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Hospital bed occupancy rate is an independent risk factor for COVID-19 inpatient mortality: a pandemic epicentre cohort study

Francesco Castagna ,[1,2] Xiaonan Xue,[3] Omar Saeed,[1,2] Rachna Kataria,[4] Yoram A Puius,[1,5] Snehal R Patel,[1,2] Mario J Garcia,[1,2] Andrew D Racine,[1,6] Daniel B Sims,[1,2] Ulrich P Jorde[1,2]

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For numbered affiliations see end of article.

Correspondence to
Dr Ulrich P Jorde;
ujorde@montefiore.org

ABSTRACT

Introduction COVID-19 first struck New York City in the spring of 2020, resulting in an unprecedented strain on our healthcare system and triggering multiple changes in public health policy governing hospital operations as well as therapeutic approaches to COVID-19. We examined inpatient mortality at our centre throughout the course of the pandemic.

Methods This is a retrospective chart review of clinical characteristics, treatments and outcome data of all patients admitted with COVID-19 from 1 March 2020 to 28 February 2021. Patients were grouped into 3-month quartiles. Hospital strain was assessed as per cent of occupied beds based on a normal bed capacity of 1491.

Results Inpatient mortality decreased from 25.0% in spring to 10.8% over the course of the year. During this time, use of remdesivir, steroids and anticoagulants increased; use of hydroxychloroquine and other antibiotics decreased. Daily bed occupancy ranged from 62% to 118%. In a multivariate model with all year's data controlling for demographics, comorbidities and acuity of illness, percentage of bed occupancy was associated with increased 30-day in-hospital mortality of patients with COVID-19 (0.7% mortality increase for each 1% increase in bed occupancy; HR 1.007, CI 1.001 to 1.013, p=0.004)

Conclusion Inpatient mortality from COVID-19 was associated with bed occupancy. Early reduction in epicentre hospital bed occupancy to accommodate acutely ill and resource-intensive patients should be a critical component in the strategic planning for future pandemics.

Strengths and limitations of this study

- This is a large cohort study with 7390 patients with COVID-19.
- This is a longitudinal analysis over 1 year of management and hospital policy changes.
- The study analyses mortality changes after adjustment for different therapies and clinical parameters.
- The study identified the association between level of hospital system stress and mortality, with important public health ramifications.
- Data on most recent variants were not included.

INTRODUCTION

COVID-19 was declared a global pandemic by the WHO on 11 March 2020.[1] In the USA, after a cluster of cases reported from Washington state,[2] New York State quickly became the initial epicentre of this pandemic, with over 1.27 million of cases to date and over 50 000 fatalities, with the highest concentration in the Bronx and Queens boroughs of New York City.[3] Montefiore Einstein, with its three principal teaching hospitals and combined adult bed capacity of 1491, is the primary healthcare provider for the large, nearly 1.5 million diverse population of the Bronx[4] and experienced a 'first wave' of COVID-19 admissions in the spring of 2020,[3] followed by a significant reduction of cases until a second surge in hospitalisations was noted in the winter of 2020. Throughout the course of the year, multiple public health measures, including those adapting hospital operation to a disaster-level pandemic, such as cancellation of all elective procedures and waiver of state-specific licensing for healthcare providers, were put in place. In addition, the understanding of COVID-19 pathophysiology improved,[5 6] new treatments were developed,[7–10] parts of the general population[11 12] as well as hospital personnel developed antibodies after COVID-19 illness,[13] and our hospital system adapted to and then recovered from crisis mode.[14] Here, we report the outcomes of patients hospitalised with COVID-19 through 1 year since the first case, focusing on the differences observed between the spring and the winter surges.

METHODS

Study population

We retrospectively reviewed all adult patients admitted to Montefiore Medical Center with

a real-time reverse transcription-PCR assay positive for COVID-19 between 1 March 2020 and 28 February 2021. We divided this timeframe into four 3-month seasons based on the northern hemisphere calendar: spring (from 1 March 2020 to 31 May 2020), summer (from 1 June 2020 to 30 August 2020), fall (from 1 September 2020 to 30 November 2020) and winter (1 December 2020 to 28 February 2021).

