

Original Research

Prior cardiovascular risk and screening echocardiograms predict hospitalization and severity of coronavirus infection among elderly medicare patients



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ABSTRACT

Background: The COVID-19 pandemic has disproportionately impacted the elderly. In the United States and Europe the mortality rate of elderly patients with COVID-19 is greater than 30%. Our aim is to determine predictors of COVID-19 related hospitalization and severity of disease among elderly Medicare patients in the United States.

Methods: We conducted a retrospective cohort study including elderly Medicare COVID-19 patients across eight states. We collected data from the inpatient and outpatient electronic health record, demographic, clinical and echocardiographic predictors. Our primary outcomes were hospitalization and adult respiratory distress syndrome (ARDS). Our secondary outcome was mortality.

Results: We identified 400 COVID-19 positive patients (incidence 5.2; (95% CI 4.7–5.7) per 1000 patients). The mean age of our patients was 72 ± 8 , 60% were female, 82% were minorities and had a mean Charlson score of 2.9 ± 1.4 . Two-hundred and forty-four patients were hospitalized due to COVID-19 (63%) and the mortality rate was 18%; 95% CI 14–22 with 1 patient still in the hospital. Age, socioeconomic status, Charlson score, systolic blood pressure, body mass index, grade 2 or 3 diastolic dysfunction, moderate or severe left ventricular hypertrophy were significant predictors of hospitalization and ARDS ($p < 0.05$).

Conclusions: Our study reports a lower incidence on a COVID-19 cohort than previously reported. Predictors of poor outcomes included socio-economic, cardiovascular risk and echocardiographic measures. High touch care with early cardiovascular risk factor modification could explain the low risk of events in our population.

1. Introduction

The coronavirus (COVID-19) pandemic is causing widespread morbidity, and in certain states, is directly responsible for the collapse of the healthcare system due to the increased demand for ICU beds and mechanical ventilators [1].

Several studies from Asia, Europe and the United States have reported higher mortality among the elderly [2,3]. Patients with severe COVID-19 who die have twice the prevalence of cardiovascular risk factors and coronary artery disease than COVID patients without severe disease [4–7]. At the same time, finding myocardial

injury, as measured by troponins, on COVID-19 positive patients carry a significant risk of mortality [6]. It seems myocardial dynamics play a role in the morbidity and mortality of COVID-19 patients.

Understanding the cardiovascular profile of elderly patients who have poor outcomes is of paramount importance to tailor surveillance and preventive efforts more effectively. Therefore, our aim was two-fold. First, evaluate the incidence of COVID-19 among an elderly Medicare population. Second, to evaluate demographic, clinical and echocardiographic predictors of poor outcomes among Medicare patients who acquired the COVID19 virus.

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2. Methods

2.1. Study setting

We conducted the study at Chen Senior Medical Centers (CSMC), JenCare Senior Medical Center (JCSMC) and Dedicated Senior Medical Centers (DSMC). These are fully capitated group network practices located across eight states. Patients are insured through Medicare Advantage Plans that serve as fiscal intermediaries for processing claims. As part of a system-wide focus on prevention and wellness, patients are seen every month by their primary care providers and undergo an initial research screening echocardiogram upon establishing care. The population served is well over 75,000 Medicare Advantage patients.

2.2. Study design and study population

We conducted a retrospective cohort study of all the patients who tested positive for COVID-19 in the clinics between January 1, 2020 and May 4, 2020. Follow-up of the cases concluded on May 17, 2020. We included all consecutive COVID-19 patients and defined COVID-19 positive patients as a positive RT-PCR test for SARS-CoV-2. The tests were done at Quest labs. Given the high touch care model [8] and the fact that all of the clinics remained fully operational, we expect that most patients with COVID-19 symptoms would contact their clinic and/or primary care provider for arranging testing and/or care. However, if patients should receive testing in a hospital setting, clinical documentation indicating this may be received at a later time.

2.3. Outcome

Our primary outcomes were hospitalization and the diagnosis of acute respiratory distress syndrome (ARDS). We obtained hospitalization status from our electronic health record (EHR) and defined it as any patient who was admitted to the hospital for observation or for more than 24 h. The medical records contain as text files all notes and procedures of each hospital admission. We reviewed the notes from the hospitalist service and intensive care units and recorded if patients had ARDS. We defined ARDS if the chest x ray or chest computed tomography was compatible with ARDS and the physician notes mentioned ARDS in the problem list. Both primary outcomes were collected from the medical record by one of the co-authors (B.C) and a second co-author reviewed the collected information (E.D).

