



BMJ Open Retrospective cohort study of clinical characteristics of 2199 hospitalised patients with COVID-19 in New York City

Ishan Paranjpe ¹, Adam J Russak,^{1,2} Jessica K De Freitas,^{1,3} Anuradha Lala,^{4,5} Riccardo Miotto,^{1,3} Akhil Vaid,^{1,3} Kipp W Johnson,^{1,3} Matteo Danieletto,^{1,3} Eddy Golden,^{1,3} Dara Meyer,³ Manbir Singh,¹ Sulaiman Somani,¹ Arjun Kapoor,¹ Ross O'Hagan,¹ Sayan Manna,¹ Udit Nangia,¹ Suraj K Jaladanki ¹, Paul O'Reilly,^{3,6} Laura M Huckins,^{3,6} Patricia Glowe,^{1,3} Arash Kia,^{5,7} Prem Timsina,^{5,7} Robert M Freeman,^{5,7} Matthew A Levin,^{3,5,7,8} Jeffrey Jhang,⁹ Adolfo Firpo,⁹ Patricia Kovatch,¹⁰ Joseph Finkelstein,⁵ Judith A Aberg,^{11,12} Emilia Bagiella,^{4,5} Carol R Horowitz,^{5,12} Barbara Murphy,¹² Zahi A Fayad,^{13,14} Jagat Narula,^{12,15} Eric J Nestler,^{16,17} V Fuster,¹⁸ Carlos Cordon-Cardo,⁹ Dennis Charney,^{19,20} David L Reich,²¹ Allan Just,^{22,23} Erwin P Bottinger,¹ Alexander W Charney,^{3,6} Benjamin S Glicksberg,^{1,3,6} Girish N Nadkarni,^{1,12,24} On behalf of Mount Sinai COVID Informatics Center (MSCIC)

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IP, AJR, JKDF and AL contributed equally.

EPB, AWC, BSG and GNN are joint senior authors.

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For numbered affiliations see end of article.

Correspondence to

Mr Ishan Paranjpe;
iparanjpe@berkeley.edu

ABSTRACT

Objective The COVID-19 pandemic is a global public health crisis, with over 33 million cases and 999 000 deaths worldwide. Data are needed regarding the clinical course of hospitalised patients, particularly in the USA. We aimed to compare clinical characteristic of patients with COVID-19 who had in-hospital mortality with those who were discharged alive.

Design Demographic, clinical and outcomes data for patients admitted to five Mount Sinai Health System hospitals with confirmed COVID-19 between 27 February and 2 April 2020 were identified through institutional electronic health records. We performed a retrospective comparative analysis of patients who had in-hospital mortality or were discharged alive.

Setting All patients were admitted to the Mount Sinai Health System, a large quaternary care urban hospital system.

Participants Participants over the age of 18 years were included.

Primary outcomes We investigated in-hospital mortality during the study period.

Results A total of 2199 patients with COVID-19 were hospitalised during the study period. As of 2 April, 1121 (51%) patients remained hospitalised, and 1078 (49%) completed their hospital course. Of the latter, the overall mortality was 29%, and 36% required intensive care. The median age was 65 years overall and 75 years in those who died. Pre-existing conditions were present in 65% of those who died and 46% of those discharged. In those who died, the admission median lymphocyte percentage was 11.7%, D-dimer was 2.4 µg/mL, C reactive protein was 162 mg/L and procalcitonin was 0.44 ng/mL. In those discharged, the admission median lymphocyte percentage

Strengths and limitations of this study

- Our cohort includes greater racial diversity as compared with previously published work.
- Largest hospitalised cohort of patients with COVID-19 in New York City.
- Limited by lack of access to clinical notes.
- Our study is limited by the short follow-up time period.
- Our study is limited by high censoring rate.

was 16.6%, D-dimer was 0.93 µg/mL, C reactive protein was 79 mg/L and procalcitonin was 0.09 ng/mL.

Conclusions In our cohort of hospitalised patients, requirement of intensive care and mortality were high. Patients who died typically had more pre-existing conditions and greater perturbations in inflammatory markers as compared with those who were discharged.

INTRODUCTION

The COVID-19 pandemic, caused by SARS-CoV-2, has held the world at a standstill with its virulence. As of 28 September 2020, over 33 million people have been affected, and >999 000 patients have died worldwide.¹ In addition to being highly contagious, the disease manifestations and clinical course are variable, spanning from asymptomatic status to severe acute respiratory distress syndrome with multiorgan failure and

death,^{2,3} acute kidney injury,^{4,5} acute myocardial injury⁶ and coagulopathy.⁷⁻¹¹

Reports from China and Italy provided early data on disease presentation and management¹²⁻¹⁴ but also revealed varying geographic disease expressions. Nearly one-third of the world's cases are now in the USA, and with nearly one-third of those cases located in New York, it represents the current epicentre of the COVID-19 pandemic.¹⁵

As the number of cases continues to climb, hospitals are being stretched well-beyond capacity while facing challenges of insufficient personal protective equipment, ventilators and workforce. Thus, understanding the clinical course of hospitalised patients with COVID-19 is critical for providing optimal patient care and to inform resource management in other locations across the USA likely to experience similar case surges.¹⁶ Data specifically examining differences in admission laboratory data in patients who died as compared with those who were ultimately discharged are lacking.

The Mount Sinai Healthcare System (MSHS) is the largest academic health system in New York City and serves as an ideal platform to better understand the evolving landscape of COVID-19 across a diverse population. Here, we present the largest case series of patients hospitalised with laboratory confirmed COVID-19 to date in the USA.