Data collection

Medical data including demographic, clinical and laboratory variables were extracted from the electronic medical record system. The primary outcome was 30-day in-hospital mortality.

Statistical analysis

Continuous variables are displayed as mean±SD or median (25%–75% IQR) and compared with the Student's t-test or Wilcoxon rank-sum, as appropriate. Categorical data are presented as per cent and compared by χ^2 test. We estimated the cumulative incidence of the primary endpoint in-hospital mortality for each season, treating hospital discharge as a competing event.[15] To avoid any bias due to differential follow-up length, we censored the follow-up time at 30 days after admission.

A multivariable competing risk proportional hazard model was used to estimate the subdistribution HR[16 17] for time to in-hospital death. The covariates in the multivariable analyses included factors present in >90% of our data set, known to be associated with in-hospital COVID-19 mortality based on prior literature,[6 18 19] or with a univariate association with in-hospital mortality (p<0.05) and a clinical (relative difference >5%) difference between survivors and non-survivors (online supplemental table 1). These variables included age, sex, body mass index (BMI), vital signs at presentation (temperature, systolic and diastolic blood pressure, heart rate, respiratory rate, pulse oxygen saturation), platelet count, white cell count, potassium, bicarbonate, creatinine, glucose, alanine transaminase, aspartate transaminase, history of hypertension, dyslipidaemia, chronic kidney disease, heart failure, coronary artery disease, asthma/chronic obstructive pulmonary disease, diabetes mellitus and statin use. Additionally, lactic acid level and per cent of hospital bed saturation were forced into the model as markers of illness severity and level of hospital stress, respectively.

Then we focused on examining the difference in in-hospital death between patients admitted in the spring and in the winter, as they represented the two largest and most temporal distant waves of the COVID-19 pandemic occurring before and after public health policies, specific therapeutic approaches and hospital management changes had been implemented. The selection method for covariates is presented in online supplemental table 2.

The proportionality assumption was examined[20] and no violation was identified. A two-sided p<0.05 was considered statistically significant.

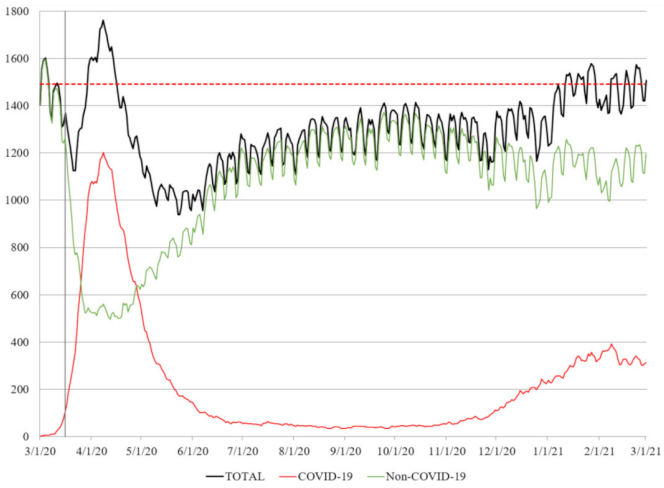

Figure 1 Simultaneously admitted patients. This graph includes hospitalised and admitted patients in the emergency department waiting for a bed. A precipitous decline of non-COVID-19 admissions began on 16 March 2020 (vertical grey line) coinciding with gubernatorial healthcare-associated directives in New York State. The dotted red line indicates the nominal bed capacity of our institution (1491 beds).

Propensity score analysis

To fully control the potential differences in patient population and hospital stress between spring and winter patients with COVID-19, we also used propensity score (PS) matching to compare the 30-day in-hospital mortality between spring and winter admissions. The same covariates used for the multivariable competing risk regression were used for PS matching. PS matching was carried out through a 1:1 greedy matching algorithm, with a calliper width of 0.1 SD. We then stratified on matched pair in the competing risk regression model.[21 22] Because one-to-one matching led to a reduction in sample size, we used this analysis as a sensitivity analysis.

All statistical analyses was performed with SPSS V.25 and the R packages cmprsk and crrSC (V.3.5; R Foundation for Statistical Computing).