Our secondary outcome was mortality. We captured mortality from our EHR as all-cause mortality. All-cause mortality was ascertained and defined as at least one of the following: a) self-report from the patient's family during monthly calls conducted to all patients by the transitional care team, b) hospitalization reports from the hospitalist team and c) the Medicare claims flag. Our team retrieved data on all-cause mortality from the EHR.

2.4. Predictors

We included four types of predictors: socio-economic, clinical, echocardiographic characteristics and treatment. Our demographic predictors included age, gender, race, census based median household income and area deprivation index (ADI) of their place of residence. Age, gender and race were obtained from the EHR. We ascertained the median household income and ADI by linking the 9-digit zip code of the patient's residence to the 2010 census data [9,10]. The clinical predictors from the EHR included the Charlson score as a measure of disease burden [11], diagnoses of diabetes, hypertension and heart failure in the 3 months before the diagnosis of COVID-19. We also collected the values for systolic blood pressure, body mass index, hbA1c, total cholesterol and calculated the Framingham risk score for the last in-person visit before the COVID-19 diagnosis. For this analysis we used the values from the last primary care visit and used the last set of laboratory values available.

The median time between the last clinical visit and the COVID diagnosis was 44 days; interquartile range (IQR) (25–67). The echocardiographic predictors included diastolic dysfunction, ejection fraction and left ventricular hypertrophy. These predictors were collected from the EHR as all of our patients have a screening echocardiogram. The screening echocardiogram includes doppler mitral flow and tissue velocities tracings. Diastolic function was classified according to the recommendations of the American Society of Echocardiography (ASE) on diastolic functional evaluation. The grading scheme for diastolic dysfunction was mild or grade I, moderate or grade II, and severe (restrictive pattern) or grade III. Left ventricular hypertrophy is defined as any abnormal left ventricular wall size measurement (infero-lateral or septal thickness >0.9 cm in women or >1.0 cm in males) or a left ventricular mass index >95 g/m² in women or >115 g/m² in males. We also measured the LV ejection fraction (EF) using the modified biplane Simpson's method. A mean of three cardiac cycles was used. The mean time between the echocardiogram and the COVID diagnosis was 42.2 months; IQR (20–75). As treatment predictors we included hydroxychloroquine and azithromycin as those were the only medications used at the time the data was collected. We collected treatment information from the EHR.

2.5. Statistical analysis

We evaluated the distribution of the continuous variables using measures of central tendency and skewedness. We calculated incidence by dividing the number of new cases by the total population and the corresponding 95% confidence interval with continuity correction. We calculated mortality rates among those who were hospitalized by dividing the number of deaths by those who were hospitalized. We compared baseline characteristics using Wilcoxon rank sum test as the skewedness was greater than 0.5 and chi-square.

We conducted a multivariate analysis to evaluate how predictors were associated with the primary and secondary outcome and used cox proportional models to calculate the relative hazard (RH) and corresponding 95% confidence interval (CI). For left ventricular hypertrophy and grade of diastolic dysfunction we used dummy variables; we used no diastolic dysfunction and no left ventricular hypertrophy as the reference groups. Because of collinearity and sample size restrictions, we tested each group of predictors separate; demographics, clinical, echocardiographic and treatment. However, all models included age, gender and Charlson score.

The fitness of the data was assessed using the deviance ratio. Analyses were performed using STATA version (College Station, Texas), and all significance tests were two-tailed.

3. Results

3.1. Baseline characteristics

During our study period we identified 400 SARS-CoV-2 positive patients. The incidence of COVID-19 is 5.2 (95% CI 4.7–5.7) per 1000 patients. The median number of visits was 5 IQR (3–7). The mean age of our patients was 72 ± 8 , 60% were female and the majority came from Louisiana and Florida. The majority of the patients were Black or Hispanic 82%, most of the patients were African-American (73%) and the mean number of Charlson comorbidities was 2.9 ± 1.4 . The cohort had a median household income of \$28,249 (20,955–35,391). Table 1 shows the baseline clinical characteristics of the patients who tested positive for SARS-CoV-2 and both the hospitalized and non-hospitalized subsamples. Hospitalized patients were older, had a higher Framingham and Charlson scores and had lower income ($p < 0.05$).

