 48	37.8 (37.3–38.4) n = 23	1.07 (0.57–1.98) p = 0.836	0.55 (0.41–0.69)
Temperature >38°C, n (%)	54 (55%)	32 (50%)	22 (63%)	1.69 (0.73–3.93) p = 0.221	0.56 (0.46–0.67)
Laboratory results*					
Blood gas analysis on ambient air, n (%)	73 (100%)	52 (71%)	21 (29%)		
– pO_2 (mm Hg), median (IQR)	68 (61–73)	69 (64–74)	57 (42–68)	0.93 (0.88–0.99) p = 0.024	0.75 (0.55–0.94)
– pCO_2 (mm Hg), median (IQR)	31 (28–33)	32 (29–33)	31 (27–32)	0.95 (0.79–1.12) p = 0.523	0.60 (0.40–0.80)
– Lactate (mmol/l), median (IQR)	1.2 (0.9–1.6)	1.1 (0.8–1.5)	1.3 (1.2–1.6)	2.41 (0.85–6.83) p = 0.097	0.67 (0.51–0.83)
– FiO_2 (%), median (IQR)	21	21	21	NA	NA
Blood gas analysis on initial O_2 supply, n (%)	25 (100%)	11(44%)	14 (56%)		
– pO_2 (mm Hg), median (IQR)	65 (56–76)	64 (47–102)	66 (56–73)	1.00 (0.96–1.03) p = 0.907	0.51 (0.26–0.77)
– pCO_2 (mm Hg), median (IQR)	32 (31–35)	32 (31–33)	33 (31–35)	0.98 (0.83–1.15) p = 0.783	0.56 (0.32–0.80)
– Lactate (mmol/l), median (IQR)	1.3 (1.0–1.5)	1.1 (0.7–1.4)	1.4 (1.1–1.5)	2.77 (0.63–12.10) p = 0.176	0.70 (0.48–0.92)
– FiO_2 (%), median (IQR)	44 (32–95)	36 (28–50)	70 (40–95)	1.04 (1.00–1.08) p = 0.038	0.76 (0.56–0.95)
Blood test results					
– Leucocytes (G/l), median (IQR)	7.1 (4.6–9.2) n = 96	6.9 (4.4–8.6) n = 61	7.5 (5.1–9.4)	1.09 (0.97–1.22) p = 0.149	0.58 (0.46–0.70)
– Lymphocytes (G/l), median (IQR)	0.84 (0.55–1.19) n = 75	0.84 (0.60–1.46) n = 49	0.83 (0.43–1.09) n = 26	0.43 (0.15–1.20) p = 0.107	0.59 (0.45–0.73)
– Neutrophil to lymphocyte ratio, median (IQR)	5.45 (3.43–8.49) n = 74	4.30 (3.11–7.80) n = 60	6.32 (5.42–12.19) n = 14	1.08 (0.97–1.19) p = 0.152	0.65 (0.49–0.81)
– Sodium (mmol/l), median (IQR)	136.0 (133.0–139.0) n = 95	136.0 (133.0–138.0) n = 61	137.0 (134.0–139.0) n = 34	1.05 (0.93–1.19) p = 0.431	0.57 (0.44–0.70)
– Glucose (mmol/l), median (IQR)	6.6 (5.7–8.2) n = 86	6.4 (5.6–7.4) n = 57	7.3 (6.4–8.6) n = 29	1.12 (0.94–1.34) p = 0.201	0.65 (0.52–0.77)
– CRP (mg/l), median (IQR)	79 (35–148) n = 96	62 (25–124) n = 61	123 (76.9–189)	1.01 (1.00–1.02) p = 0.001	0.72 (0.62–0.83)
– PCT (µg/l), median (IQR)	0.105 (0.05–0.3) n = 86	0.08 (0.04–0.14) n = 53	0.26 (0.11–0.61) n = 33	54.61 (4.18–713.45) p = 0.002	0.80 (0.71–0.90)
– Creatinine (µmol/l), median (IQR)	93.0 (74.0–122.0) n = 95	93.0 (74.0–119.0) n = 61	97.0 (82.0–133.0) n = 34	1.00 (0.99–1.01) p = 0.104	0.55 (0.43–0.68)
– ALAT (U/l), median (IQR)	36.0 (25.5–49.0) n = 88	32.0 (24.0–44.5) n = 56	43.0 (28.5–62.0) n = 32	1.03 (1.01–1.05) p = 0.013	0.66 (0.53–0.78)
– Alkaline phosphatase (U/l), median (IQR)	68.0 (54.5–87.0) n = 84	65.5 (52.0–81.0) n = 54	75 (60–108.0) n = 30	1.01 (1.00–1.02) p = 0.021	0.63 (0.51–0.76)

ALAT = alanine aminotransferase; AUC = area under the curve; BMI = body mass index; bpm = beats per minute; CI = confidence interval; COPD = chronic obstructive pulmonary disease = CRP = C-reactive protein; FiO_2 = fraction of inspired oxygen; ICU = intensive care unit; IQR = interquartile range; NA = not applicable; OR = odds ratio; pCO_2 = partial pressure of carbon dioxide; PCT = procalcitonin; pO_2 = partial pressure of oxygen; SpO_2 = oxygen saturation * Laboratory results correspond to first blood draw obtained within 24 h after admission.

fected patients would infect other people in these facilities. The lack of a laboratory or clinical parameter indicating when a patient is finally cured and not infectious anymore has important clinical consequences. PCR is not an optimal method to classify patients as infectious or not, as PCR may remain positive for a long period of time despite the virus not being viable.