Patient and public involvement

Given the retrospective nature of our analysis, it was not appropriate or possible to involve patients or the public in the design, or conduct, or reporting or dissemination plans of our research.

RESULTS

There were 7390 COVID-19-positive adult patients admitted between 1 March 2020 and 28 February 2021 (figure 1). Of these, 4495 patients were admitted during the spring, 264 during the summer, 377 during the fall and 2254 during the winter.

On 8 April 2020, the peak of the spring season, the total number of simultaneously adult patients admitted to our hospital (including those admitted to emergency adult wards at our children's hospital[23]) was 1762 (118% of nominal bed capacity); 1201 of them (68.2%)

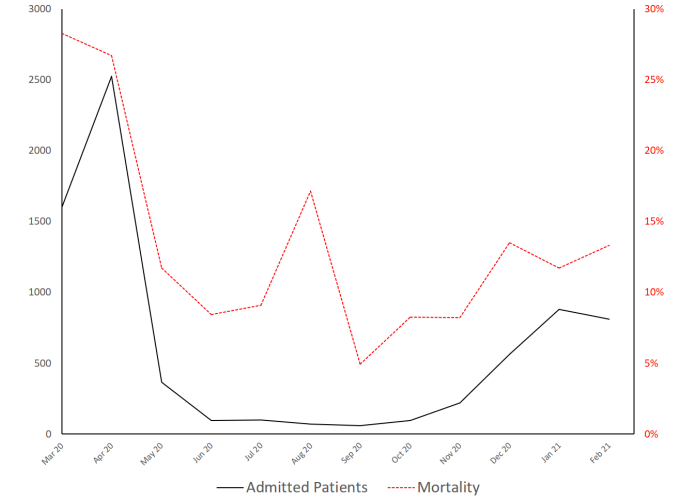

Figure 2 Cumulative monthly admissions (black line, left axis) and mortality (dotted red line, right axis) over the year.

were patients with COVID-19. On 8 February 2021, the peak of winter season, 1512 patients (101% of nominal bed capacity) were admitted to our hospital and 393 of them (26.0%) were patients with COVID-19 (figure 1). Following cancellation of elective procedures, bed occupancy decreased to 70% by the end of the spring season and remained at 90% until the beginning of the winter season, when the second wave occurred in December 2020. Unadjusted mortality of patients admitted at the beginning of spring, end of spring, beginning of winter and end of winter was 28%, 8%, 14% and 13%, respectively (figure 2).

Patient population

Demographics, medical history and vital signs at arrivals are presented in table 1. Initial laboratory blood tests are presented in online supplemental table 3. Overall, the median age was 66 (55–77) years, 3835 (51.9%) patients were male and 5519 (74.2%) were of black race and/or