3.2. Frequency of outcomes

Two-hundred and forty-four patients were hospitalized due to COVID-19 over a follow-up of 59.7 ± 6 days. Of those, 41% developed

Table 1

Baseline characteristics of the 400 SARS-CoV-2 cohort of Medicare patients and stratified by developing the primary outcome.

Characteristics	SARS-CoV-2 positive	Hospitalized COVID-19	Non- hospitalized COVID-19	p-value
Number	400	244	146	
Median age, IQR	72 (69–79)	74 (69–80)	73 (69–80)	<0.01
Female gender, %	60%	56%	64%	0.13
State, %				<0.01
Florida	24%	23%	22%	
Georgia	9%	13%	3%	
Illinois	18%	20%	13%	
Kentucky	2%	2%	2%	
Louisiana	36%	28%	48%	
Pennsylvania	5%	6%	6%	
Virginia	3%	6%	6%	
Ohio	0%	0%	0%	
Race/ethnicity, %				0.54
Black	73%	76%	68%	
Hispanic	9%	9%	11%	
Non-Hispanic White	12%	10%	15%	
Median Framingham score (IQR)	19 (12–30)	22 (13–32)	17 (10–21)	0.04
Comorbidities, %				
Heart failure	50%	52%	45%	0.18
COPD	34%	35%	31%	0.49
Diabetes	49%	51%	47%	0.45
Hypertension	60%	64%	55%	0.08
Coronary artery disease	14%	15%	14%	0.83
Median Charlson score, IQR	3 (2–4)	3 (2–4)	3 (2–4)	0.01
Census based median household income, \$ (IQR)	28,249 (20,955–35,391)	28,249 (20,955–35,391)	33,351 (28,505–39,632)	0.01
Median area deprivation index, IQR	0.3 (0.1–0.5)	0.4 (0.2–0.6)	0.3 (0.1–0.5)	0.01
Hydroxychloroquine, %	5%	9%	1%	<0.01
Azithromycin, %	26%	24%	29%	0.23
Hydroxychloroquine and azithromycin, %	20%	35%	1%	<0.01

IQR: Interquartile range.

ARDS and 34% had a diagnosis of pneumonia. Fig. 1 shows the event rates by age group. Increasing age was associated with increase probability of events ($p < 0.05$) but with the same hospital length of stay ($p = 0.34$). The median length of stay of those admitted to the hospital was 14; IQR (6–32) days. One patient is still in the hospital with a length of stay of 36 days and on mechanical ventilation. The mortality rate was 18%; 95% CI 14–22% among all SARS CoV-2 positive and 27% among patients hospitalized for COVID-19. Five patients died without ever being hospitalized and four of them died in a long-term facility and one at home. Seventeen percent of all the patients who died resided in long-term facilities. There was no difference in mortality between hospitalized patients who came from a Skilled Nursing Facility (SNF) or Long Term

Acute Care Facility (LTAC), and those who were hospitalized coming from home ($p = 0.67$).

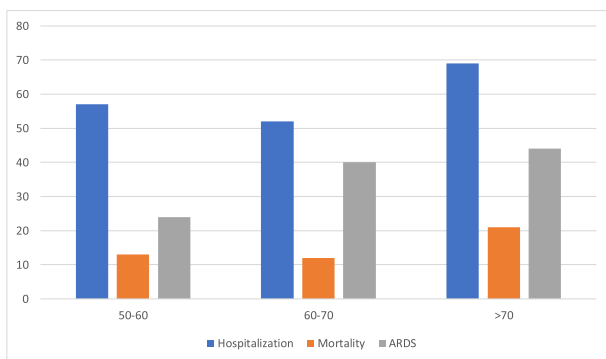
With respect to geographic differences, the mortality rates for hospitalized patients were: 28% for Florida, Georgia 19%, Illinois 26%, Kentucky 50%, Louisiana 28%, Pennsylvania 27%, and Virginia 27%. There were no statistically significant differences among states ($p > 0.05$).