METHODS

Patient involvement

Patients and the public were not directly involved in the study design or implementation.

Study population

The MSHS serves a large, racially and ethnically diverse patient population. In this study, patient data came from five major hospitals: the Mount Sinai Hospital located in East Harlem, Manhattan; Mount Sinai Morningside located in Morningside Heights, Manhattan; Mount Sinai West located in Midtown and the West Side, Manhattan; Mount Sinai Brooklyn located in Midwood, Brooklyn and Mount Sinai Queens located in Astoria, Queens. We included patients who were at least 18 years of age, had a laboratory-confirmed COVID-19 infection and were admitted to any of the aforementioned five MSHS hospitals between 27 February and 00:00 hours 2 April 2020 (time of data freeze). A confirmed case of COVID-19 was defined by a positive reverse transcriptase polymerase chain reaction (RT-PCR) assay of a specimen collected via nasopharyngeal swab.

Detection of viral RNA

Nasopharyngeal swab specimens were taken from all patients and placed in 120–140 µL viral transport media (VTM). RNA was purified from specimens using either the Qiacube Connect (Qiagen), QIAamp Viral RNA mini kit (Qiagen), or the EZ1 DSP Virus kit (Qiagen).

SARS-CoV-2 RNA was detected using the qualitative cobas SARS-CoV-2 kit using the cobas 6800 system. For detection, a two-target RT-PCR using (1) SARS-CoV-2-specific primers and (2) pan-sarbecovirus primers as included in the cobas master mix. All assays were performed in a CLIA-certified high complexity laboratory at the Mount Sinai Health System.

Data collection

The dataset was obtained from different sources and aggregated by the New York Covid Informatics Taskforce (NYCIT) (further description of NYCIT is provided in online supplemental material 1). We obtained demographics, diagnosis codes (International Classification of Diseases-9/10-Clinical Modification (ICD-9/10-CM)) codes and procedures, as well as vital signs and laboratory measurements during hospitalisation. Demographics included age, sex and language, as well as race and ethnicity in the electronic health records (EHRs). Racial groups included white, black or African-American, Asian, Pacific Islander, other and unknown. Ethnic groups included non-Hispanic/Latino, Hispanic/Latino or unknown. All vital signs and laboratory values were obtained as part of indicated clinical care.

Definitions of pre-existing conditions

We defined a pre-existing condition as the presence of diagnosis codes associated with specific diseases. Diagnoses and corresponding ICD codes are provided in online supplemental table 1.

Definitions of outcomes

We assessed in-hospital mortality and admission to intensive care.

Statistical analysis

Results are reported as medians and IQRs or means and SD, as appropriate. Categorical variables were summarised as counts and percentages. Statistical significance was evaluated using a Wilcoxon test for continuous variables and a Fisher's exact test for categorical variables. We visualised length of stay (LOS) using a cumulative incidence function with competing risks for mutually exclusive events of in-hospital mortality or discharge. Patients who were still hospitalised at the time of data freeze were regarded as having a censored LOS. We assumed censored observations from patients with ongoing hospitalisation will not exceed the longest LOS in our dataset when calculating the restricted mean LOS. No imputation was made for missing data. Analysis was performed with R.¹⁷

Data availability

Please contact authors for information on data availability.

RESULTS

A consort diagram of included patients and outcomes is depicted in online supplemental figure 1.

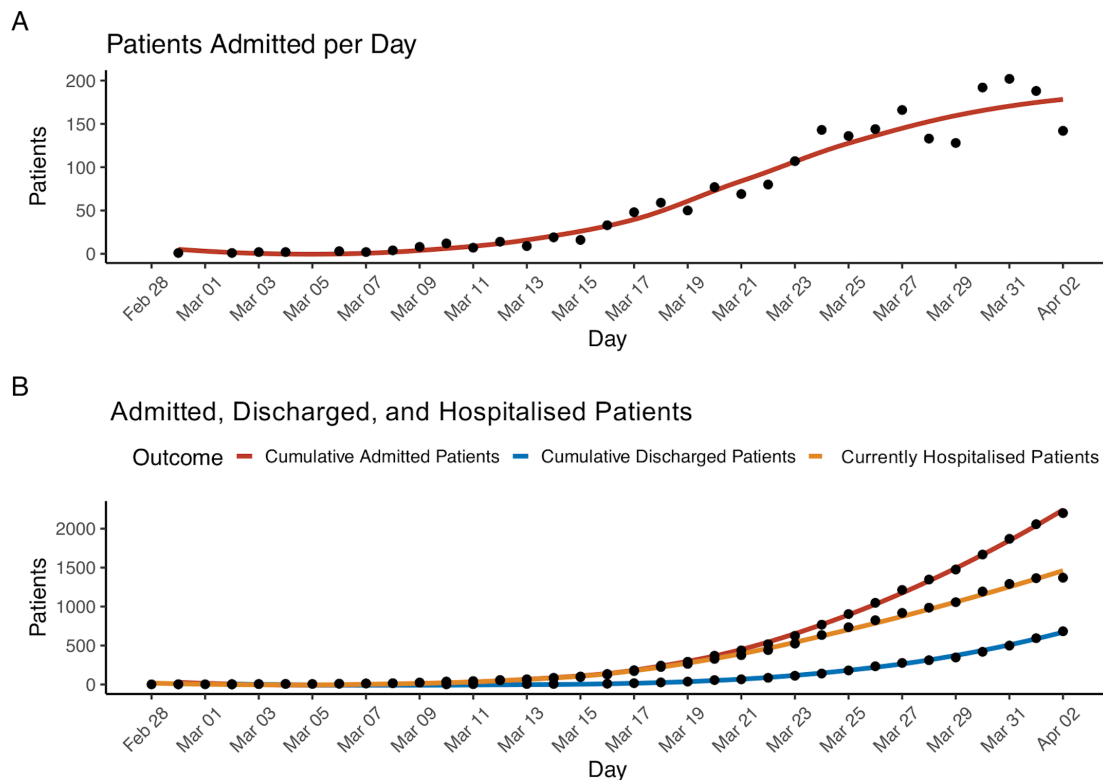


Figure 1 Hospital admissions of patients with COVID-19 within our cohort. Panel A: the number of total patients (n=2199) admitted each day to one of the hospitals for the duration of the study period. Panel B: the number of patients cumulatively admitted, cumulatively discharged or still hospitalised by day.