Table 1 Demographics, medical history and vital signs of admitted patients

	Spring (n=4495)	Summer (n=264)	Fall (n=377)	Winter (n=2254)
30-day hospital outcome, n (%)				
Still admitted	194 (4.3)	6 (2.3)	15 (4.0)	103 (4.6)
Discharged alive	3177 (70.7)	229 (86.7)	336 (89.1)	1893 (84.0)
Died in the hospital	1124 (25.0)	29 (11.0)	26 (6.9)	258 (11.4)
Demographics				
Age (IQR), years	66 (55–77)	66 (50–76)	63 (50–73)	67 (56–77)
Male sex, n (%)	2377 (52.9)	138 (52.3)	198 (52.5)	1122 (49.8)
Black race and/or Hispanic ethnicity, n (%)	3345 (74.4)	219 (83.0)	286 (75.9)	1635 (74.2)
Body mass index (IQR), kg/m^2	28.4 (24.6–33)	27.6 (22.5–32.7)	28.6 (25–34.1)	28.2 (24.4–33.1)
Hospital bed saturation, % (IQR)	97.4 (86.5–107.6)	81.7 (76.3–85.8)	87.6 (83.2–90.2)	95.3 (91.9–101.8)
Medical history, n (%)				
Hypertension	3370 (75)	197 (74.6)	254 (67.4)	1713 (76)
Sleep apnoea	521 (11.6)	28 (10.6)	47 (12.5)	270 (12)
Hyperlipidaemia	2609 (58)	153 (58)	199 (52.8)	1380 (61.2)
Atrial fibrillation	449 (10)	30 (11.4)	35 (9.3)	267 (11.8)
Chronic kidney disease	1406 (31.3)	70 (26.5)	85 (22.5)	620 (27.5)
Heart failure	980 (21.8)	72 (27.3)	66 (17.5)	519 (23)
Coronary artery disease	1316 (29.3)	95 (36)	108 (28.6)	721 (32)
Asthma/COPD	1371 (30.5)	84 (31.8)	98 (26)	753 (33.4)
Diabetes mellitus	2522 (56.1)	148 (56.1)	187 (49.6)	1244 (55.2)
Vitals at presentation				
Temperature (IQR), °C	37.2 (36.8–37.8)	36.9 (36.6–37.2)	37.1 (36.7–37.7)	37.1 (36.7–37.7)
SBP (IQR), mm Hg	131 (114–148)	132 (117–149)	131 (117–147)	132 (117–148)
DBP (IQR), mm Hg	75 (65–84)	77 (67–87)	74 (68–84)	75 (67–84)
HR (IQR), beats per minute	98 (85–112)	92.5 (76.3–105)	94 (80–107)	95 (82–107)
Oxygen saturation (IQR), %	95 (91–98)	98 (96–99)	96 (94–98)	96 (92–98)
Respiratory rate (IQR), breaths per minute	20 (18–22)	18 (17–20)	18 (18–20)	19 (18–22)

COPD, chronic obstructive pulmonary disease; DBP, diastolic blood pressure; HR, heart rate; SBP, systolic blood pressure.

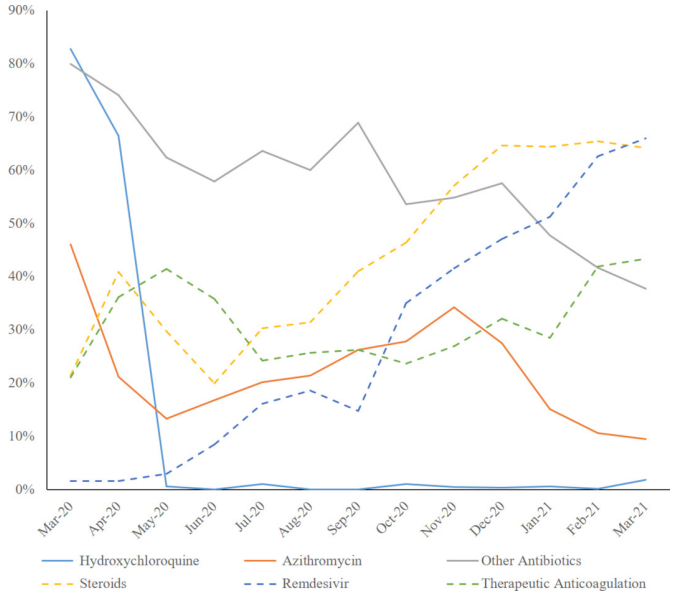

Figure 3 Change in therapies: per cent of patients receiving specific therapies over the year.