3.3. Predictors of outcomes

Fig. 1 and appsec1 of the appendix shows the event rates by predictors. Table 2 shows the predictors of hospitalization, ARDS and mortality. Older age, lower socioeconomic status, higher Charlson score, higher systolic blood pressure, higher body mass index, higher Framingham risk score, grade 2 or 3 diastolic dysfunction, moderate or severe left ventricular hypertrophy were significant predictors of hospitalization; while age, socioeconomic status, hypertension, Charlson score, grade 2 or 3 diastolic dysfunction, moderate or severe left ventricular hypertrophy were significant predictors for ARDS. In the case of mortality, age, Charlson score, coronary artery disease and ejection fraction less than 40 were significant predictors. Azithromycin decreased the occurrence of ARDS and the combination of both azithromycin and hydroxychloroquine was associated with an increase in mortality and ARDS.

4. Discussion

Our study found a lower than expected incidence of COVID-19 among minority Medicare patients with low socioeconomic status residing in several states in the United States. As previously described by others, our study found that age and comorbidity burden predicted hospitalization, ARDS and all-cause mortality among patients diagnosed with COVID-19. However, our study goes further by describing other distinct predictors of COVID-19 outcomes. Predictors of hospitalization and ARDS were: (1) census-based socioeconomic status (as demographic predictors), (2) Framingham risk score (as clinical predictors); and (3) left ventricular



Age category	Number	Mortality, No. (%)
50-60 years	28	3 (11%)
60-70 years	118	14 (12%)
>70	254	55 (22%)

Fig. 1. Percentage of the primary and secondary outcome (y axis) by age group category and number of deaths by age category.

Table 2
Multivariate predictors of hospitalization, mortality and ARDS.

Predictors	RH of hospitalization (95% CI) p-value	RH of death (95% CI) p-value	RH of ARDS (95% CI) p-value
Demographics			
Age	1.03 (1.01–1.06) <0.01	1.05 (1.01–1.08) <0.01	1.03 (1.00–1.05) <0.01
Male gender	1.40 (0.92–2.15) 0.11	1.32 (0.75–2.33) 0.33	1.02 (0.67–1.56) 0.90
Black or Hispanic	1.11 (0.67–1.84) 0.66	1.30 (0.62–2.7) 0.47	1.30 (0.78–2.16) 0.31
Census based income (n = 325)	0.99 (0.99–0.99) 0.02	1.00 (0.99–1.00) 0.84	0.99 (0.99–0.99) 0.03
Area deprivation index (n = 325)	0.99 (0.99–0.99) 0.02	1.00 (0.99–1.00) 0.95	0.99 (0.99–0.99) 0.03
Clinical			
Hypertension	1.45 (0.95–2.02) 0.08	1.38 (0.76–2.50) 0.28	2.03 (1.35–3.2) <0.01
Diabetes	1.17 (0.77–1.77) 0.45	1.03 (0.58–1.02) 0.90	1.13 (0.75–1.71) 0.55
Heart failure	1.32 (0.87–2.00) 0.18	1.25 (0.71–2.21) 0.41	1.17 (0.77–1.76) 0.45
Charlson score	1.17 (1.01–1.35) 0.02	1.12 (0.92–1.36) <0.01	1.15 (1.00–1.33) 0.04
Coronary artery disease	1.06 (0.58–1.92) 0.63	1.84 (1.05–2.36) 0.04	0.85 (0.42–1.53) 0.59
Systolic blood pressure (n = 374)	1.28 (1.10–1.35) <0.01	1.00 (0.98–1.02) 0.73	1.25 (1.01–1.36) 0.03
Body mass index (n = 389)	1.06 (1.03–1.16) 0.02	1.00 (0.96–1.05) 0.67	1.02 (0.98–1.05) 0.23
Total cholesterol (n = 369)	0.99 (0.98–1.00) 0.52	0.99 (0.99–1.00) 0.94	0.99 (0.99–1.00) 0.16
Hba1c (n = 259)	1.08 (0.91–1.29) 0.33	1.02 (0.82–1.27) 0.80	1.03 (0.88–1.22) 0.64
Framingham risk score (n = 365)	3.47 (1.5–6.7) <0.01	2.15 (0.8–10.7) 0.12	3.75 (1.3–5.5) <0.01
Treatment			
Azithromycin	0.80 (0.53–1.21) 0.30	0.73 (0.40–1.32) 0.30	0.58 (0.36–0.91) 0.01
Azithromycin and hydroxychloroquine	NA	2.78 (1.61–4.78) <0.01	21.1 (11.3–39.6) <0.01
Echocardiographic			
Ejection fraction	1.0 (0.97–1.04) 0.69	0.96 (0.92–0.99) 0.04	1.01 (0.97–1.04) 0.56
Diastolic dysfunction			
Grade 1	1.14 (1.01–1.75) 0.04	1.03 (0.85–1.10) 0.45	1.25 (0.83–2.11) 0.22
Grade 2 or 3	1.77 (1.10–3.3) 0.03	1.06 (0.80–1.25) 0.25	1.33 (1.02–2.0) 0.03
Left ventricular hypertrophy			
Mild	1.16 (0.70–1.93) 0.54	1.04 (0.11–9.56) 0.96	1.05 (0.65–1.73) 0.56
Moderate	1.87 (1.01–3.30) 0.03	1.14 (0.12–10.8) 0.90	1.66 (1.02–3.10) 0.03