Demographic and clinical characteristics

From February 27 to 2 April 2020, 2199 patients with COVID-19 were hospitalised at one of five MSHS New York City hospitals. At the time of writing this report, 1121 (51%) patients remained hospitalised and 1078 (49%) completed their hospital course, with 768 discharges and 310 deaths. **Figure 1** details the number of patients admitted to the hospital per day and the total number of patients admitted cumulatively over time. During the study period, the trend of hospital admissions per day consistently increased.

Patient demographics, pre-existing conditions as well as vital signs and laboratory values at the time of admission are displayed in **table 1**. Median age was 65 years with only 3% of patients <30 years and 36% over 70 years. The proportion of men was higher (59%) than women (41%) and 25% had their race identified as white, 25% as African-American and 3% as Asian. One-quarter of the population has their ethnicity identified as Hispanic/Latino. More than half of the population had at least one pre-existing condition. Specifically, 37% presented with a history of hypertension, 27% with diabetes mellitus, 16% with coronary artery disease, 10% with heart failure and 9% with chronic kidney disease.

Laboratory results and vital signs at presentation

Overall, 1558 (71%) patients were admitted through the emergency department. On hospital admission, 39% of all patients were tachycardic and 88% of all patients were

febrile (**table 1**). The median white blood cell count was 7K/ μ L and lymphocyte percentage was 13.8. The median serum creatinine was 1mg/dL. Select inflammatory markers performed in subsets of patients in accordance with clinical indication were markedly elevated on admission (**table 1**). Specifically, the median C reactive protein (CRP) was 110mg/L, lactate dehydrogenase (LDH) was 416U/L and ferritin was 714ng/L. Over one-quarter of patients (28%) had a procalcitonin level above 0.49ng/mL, and nearly half of patients (46%) had a procalcitonin level <0.15ng/mL. The median D-dimer was 1.31 μ g/mL; one-third of patients (33%) had a D-dimer >2 μ g/mL.

The frequencies of otherwise non-routine laboratory assessments ordered on day of admission increased over time and are shown in online supplemental figure 2. In contrast, haemoglobin, a routinely measured clinical lab value, was ordered at admission in the majority of patients without variation over the study period.

Clinical outcomes

Due to the unknown future clinical course of those patients hospitalised at the time of data freeze, below we present clinical characteristics of only those patients who had completed their hospital course. A total of 1078 COVID-19 confirmed hospitalised patients completed their hospital course (died or discharged alive) by the date of data freeze on 2 April 2020. Of these, 768 (71%) were discharged and 310 (29%) died in the hospital.

Table 1 Characteristics of hospitalised patients with COVID-19 at baseline (n=2199)

Characteristics of admission	N with characteristic available	
Admission source, n (%)		
Emergency department	1558 (71)	2199
Other	641 (29)	
Race, n (%)		
White	554 (25.2)	2199
Black or African-American	543 (24.7)	
Asian	74 (3.4)	
Pacific Islander	25 (1.1)	
Other	912 (41.5)	
Unknown	91 (4.1)	
Ethnicity, n (%)		
Hispanic/Latino	576 (26.2)	2199
Non-Hispanic/Latino	1305 (59.4)	
Unknown	318 (14.5)	
Age, median (IQR)		
	65 (54–76)	2199
Age groups, n (%)		
18–30	73 (3.3)	2199
31–40	179 (8.1)	
41–50	225 (10.2)	
51–60	395 (18.0)	
61–70	527 (24.0)	
71–80	444 (20.2)	
81–90	274 (12.5)	
91 or older	82 (3.7)	
Sex, n (%)		
Male	1293 (58.8)	2199
Female	906 (41.2)	
Body mass index in kg/m², median (IQR)		
	28 (6)	1316
Medical history, n (%)		
Atrial fibrillation	156 (7.1)	2199
Asthma	180 (8.2)	
Coronary artery disease	343 (15.6)	
Cancer	151 (6.9)	
Chronic kidney disease	207 (9.4)	
Chronic obstructive pulmonary disease	113 (5.1)	
Diabetes mellitus	583 (26.5)	
Heart failure	217 (9.9)	
Hypertension	812 (37)	
Stroke	153 (7)	
Vital signs at hospital admission		
Heart rate in beats per min, median (IQR)	95 (83–108)	2181
Number of patients >100 beats per min, n (%)	857 (39)	
Temperature in °F, median (IQR)	99 (98.2–100.4)	2180
Number of patients >100.4°F, n (%)	1926 (88)	
Respiratory rate in breaths/min, median (IQR)	20 (18–21)	