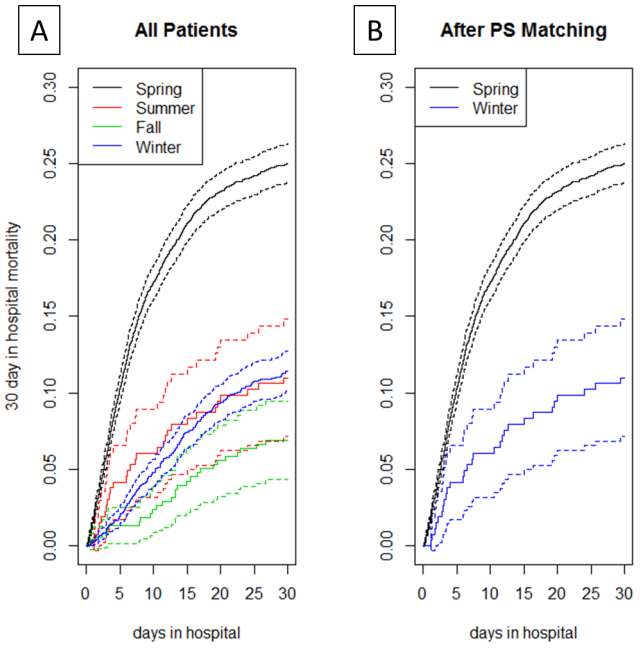

Figure 4 Cumulative incidence: 30-day in-hospital mortality (A) by season and (B) spring vs winter after propensity score matching. PS, propensity score.

Hispanic ethnicity. The median age ranged from 63 years in fall to 67 years in winter. Sex distribution was similar throughout the year. Summer and fall patients had the lowest and the highest BMI: 26.7 kg/m^2 and 28.6 kg/m^2, respectively.

Pharmacotherapy

Changes in pharmacological approach are presented in online supplemental table 4 and figure 3.

Spring patients were more likely to receive hydroxychloroquine, azithromycin and other antibiotics. The use of remdesivir substantially increased throughout the year (from less than 2% during spring to almost 70% by the end of the winter). Steroids prescription (from 33% during spring to almost 70% in February 2021), therapeutic anticoagulation therapy, as well as use of statins, ACE inhibitors (ACE-I) or angiotensin receptor blockers (ARBs) also increased.

Death, intubation and length of stay

Over the course of a year, 1437 (19.4%) died while hospitalised. Patients who died were older, had more comorbidities and were more acutely ill, consistent within prior reports on risk factors for death in COVID-19[5 6] (online supplemental table 1). The average unadjusted monthly mortality is presented in figure 2. The 30-day in-hospital mortality (figure 4A) was 25.0% for spring patients, 11.0% for summer patients, 6.9% for fall patients and 11.4% for winter patients (p<0.001). On average, spring patients died 6.4 (3.2–12.9) days after arrival to the emergency department, summer patients 7.2 (3.0–15.7) days after arrival, fall patients 13.4 (8.7–21.6) days after arrival and winter patients 13.3 (6.8–20.7) days after arrival (p<0.001). The frequency of invasive ventilatory support was higher during the spring, with 892 patients (19.4%) intubated, vs 27 (10.2%) in summer, 36 (9.5%) in fall and 268 (11.9%) in winter (p<0.001). The median time from arrival to intubation was 0.7 (0.1–4.1) days for spring patients, 0.6 (0.1–8.1) days for summer patients, 2.2 (0.1–7.3) days for fall patients and 2.8 (0.3–7.0) days for winter patients (p<0.001). The median length of stay was 6.1 (3.5–11.1) days during spring, 5.1 (2.7–10.1) days during summer, 5.0 (3.0–10.1) days during fall and 6.3 (3.8–12.0) days during winter (p<0.001).

Bed saturation and mortality

We defined bed saturation as the percentage of bed occupancy calculated from the ratio between the number of admitted patients over the nominal bed capacity of our institution (1491).

In the multivariable competing risk proportional hazard model of the entire cohort, per cent of bed occupancy was associated with increased 30-day in-hospital mortality (HR 1.007, CI 1.001 to 1.013, p=0.004); that is, mortality increases by 0.7 % for each 1% increase in bed occupancy. Consistent results were observed per level increase in bed occupancy quartile (HR 1.086, CI 1.026 to 1.148, p value for linear trend=0.004). The results of the competing risk regression analysis are presented in table 2.

Spring versus winter mortality comparison and propensity matched analysis

In the multivariable competing risk proportional hazard model comparing spring and winter season, the 30-day in-hospital mortality was lower in winter (HR 0.520, CI 0.448 to 0.604, p<0.001) when compared with spring. After PS calliper matching, there were 1722 matched pairs. Spring and winter patients had similar distribution of PS (online supplemental figure 1) and the standardised average difference among covariates was greatly

Table 2 Association with in-hospital mortality (regression models with competing risks)