Second, comparison of different international cohorts with patients affected by COVID-19 from China, the US and Europe suggests that our Swiss cohort and the US patients [14] were comparable with regard to age, gender and most comorbidities. The prevalence of obesity was higher in the US at 41% compared with Switzerland (only 27%). Based on available reports, patients from Wuhan [15] had a lower burden of comorbidities. This may be explained by a difference in the quality of data collection and reporting, or

by a different time during the pandemic affecting different segments of the population when these cohorts were reported, as the Wuhan cohort came from a very early phase in the pandemic (December 2019 / January 2020), when there was a lack of information regarding this novel disease.

Third, looking at clinical outcomes, a comparable rate of ICU care is reported in our data: 24% compared with 26% in Wuhan [15]. The US, however, reported a very low ICU admission rate of only 14%, again indicating that severity of illness in their hospitalised patient population was lower or they used stricter criteria for ICU admission [14]. Our Swiss cohort had a mortality rate of 18%, which was lower than in the Wuhan cohort (28%) [15] and similar to the US cohort (21%) [14]. Given the differences in healthcare systems and in selection of patients for in-hospital treatment, it is difficult to draw any strong conclusions from these data.

Forth, this analysis showed an association of different admission laboratory findings with severe COVID-19 progression, such as markers of inflammation, shock and respiratory failure among others. These findings were also confirmed for the COVID-19 cohort in Wuhan [15]. Interestingly, in Wuhan, different comorbidities such as chronic obstructive lung disease, coronary heart disease and hypertension were significantly associated with mortality. In our cohort, however, we found that comorbidities and patient age provided little prognostic information regarding severe COVID-19 progression and the initial clinical presentation was much more important regarding adverse inpatient outcomes. Although the small sample size of our cohort may have masked significant associations (type II error), the AUC of receiver operating characteristic curves as a measure of discrimination suggests that the outcome may be more dependent upon severity of initial presentation in regard to laboratory findings, rather than patient characteristics. Importantly, our cohort was highly selected and only included severe COVID-19 patients seeking inpatient medical care at a tertiary care centre, whereas previous reports also included outpatients and patients with lower disease severity. It would thus be interesting to validate our findings in a larger patient group within other healthcare settings. Furthermore, parameters of disease severity on admission strongly depended on the time-point of admission and performed better the closer hospitalisation was from ICU-transfer/death. In institutions with softer indications for hospitalisation (i.e., with hospitalisation for monitoring of patients with low severity disease), the predictive value of these factors may be lower. Despite the preliminary nature of our data, the findings of our report may still be important for selection of patients for ICU treatment based on age or comorbidities (rationing of care). As age and pre-existing lung and cardiac disease were not strong predictors of severe COVID-19 outcome, these parameters *per se* seem not to be useful for the decision regarding ICU admission in a Swiss tertiary care setting.

This early report has limitations. First, because of the retrospective single centre study design, not all evaluated laboratory parameters and characteristics were available for all patients, resulting in some missing data. Also, some of the most valuable laboratory values to predict outcome, such as D-dimers or serum ferritin, were not routinely collected in our hospital. Third, some patients were transferred to other hospitals and it was not possible to verify their outcome. Most of these patients, however, showed a favourable treatment response before transfer to another hospital. Also, COVID-19 specific treatment was not standardised in our institution and indications may have changed over time when evolving results from trials becoming available. We did not perform multivariable analysis because, due to the small number of patients and outcomes, the main purpose was not to derive a multivariable prediction model, but rather to provide a timely overview of COVID-19 patient hospitalised at our centre. However, we strongly encourage such an effort using data of multiple Swiss centres, which would be much more generalisable. Also, the low number of patients has limited the statistical evaluation and risk for type II error. Last, the study population only included patients within one Swiss region and one hospital. Nevertheless, this study highlights some characteristics of patients affected by COVID-19 in Switzerland and allows some early insights for this pandemic disease.

Table 2: In-hospital treatment and endpoints of patients hospitalised owing to COVID-19.

	Overall n = 99	Patients without severe COVID-19 progression n = 64	Patients with severe COVID-19 progression n = 35
In-hospital treatment			
Treatment specification, n (%)			
– Hydroxychloroquine	39 (39%)	23 (36%)	16 (46%)
– Hydroxychloroquine + azithromycin	3 (3%)	0 (0%)	3 (9%)
– Hydroxychloroquine + tocilizumab	1 (1%)	0 (0%)	1 (3%)
– Lopinavir/ritonavir	2 (2%)	1 (2%)	1 (3%)
– Tocilizumab	2 (2%)	0 (0%)	2 (6%)
– Symptomatic treatment only	52 (53%)	40 (63%)	12 (34%)
Antibiotic treatment, n (%)	41 (41%)	14 (22%)	27 (77%)
In-hospital endpoints			
In-hospital mortality, n (%)	18 (19%)	0 (0%)	18 (55%)
ICU care, n (%)	24 (24%)	0 (0%)	24 (69%)
Length of stay, median (IQR)	8.5 (4.0, 14.5)	6.0 (4.0, 10.0)	19.0 (9.0, 24.0)
Need for mechanical ventilation, n (%)	19 (19%)	0 (0%)	19 (54%)

ICU = intensive care unit; IQR = interquartile range; SD = standard deviation

Conclusion

This analysis provides an insight into admission characteristics, risk factors and outcomes of the first hospitalised patients with confirmed COVID-19 at a Swiss tertiary care hospital during the initial phase of the pandemic. Severity of initial presentation assessed through initial blood markers and to a lesser extent male gender provided the most prognostic information in our small patient sample, whereas age and comorbidities provided little additional information. Our findings are preliminary, however serve as basis for further investigations concerning prognostication of hospitalised COVID-19 patients.

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