Continued

Table 1 Continued

Characteristics of admission	N with characteristic available	
Systolic blood pressure in mm Hg, median (IQR)	130 (116–145)	2176
Diastolic blood pressure in mm Hg, median (IQR)	74 (65–82)	
Oxygen saturation, median (IQR)	95 (92–97)	2176
Admission laboratory parameters, median (IQR)		
White blood cell count in K/ μ L, median (IQR)	7 (5.2–9.9)	2029
Number of patients >10, n (%)	498 (25)	2029
Number of patients <4, n (%)	224 (11)	2029
Lymphocyte count in K/ μ L, median (IQR)	0.9 (0.7–1.3)	1888
Lymphocyte percentage, median (IQR)	13.8 (8.5–20.5)	1972
Haemoglobin g/dL, median (IQR)	131 (116–144)	2031
Platelet count in K/ μ L, median (IQR)	195 (151–253)	2028
Prothrombin time in s, median (IQR)	13.9 (13.2–15)	937
Activated partial thromboplastin time in s, median (IQR)	32 (29–36.1)	926
Serum sodium in mEq/L, median (IQR)	137 (134–140)	2015
Serum potassium in mEq/L, median (IQR)	4.2 (3.9–4.6)	2006
Serum creatinine in mg/dL, median (IQR)	1 (0.8–1.5)	2018
Aspartate aminotransferase in U/L, median (IQR)	42 (30–70.5)	1815
Alanine aminotransferase in U/L, median (IQR)	32 (19–56)	1025
Serum albumin in g/dL, median (IQR)	3.2 (2.8–3.6)	1848
Venous lactate in mmol/L, median (IQR)	1.5 (1.1–2)	775
Number of patients >1.5, n (%)	392 (51)	775
Inflammatory markers		
C reactive protein in mg/L, median (IQR)	110 (57.8–196)	1354
Ferritin in ng/L, median (IQR)	714 (324–1730)	1324
Procalcitonin in ng/mL, median (IQR)	0.18 (0.08–0.57)	1241
Number of patients >0.49, n (%)	344 (28)	1241
Number of patients <0.15, n (%)	573 (46)	1241
Lactate dehydrogenase in U/L, median (IQR)	416 (314–545)	1290
Creatine kinase in U/L, median (IQR)	199 (97–565)	502
D-dimer in μ g/mL, median (IQR)	1.31 (0.74–2.44)	1079
Number of patients >2.0, n (%)	352 (33)	1079
Required mechanical ventilation, n (%)	446 (20)	2199

Baseline values are defined as first values on hospitalisation. All continuous characteristics are in median (IQR) unless specified otherwise and all categorical characteristics are in number (percentage). The percentage is calculated with the number of patients who had the characteristic available as the denominator. For further clarity, the number in which that characteristic was available for is provided separately in adjacent column.

Estimates for mortality and need for intensive care unit (ICU) admissions over time are displayed in [figure 2](#).

The median LOS was 7.7 days, accounting for censoring of patients with ongoing hospitalisation. By the mean LOS (10.5 days), 45% of patients had been discharged, 17% had died and 38% were still hospitalised ([figure 2](#)). Demographics and admission laboratory measurements for patients who completed their hospital course are displayed in [table 2](#), stratified by mortality. The median age was significantly greater in those who died as compared with those who were discharged (75 years vs

59 years; $p < 0.001$). Pre-existing conditions were present in 64% of those who died and 46% of those discharged. We observed a significantly greater prevalence of diabetes (34% vs 20%; $p = 0.004$), chronic obstructive pulmonary disease (9% vs 4%; $p = 0.002$), heart failure (21% vs 7%; $p < 0.001$), stroke (10% vs 5%; $p = 0.004$) and hypertension (45% vs 30%; $p < 0.001$) in those who died as compared with those who were discharged. Prevalence of other comorbidities are provided in [table 2](#).

We present key laboratory markers at the time of hospital admission in subsets of patients for whom they

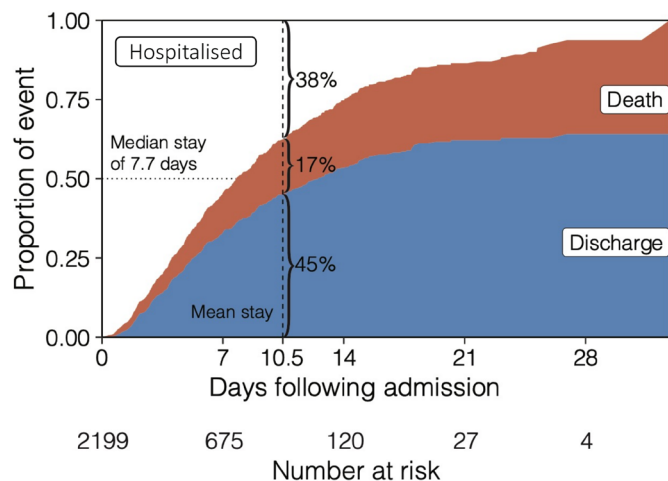


Figure 2 Cumulative incidence function displays the probability of mutually exclusive events of discharge (blue) or death (red) by a given day of hospitalisation, accounting for the changing number of patients at risk including censoring. The remaining portion (white) shows patients that are still hospitalised, where the median length of stay is 7.7 days.

were measured. Patients who died had a significantly lower median lymphocyte percentage (11.7% vs 16.6%; $p<0.001$) and greater aspartate aminotransferase (AST) (58.5 U/L vs 36 U/L; $p<0.001$), CRP (162 mg/L vs 79 mg/L; $p<0.001$), ferritin (798 ng/L vs 509 ng/L; $p<0.001$), LDH (517 U/L vs 347 U/L; $p<0.001$) and procalcitonin (0.44 ng/mL vs 0.09 ng/mL; $p<0.001$) as compared with those who were discharged (table 2). We also observed a significant elevation in D-dimer (56% $>2.0 \mu\text{g/mL}$ vs 21% $>2.0 \mu\text{g/mL}$; $p<0.001$) in those who died as compared with those who were discharged.