	Multivariable	
Variable	HR (95% CI)	P value
Age, years	1.046 (1.04 to 1.051)	<0.001
Male sex, yes/no	1.352 (1.187 to 1.54)	<0.001
Body mass index, kg/m^2	1.022 (1.012 to 1.032)	<0.001
Temperature, °C	1.129 (1.063 to 1.200)	<0.001
SBP, mm Hg	0.994 (0.991 to 0.997)	<0.001
DBP, mm Hg	0.996 (0.991 to 1.001)	0.14
HR, beats per minute	1.003 (0.999 to 1.006)	0.11
Oxygen saturation, %	0.967 (0.961 to 0.972)	<0.001
Respiratory rate, breaths per minute	1.027 (1.019 to 1.035)	<0.001
White cell count, ×10^9/L	1.008 (1.001 to 1.016)	0.02
Glucose, mg/dL	1.001 (1 to 1.001)	0.001
Aspartate aminotransferase, U/L	1 (1 to 1.001)	0.21
Alanine aminotransferase, U/L	1 (0.999 to 1)	0.25
Lactic acid, mmol/L	1.071 (1.036 to 1.107)	<0.001
Platelet count, k/µL	0.999 (0.998 to 0.999)	<0.001
Potassium, mEq/L	1.096 (1.028 to 1.168)	0.0052
Bicarbonates, mEq/L	0.957 (0.944 to 0.971)	<0.001
Creatinine, mg/dL	1.023 (0.998 to 1.049)	0.069
HTN, yes/no	1.008 (0.851 to 1.194)	0.93
HLD, yes/no	1.196 (1.02 to 1.401)	0.027
CKD, yes/no	1.263 (1.09 to 1.462)	0.002
HF, yes/no	1.33 (1.146 to 1.543)	<0.001
COPD/asthma, yes/no	0.948 (0.827 to 1.088)	0.45
DM, yes/no	0.946 (0.819 to 1.093)	0.45
CAD, yes/no	1.101 (0.955 to 1.271)	0.19
Statin use, %	0.577 (0.501 to 0.664)	<0.001
Bed occupancy, %	1.007 (1.001 to 1.013)	0.004

CAD, coronary artery disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; DBP, diastolic blood pressure; DM, diabetes mellitus; HF, heart failure; HLD, hyperlipidaemia; HR, heart rate; HTN, hypertension; SBP, systolic blood pressure.

reduced. PS analysis showed a significant reduction in in-hospital mortality during winter (HR 0.580, CI 0.507 to 0.663, p<0.001), confirming what we observed in the multivariable adjusted analysis (figure 4B).

DISCUSSION

We examined inpatient mortality from COVID-19 over the course of a 1-year pandemic at our hospital system in New York City. Our principal findings are as follows. First, we observed a substantial reduction in in-hospital mortality, coinciding with multiple pandemic-related public health measures focusing on hospital resource management—and preceding comprehensive changes in pharmacotherapy—towards the end of the first surge. Second, we describe for the first time hospital bed occupancy as an independent risk factor for inpatient mortality from COVID-19.

Public health measures in response to COVID-19

After declaring a state of disaster emergency (7 March 2020), New York State introduced different measures to limit the spread of the disease, including public school closures (16 March 2020), limitation in indoor dining (17 March 2020), stay-home order for non-essential workers (22 March 2020), mandatory face coverings in public (15 April 2020) and night subway closure (30 April 2020).[24] Despite these measures to limit the diffusion of the disease and a generalised reduction of movements around New York City (as evidenced by a more than 90% reduction of subway ridership compared with 2019),[25] more than 30% of Bronx residents were found to have positive antibodies (and thus possibly temporary immunity) against SARS-CoV-2 in August 2020.[26]

Specifically relevant to hospital operations, executive order number 202.5 (16 March 2020)[27] allowed healthcare providers not licensed or registered in New York State to temporarily work in the state, and executive order number 202.10 (22 March 2020)[27] suspended elective operations. These executive orders were associated with a dramatic drop in non-COVID-19 admissions at our institution beginning 16 March 2020 (figure 1). On 26 March 2020 New York State Governor Cuomo additionally mandated all hospitals to increase their bed capacity by 50% to accommodate the surge of patients with COVID-19.[27] Despite this order, the actual bed occupancy at our institution (while accommodating all patients with COVID-19 presenting to our hospitals) remained below the usual operating capacity until December 2020.