Patients requiring ICU admission

Of the 1078 patients who completed their hospital course, 385 (36%) required intensive care during their hospital stay. For these patients, vital signs and laboratory values immediately before transfer to intensive care are displayed in table 3, stratified by mortality outcome. Immediately before their ICU admission, patients who died were more likely to be tachycardiac (38% vs 19%; $p<0.001$) and hypotensive (22% vs 4%; $p<0.001$) as compared with those who were ultimately discharged. We also observed that patients who died had a lower median lymphocyte percentage (9.6% vs 16.6%) and greater serum creatinine (1.5 mg/dL vs 0.6 mg/dL; $p<0.001$), AST (62 U/L vs 35 U/L; $p<0.001$), CRP (220 mg/L vs 76 mg/L; $p<0.001$), ferritin (920 ng/L vs 503 ng/L; $p<0.001$), procalcitonin $>0.49 \text{ mg/mL}$ (59% vs 10%; $p<0.001$), LDH (513 U/L vs 333 U/L; $p<0.001$), CK (659 U/L vs 146 U/L; $p<0.001$) and D-dimer $>2 \mu\text{g/mL}$ (63% vs 22%; $p<0.001$) as compared with those who were discharged (table 3).

Outcomes by race

Including all individuals ($n=2199$), we then investigated survival probability stratified self-reported race and ethnicity by fitting a Cox proportional hazards model

adjusted for age and sex (see online supplemental figure 3). As compared with white individuals, we did not find a significant association of survival with self-reported black (HR=0.88; $p=0.37$), or other race (HR=1.12; $p=0.45$). We also did not find a significant association of survival with Hispanic ethnicity (HR=1.10; $p=0.50$).

DISCUSSION

The COVID-19 pandemic represents the greatest public health emergency in the modern world. Limited data, especially in the USA, exists to guide clinical care, resource management and risk stratification in hospitalised patients. Our study is of the case series of patients reported with confirmed COVID-19 in the USA. Previous reports were either from other countries, examined smaller cohorts, or were focused on critically ill patients.^{13 14 18–23} The present report provides a broad perspective on patients admitted with COVID-19 in both general medicine ward and intensive care settings. Our study presents key clinical differences from laboratory data available at time of admission and thus would aid clinical management decision-making early in the hospital course. Additionally, our health system serves a unique population representative of the ethnic and socio-economic diversity seen in both New York City and across the USA.

We highlight several key findings. Among the 1078 patients who completed their hospital course (discharge or in-hospital death), the overall mortality rate was 29% and 31% in patients who received ICU care. The overall case fatality rate likely represents an overestimation of the true disease mortality rate since patients who remained hospitalised at the date of data freeze were not included in this calculation. The mortality rate in intensive care is lower than previously described^{13 23–25} and may be reflective of early care escalation.

We observed that patients who died had a significantly higher median age with significantly more pre-existing conditions than those who were discharged. Although 25% of patients were febrile on admission, this may be an underestimation due to possible antipyretic use and/or selection bias. A substantial proportion of patients with COVID-19 displayed abnormal laboratory measurements at the time of admission. These included lymphopenia and elevated inflammatory markers such as D-dimer, CRP, LDH and ferritin. These trends persisted among those who died and/or received intensive care, both on admission and at the time of ICU transfer. If formal epidemiological analyses confirm these observations, early laboratory evaluation may be crucial in identifying patients suspected for COVID-19 prior to RT-PCR test result. It may also aid clinicians in identifying patients at high risk of decompensation, ICU admission and potentially even death. Early identification of high-risk patients could enable timely patient triage and improved resource allocation. Additional work is needed to develop real-time, accurate predictive models for risk stratification in

Table 2 Characteristics of hospitalised patients with COVID-19 by patients who had in-hospital mortality versus those who were discharged alive (n=1078)

	Patients with in-hospital mortality (n=310)	N with characteristic available	Patients who were discharged alive (n=768)	N with characteristic available	P value
Required mechanical ventilation, n (%)	165 (53)	310	14 (1.8)	768	<0.001
Time to ICU (hours), median (IQR)	11.0 (4.9–44.7)	121	12.6 (3.1–11.1)	264	<0.001
Race, n (%)					<0.001
White	98 (31.6)	310	216 (28.1)	768	
Black or African-American	71 (22.9)		185 (24.1)		
Asian	17 (5.5)		22 (2.9)		
Pacific Islander	1 (0.3)		7 (0.9)		
Other	111 (35.8)		310 (40.4)		
Unknown	12 (3.9)		28 (3.6)		
Ethnicity, n (%)					0.005
Hispanic/Latino	58 (18.7)	310	208 (27.1)	768	
Non-Hispanic/Latino	198 (63.9)		464 (60.4)		
Unknown	54 (17.4)		96 (12.5)		
Age, median (IQR)	75 (64–85)		59 (45–72)		<0.001
Age groups, n (%)					
18–30	1 (0.32)	310	53 (6.9)	768	
31–40	2 (0.65)		108 (14.1)		
41–50	17 (5.5)		100 (13.0)		
51–60	37 (11.9)		143 (18.6)		
61–70	66 (21.3)		157 (20.4)		
71–80	83 (26.8)		130 (16.9)		
80–90	75 (24.2)		62 (8.1)		
>90	29 (9.4)		15 (2.0)		
Sex, n (%)					0.16
Male	191 (61.6)	310	436 (56.8)	768	
Female	119 (38.4)		332 (43.2)		
Body mass index in kg/m ² , median (IQR)	32 (27–34)	241	28 (25–32)	627	<0.001
Previous medical history, n (%)					
Atrial fibrillation	43 (13.9)	768	42 (5.5)	768	0.91
Asthma	23 (7.4)		61 (7.9)		0.87
Coronary artery disease	83 (26.8)		84 (10.9)		<0.001
Cancer	24 (7.7)		40 (5.2)		0.147
Chronic kidney disease	41 (13.2)		54 (7)		0.002
Chronic obstructive pulmonary disease	28 (9)		31 (4)		0.002
Diabetes mellitus	105 (33.9)		151 (19.7)		<0.001
Heart failure	64 (20.6)		53 (6.9)		<0.001
Hypertension	140 (45.2)		233 (30.3)		<0.001
Stroke	32 (10.3)		40 (5.2)		0.004
Admission laboratory parameters					
White blood cell count in K/μL, median (IQR)	8.6 (5.9–12)	281	6.2 (4.7–8.2)	728	<0.001
Number of patients >10 K/μL, n (%)	105 (37)	281	101 (14)	728	<0.001
Number of patients <4 K/μL, n (%)	17 (6)	281	106 (15)	728	<0.001