Notably, COVID-19 mortality remained stable throughout the summer and fall of 2020, with low case counts and increased utilisation of steroids, anticoagulation and remdesivir. Although randomised controlled trials have shown morbidity benefits with the use of remdesivir[7] and mortality reduction with steroids,[8] the magnitude of these effects cannot explain the more than 50% reduction in mortality we observed. Furthermore, pharmacotherapy, with the exception of hydroxychloroquine elimination, did not materially change within the spring season, by the end of which mortality was already decreased. Steroid, remdesivir and therapeutic anticoagulation were used in 10%–20% of patients by May 2020, but they reached 30%–70% only in the winter season. Despite this, unadjusted mortality began to increase again in December 2020 during the second wave. Of note, bed occupancy also increased at that time and proved to be an independent risk factor for COVID-19 mortality in our cohort of nearly 8000 patients.

Change in therapeutic approach

The initial widespread (more than two-thirds of first spring patients) use of hydroxychloroquine, an agent eventually proven to be ineffective[28] to treat COVID-19, probably

represents the most obvious pandemic-associated deviation from the usual multiphase clinical trial standards of therapeutic paradigm development. Only 8 of 2254 patients received hydroxychloroquine during the winter wave. Similarly, we observed a reduction in the use of azithromycin and other antibiotics, the latter possibly reflecting a more careful assessment of the need to treat superimposed bacterial infections during the second wave. Steroid therapy[8 29] and therapeutic anticoagulation[9] were implemented in the majority of patients during the winter after the knowledge on the likely disease modulating inflammatory proprieties and prothrombotic effect of COVID-19 had been recognised[30] and, in the case of steroids, a therapeutic effect had been proven.[8] Remdesivir, an inhibitor of the viral RNA-dependent RNA polymerase that showed shortening of recovery time in hospitalised patients with COVID-19,[7] received emergency approval from the Food and Drug Administration on 22 October 2020[31] and was administered to almost half of the admitted patients during the winter. If initial concerns of possible interactions between ACE-I or ARBs and SARS-CoV-2[32] led to a possible underutilisation or discontinuation of these drugs during the spring, we observed a significant increase in their use during the following months, after no increased risks were reported.[33 34]

Similarly, after several reports showed a possible protective effect associated with the use of statins,[35 36] their utilisation markedly increased during the winter.

Lastly, after the spring wave provided anecdotal evidence for early proning in COVID-19 pneumonia, an approach strongly favouring non-invasive ventilation and avoiding intubation was developed to address respiratory distress in COVID-19; more data about such an approach have since accumulated.[10 37] The cumulative effect of these therapeutic changes, in combination with a better preparedness to respond to a pandemic, can be estimate from the different mortality between the first surge (spring) and the second surge (winter). After matching the two groups in demographic and clinical variables, as well as in elements indicative of hospital distress (bed occupancy), a significant reduction in mortality was observed during the winter trimester.

Change in hospital stress load

At the peak of the pandemic, the hospital saturation reached the 118% of the nominal bed capacity and patients with COVID-19 accounted for 68.2% of all admitted patients. This increase in acutely ill patients created significant excess demand on the rest of the hospital infrastructure best characterised by the surge in the need for intensive care unit (ICU) beds and transformation of other hospital areas to ICUs.[14 23] Despite increased patient load, the number of standard ICU beds, as well as laboratories, diagnostic equipment and available personnel, remained the same as before the pandemic. This unmatched patient overload resulted in a 0.7% mortality increase for each 1% increment in hospital bed saturation. In light of these results, strategies to minimise the bed occupancy for patients without COVID-19 or non-life-saving admission should be adopted to diverge resources to improve the outcome of admitted patients with COVID-19.