Continued

Table 2 Continued

	Patients with in-hospital mortality (n=310)	N with characteristic available	Patients who were discharged alive (n=768)	N with characteristic available	P value
Haemoglobin in g/dL, median (IQR)	12.6 (10.9–14.3)	282	13.2 (11.9–14.4)	728	<0.001
Platelet count in K/ μ L, median (IQR)	185 (144–239)	282	193 (153–247)	726	0.070
Lymphocyte count K/ μ L, median (IQR)	0.9 (0.6–1.3)	248	1 (0.8–1.4)	689	<0.001
Lymphocyte percentage, median (IQR)	11.7 (6.6–18.6)	270	16.6 (11.7–24.2)	710	<0.001
Prothrombin time in s, median (IQR)	14.2 (13.6–16.3)	142	13.5 (13–14.4)	304	<0.001
Activated partial thromboplastin time in s, median (IQR)	32.9 (30.2–37.6)	140	31.4 (28.7–34.8)	302	0.001
Serum sodium in mEq/L, median (IQR)	138 (134–141)	284	137 (135–140)	708	0.059
Serum potassium in mEq/L, median (IQR)	4.4 (4–5)	288	4.1 (3.8–4.5)	706	<0.001
Serum creatinine in mg/dL, median (IQR)	1.3 (0.9–2.2)	288	0.9 (0.7–1.2)	710	<0.001
Aspartate aminotransferase in U/L, median (IQR)	58.5 (34–102)	246	36 (26–54)	628	<0.001
Alanine aminotransferase in U/L, median (IQR)	32 (19–58)	119	30 (19–52)	358	0.47
Serum albumin in g/dL, median (IQR)	3 (2.7–3.4)	255	3.4 (3–3.7)	635	<0.001
Venous lactate in mmol/L, median (IQR)	1.8 (1.3–2.6)	108	1.3 (1.1–1.8)	236	<0.001
Number of patients >1.5 mmol/L, n (%)	74 (69)	108	88 (37)	236	<0.001
Inflammatory markers					
C reactive protein in mg/L, median (IQR)	162 (75.7–266)	141	79.3 (37.5–144)	422	<0.001
Ferritin in ng/L, median (IQR)	798 (397–2020)	146	509 (235–987)	410	<0.001
Procalcitonin -in ng/mL, median (IQR)	0.44 (0.16–1.45)	138	0.09 (0.05–0.22)	392	<0.001
Number of patients >0.49 ng/mL, n (%)	67 (49)	138	46 (12)	392	<0.001
Number of patients <0.15 ng/mL, n (%)	34 (25)	138	263 (67)	392	<0.001
Lactate dehydrogenase in U/L, median (IQR)	517 (371–756)	134	347 (272–448)	419	<0.001
Creatine kinase in U/L, median (IQR)	472 (139–940)	74	154 (84–319)	126	<0.001
D-dimer in μ g/mL, median (IQR)	2.41 (1.18–3.79)	117	0.93 (0.58–1.61)	282	<0.001
Number of patients >2.0 μ g/mL, n (%)	66 (56)	117	59 (21)	282	<0.001

Continuous and categorical variables were compared using a Wilcoxon test and Fisher's exact test, respectively.

Baseline values are defined as first values on hospitalisation. All continuous characteristics are in median (IQR) unless specified otherwise and all categorical characteristics are in number (percentage). The percentage is calculated with the number of patients who had the characteristic available as the denominator. For further clarity, the number in which that characteristic was available for is provided separately in adjacent column.

ICU, intensive care unit.

COVID-19, particularly to elucidate the clinical utility of specific laboratory measurements.

Han *et al*²⁶ found a significant association of serum LDH and CRP with COVID-19 severity in patients from China.²⁶ As the number of cases rose quickly in New York City, MSHS hospitals served as early adopters, creating a COVID-19 order set in our EHR to streamline objective data gathering, facilitate more cohesive workflow among team members and minimise ancillary staff exposure by completing all necessary admission labs at one time. This laboratory order set included serum D-dimer, CRP, procalcitonin, ferritin and LDH. In turn, we observed an increase in these orders from the first day of admission

over the study period (see online supplemental figure 2). Given the abnormalities observed in patients who died, these laboratory measurements may be prognostic markers of disease severity or subsequent clinical course, although this requires further investigation. If confirmed, other health systems expecting impending case surges may consider similar workflows to promote improved healthcare delivery to affected patients.