Limitations

Our study has the shortcomings of a retrospective investigation, but there are some very specific aspects limiting the interpretation of our results. First, it is difficult to assess the true effects of pharmacotherapy given the dynamic changes in indications, doses and usage that happened over the course of the year. Regardless, we believe the propensity matched comparison between the spring and the winter waves provides compelling evidence for the validity of our principal observation of inpatient COVID-19 mortality reduction disproportionate to advances in pharmacotherapy. We chose total bed occupancy as a metric for hospital stress assuming that other resources per bed remained static. Notably, the ratio of patients with COVID-19 to those without COVID-19, ICU bed saturation and staff shortages are unaccounted for in this model. Regrettably, an indepth analysis of these metrics is beyond our ability in this retrospective pandemic analysis with disaster elements. Additionally, a significant number of patients received ICU-level-of-care interventions (mechanical ventilatory support, dialysis, vasopressor titration) on regular floors; therefore, the concept of ICU bed saturation might have been not truly representative of the burden.

However, we feel our data are sufficiently strong to support the notion that bed capacity expansion alone is not the answer. Rather, a smaller number of beds with higher staffing accomplished by drastic reductions in all non-emergent procedures and activities is likely a better approach. Although offering fewer beds in a pandemic situation appears initially quite counterintuitive, in practice we observed that mortality began to decrease once beds and resources were allocated specifically to patients with COVID-19 by executive orders 202.5 and 202.10, and most importantly that bed occupancy never exceeded 100% once hospital operations focused on the COVID-19 pandemic only. It is conceivable that an uptrend in mortality observed late in the pandemic with established treatment paradigms could be due to new viral strains or a sicker patient population. Although we are unable to provide detailed strain analysis for our study population, a meaningful number of new (and possibly more virulent) strains were not yet observed in the Bronx, where our study was conducted.[38] The small sample size of patients in summer and fall does not allow meaningful propensity matched comparisons, and when comparing summer, fall and winter populations there do not appear to be clinically meaningful differences. Lastly, single-patient data on vaccination status were not available. At the conclusion of the study, only 13.8% of the population of New York State have received at least one dose and 7.4% have received two doses.[39] Given the heterogeneous distribution

of vaccination within the state (and the city of New York), it is impossible to meaningfully account for these parameters.

CONCLUSIONS

Inpatient mortality from COVID-19 decreased to a degree disproportionate to advances in disease-specific therapeutics. Increased bed occupancy was associated with higher in-hospital mortality. Implementation of non-pharmacological approaches and other seasonal variations might also had a role in mortality reduction. Early reduction in epicentre hospital bed occupancy to accommodate acutely ill and resource-intensive patients should be a critical component in the strategic planning for future pandemics.

Author affiliations
[1]Albert Einstein College of Medicine, Bronx, New York, USA
[2]Division of Cardiology, Montefiore Medical Center, Bronx, New York, USA
[3]Department of Epidemiology and Population Health, Albert Einstein College of Medicine, Bronx, New York, USA
[4]Division of Cardiology, Massachusetts General Hospital, Boston, Massachusetts, USA
[5]Division of Infectious Diseases, Montefiore Medical Center, Bronx, New York, USA
[6]Department of Pediatrics, Montefiore Medical Center, Bronx, New York, USA

Twitter Francesco Castagna @FraCastagna_MD

Contributors Design of the project: FC, XX and UPJ. Underlying data verification: FC, XX and UPJ. Acquisition, analysis and interpretation of data: FC, XX, OS, RK, YAP, SRP, MJG, ADR, DBS and UPJ. Statistical analysis: FC and XX. Obtained funding: UPJ. Manuscript writing: FC, XX and UPJ. Critical revision of the manuscript for important intellectual content: FC, XX, OS, RK, YAP, SRP, MJG, ADR, DBS and UPJ. Supervision: UPJ. Guarantor: UPJ.

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Competing interests None declared.

Patient consent for publication Not required.

Ethics approval The Office of Human Research Affairs at Albert Einstein College of Medicine approved this study (#2020-11308). Patient consent and HIPAA forms were waived by our IRB due to the retrospective nature of our research.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. Request for de-identified data and dictionaries will be evaluated on a case-by-case basis after the submission of a research proposal to the corresponding author and the signature of a data access agreement. Data will be available after manuscript publication.

ORCID iD
Francesco Castagna http://orcid.org/0000-0002-8983-4866

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