We found a significantly higher procalcitonin level in individuals who died as compared with those who were discharged alive. Moreover, of the 2199 individuals in the study, 28% had a procalcitonin >0.49. In the context of COVID-19, an elevated procalcitonin may signify a

Table 3 Selected characteristics for hospitalised patients with COVID-19 before transfer to intensive care stratified by in-hospital mortality (n=385)

	Patients with in-hospital mortality who required intensive care (n=121)	N with characteristic available	Patients who were discharged alive but required intensive care (n=264)	N with characteristic available	P value
Required mechanical ventilation, n (%)	95 (79)	121	11 (4)	264	<0.001
Vital signs, n (%)					
Number of patients with heart rate >100 beats per min, n (%)	45 (38)	118	51 (19)	264	<0.001
Number of patients with temperature >100.4°F, n (%)	107 (91)		228 (86)		0.370
Systolic blood pressure <100 mm Hg, n (%)	27 (22)		10 (4)		<0.001
Oxygen saturation, median (IQR)	92 (88–95)		95 (92–97)		<0.001
Admission laboratory parameters, median (IQR)					
White blood cell count in K/μL, median (IQR)	10.1 (6.3–14.7)	111	5.6 (4.3–7.7)	258	<0.001
Number of patients >10 K/μL, n (%)	58 (52)	111	28 (11)	258	<0.001
Number of patients <4 K/μL, n (%)	6 (5)	111	54 (21)	258	<0.001
Haemoglobin in g/dL, median (IQR)	12.1 (10.4–13.8)	111	13 (11.5–14.2)	258	<0.001
Platelet count in K/μL, median (IQR)	185 (146–230)	111	191(153–248)	258	0.308
Lymphocyte count in K/μL, median (IQR)	0.8 (0.6–1.25)	91	1 (0.75–1.4)	239	0.003
Lymphocyte percentage, median (IQR)	9.6 (5.5–17.2)	103	16.6 (12–26.4)	251	<0.001
Prothrombin time in s, median (IQR)	14.9 (13.8–16.7)	63	13.7 (13.2–14.7)	97	<0.001
Activated partial thromboplastin time in s, median (IQR)	34 (30.6–38.2)	62	31.4 (29.4–35)	96	0.027
Serum sodium in mEq/L, median (IQR)	137 (135–140)	114	137 (135–140)	237	0.449
Serum potassium in mEq/L, median (IQR)	4.3 (4–4.9)	112	4.1 (3.8–4.4)	237	<0.001
Serum creatinine in mg/dL, median (IQR)	1.5 (0.94–2.4)	114	0.8 (0.7–1.1)	261	<0.001
Aspartate aminotransferase in U/L, median (IQR)	62 (36.2–105)	106	35 (26–54.5)	236	<0.001
Alanine aminotransferase in U/L, median (IQR)	28 (16–54)	56	28 (19–52)	215	0.514
Serum albumin in g/dL, median (IQR)	2.7 (2.3–3.2)	107	3.3 (2.9–3.6)	236	<0.001
Venous lactate in mmol/L, median (IQR)	1.55 (1.02–2.15)	34	1.3 (1.1–1.7)	49	0.157
Number of patients >1.5 mmol/L, n (%)	19 (56)	34	20 (41)	49	0.157
Inflammatory markers					
C reactive protein in mg/L, median (IQR)	220 (113–297)	80	75.6 (36.2–134)	182	<0.001
Ferritin in ng/L, median (IQR)	920 (470–2220)	76	503 (266–1020)	187	<0.001
Procalcitonin in ng/mL, median (IQR)	1.02 (0.20–4.5)	58	0.08 (0.05–0.22)	163	<0.001
Number of patients >0.49 ng/mL, n (%)	34 (59)	58	17 (10)	163	<0.001
Number of patients <0.15 ng/mL, n (%)	11 (19)	58	106 (65)	163	<0.001
Lactate dehydrogenase in U/L, median (IQR)	513 (399–729)	67	333 (274–422)	188	<0.001
Creatine kinase in U/L, median (IQR)	659 (305–1450)	42	146 (60–268)	73	<0.001
D-dimer in μg/mL, median (IQR)	2.7 (1.6–5.8)	57	0.83 (0.5–1.6)	127	<0.001
Number of patients >2.0 μg/mL, n (%)	36 (63)	57	28 (22)	127	<0.001

Continued

Table 3 Continued

	Patients with in-hospital mortality who required intensive care (n=121)	N with characteristic available	Patients who were discharged alive but required intensive care (n=264)	N with characteristic available	P value
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Continuous and categorical variables were compared using a Wilcoxon test and Fisher's exact test, respectively.

All values are closest values in 24 hours prior to ICU admission. All continuous characteristics are in median (IQR) unless specified otherwise and all categorical characteristics are in number (percentage). The percentage is calculated with the number of patients who had the characteristic available as the denominator. For further clarity, the number in which that characteristic was available for is provided separately in adjacent column.

ICU, intensive care unit.

superimposed bacterial infection, but may also be a marker of acute respiratory distress syndrome or a result of upregulated cytokine production secondary to respiratory failure. Additionally, we found that individuals who died in the ICU had higher procalcitonin levels, supporting previous work demonstrating that patients with more severe COVID-19 infections have higher procalcitonin levels.^{22 27}

The clinical characteristics of our cohort were largely similar to other large cohorts of patients with COVID-19 from China^{2 22} and Italy. Specifically, patients in our dataset were elderly and had a male predominance, similar to previous reports. There was also a high prevalence of comorbid conditions, including hypertension and diabetes mellitus. Similar to an early report from Wuhan, patients largely had a normal procalcitonin at admission, but individual requiring ICU care had a higher procalcitonin level. However, as compared studies from Wuhan, China²² and Genoa, Italy,¹⁴ and New York City,²⁸ our cohort had a significantly lower prevalence of lymphopenia at admission.

Our study should be considered in light of several limitations. Since COVID-19 testing is frequently repeated in hospitalised patients and initial testing may result in false negatives, we are unable to determine whether patients developed their infection during or before hospital admission. Furthermore, COVID-19 has a variable incubation period of approximately 8–15 days,²⁹ and patients may present to the hospital several days after initial infection or the onset of symptoms. Thus, we are unable to determine patients' disease duration. Additionally, we separated discharged patients from those who died, but some patients may have expired after discharge. This could affect our listed case mortality rate. Our study is also confined by the inherent limitations (eg, biases) of EHR data. Although using structured EHR data allows for rapid integration of multiple data streams and real-time analysis, data present only in clinical note text, such as symptoms on presentation are missed. Additionally, symptoms present before the time of admission were not included. We chose not to perform comprehensive manual chart review to prioritise timely dissemination of our observations. Another limitation of our dataset is the large proportion of individuals that were censored.

These individuals remained in the hospital at the time of data analysis and thus had unknown outcomes. Future work will analyse these patients' hospital course in greater detail with complete outcomes data.

As the COVID-19 pandemic spreads from the current epicentre in New York City to other areas, our report provides meaningful clinical insights that may better inform care for diverse populations. Future work will aim to predict COVID-19 patient outcomes using a variety of approaches, thereby reducing healthcare system burden and permitting improved care delivery.

Author affiliations

¹The Hasso Plattner Institute for Digital Health at Mount Sinai, New York, New York, USA

²Department of Internal Medicine, Icahn School of Medicine at Mount Sinai, New York, New York, USA

³Department of Genetics and Genomic Sciences, Icahn School of Medicine at Mount Sinai, New York, New York, USA

⁴The Zena and Michael A. Wiener Cardiovascular Institute, New York, New York, USA

⁵Department of Population Health Science and Policy, Icahn School of Medicine at Mount Sinai, New York, New York, USA

⁶The Pamela Sklar Division of Psychiatric Genomics, Icahn School of Medicine at Mount Sinai, New York, New York, USA

⁷Institute for Healthcare Delivery Science, Icahn School of Medicine at Mount Sinai, New York, New York, USA

⁸Icahn Institute for Data Science and Genomic Technology, Icahn School of Medicine at Mount Sinai, New York, New York, USA

⁹Department of Pathology, Molecular and Cell-Based Medicine, Icahn School of Medicine at Mount Sinai, New York, New York, USA

¹⁰Mount Sinai Data Warehouse, Mount Sinai Health System, New York, New York, USA

¹¹Division of Infectious Diseases, Icahn School of Medicine at Mount Sinai, New York, New York, USA

¹²Icahn School of Medicine at Mount Sinai Department of Medicine, New York, New York, USA

¹³Icahn School of Medicine at Mount Sinai BioMedical Engineering and Imaging Institute, New York, New York, USA

¹⁴Department of Radiology, Icahn School of Medicine at Mount Sinai, New York, New York, USA

¹⁵Department of Cardiology, Icahn School of Medicine at Mount Sinai, New York, New York, USA

¹⁶Icahn School of Medicine at Mount Sinai Friedman Brain Institute, New York, New York, USA

¹⁷The Nash Family Department of Neuroscience, Icahn School of Medicine at Mount Sinai, New York, New York, USA

¹⁸Department of Medicine, Division of Cardiology, Zena and Michael A. Wiener Cardiovascular Institute and Marie-Josée and Henry R. Kravis Center for Cardiovascular Health, Icahn School of Medicine at Mount Sinai, New York, New York, USA

¹⁹Icahn School of Medicine at Mount Sinai Department of Psychiatry, New York, New York, USA

²⁰The Office of the Dean, Icahn School of Medicine at Mount Sinai, New York, New York, USA

²¹Icahn School of Medicine at Mount Sinai Department of Anesthesiology Perioperative and Pain Medicine, New York, New York, USA

²²Icahn School of Medicine at Mount Sinai Department of Environmental Medicine and Public Health, New York, New York, USA

²³Institute for Exposomic Research, Icahn School of Medicine at Mount Sinai, New York, New York, USA

²⁴The Charles Bronfman Institute for Personalized Medicine, Icahn School of Medicine at Mount Sinai, New York, New York, USA

Twitter Ishan Paranjpe @ParanjpeIshan, Robert M Freeman @RFreeman_RN and Benjamin S Glicksberg @BenGlicksberg

Contributors IP, AJR, JKDF, AL, MS, AV, KWJ, SS, AK, RO'H, SM, UN, SKJ, AK, PT, JJ and AF developed and performed the analysis. PG, MAL, JF, JAA, EB, CRH and BM critically appraised the manuscript and helped acquire data. IP, AL, AJR, JKDF, RM, MD, EG, DM, LMH, RF, MS, PK, VF, EPB, JN, EJJ, CC-C, DC and DLR supervised the project. IP, AL, AJR, JKDF, GNN, BSG, AWC and AJ drafted the manuscript.

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ORCID iDs

Ishan Paranjpe <http://orcid.org/0000-0001-5144-0370>

Suraj K Jaladanki <http://orcid.org/0000-0003-0406-2973>

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