Table 2 (continued)

Predictors	RH of hospitalization (95% CI) p-value	RH of death (95% CI) p-value	RH of ARDS (95% CI) p-value
Severe	3.42 (1.10–4.35) 0.04	1.45 (0.51–9.35) 0.89	2.52 (1.05–3.67) 0.04

The sample size of each cell is 400 unless specified.

RH: relative hazard.

CI: confidence interval.

thickness (as echocardiographic predictors). Increasing BMI predicted hospitalization while diastolic dysfunction predicted ARDS. Interestingly, we did not observe gender differences in any of the outcomes and our cohort had a higher representation of female patients when compared to other reports.

Our study has several strengths that lend weight to our findings. Among those are the multicenter, national design, the large sample size, the inclusion of unselected consecutive primary care patients and the availability of relevant clinical, demographic and echocardiographic data to adjust our models.

Our study contributes information on the mortality rate among minority Medicare beneficiaries in several states. Little is known about the mortality rate of patients with a diagnosis of COVID-19 since most of the published data report on hospitalized patients, our study reports mortality for all. Slightly over a quarter of all patients hospitalized for COVID-19 died during the period of follow up. This is comparable to the mortality rates reported by other investigators. The first report, by Liu et al. and conducted in the Hainan Provincial People’s Hospital reported on 18 elderly patients (mean age 68 years) [12]. The study found more severe disease and a higher mortality when compared to 38 middle aged patients. McMichael et al. reported a mortality rate of 30% among 101 elderly patients (mean age 83) residing in a long-term facility in Washington State. Of them, 60% had known heart disease [13].

Two recent larger studies have also reported on mortality. In New York (Richardson et al.), a case series of 5700 COVID-19 hospitalized patients with a mean age of 63 years had a mortality rate of 26.6% among patients who were older than 50 years of age [14]. In Italy, Grasselli et al. reported a case series of 1591 consecutive COVID-19 positive patients treated in the intensive care units of 72 hospitals. The patients were mostly males with a median age of 63 years and the study reported that 33% of those who were 60 and older died. This mortality rate, although higher than ours, was calculated for patients treated in the intensive care units in Italy making comparisons difficult [15].

Our findings that age and increasing number of co-morbid conditions are strong predictors of mortality are consistent with prior reports. The mechanisms by which comorbidity burden leads to an increase risk of COVID mortality are not fully elucidated. Having heart disease could be a potential mechanism by which having comorbidity is associated with mortality. Studies in China that evaluated the characteristics of patients with severe COVID-19 disease or who died from it, revealed the strong association between heart disease and these adverse outcomes [4–7].

Data on the potential mechanisms by which CVD increases COVID-19 mortality is starting to emerge. For example, patients with COVID-19 and significant heart disease when hospitalized can have worsening ischemia or fluid shifts which can lead to worsening heart failure, and our results support this [5]. Others have showed that seventy percent of patients who have elevated troponins and cardiovascular disease (CVD) die from COVID-19 while only 8% of those who die have no CVD and normal troponins. These findings hint that pre-existing heart disease can somehow increase the risk for myocardial damage or inflammation that is evidenced by the increase in troponins.

A recent study by Leung et al. reported on 154 COVID patients in China with a median age of 75 years reported a mortality rate of 58% and reported age, dyspnea and chest pain as predictors of mortality [16].

Access to the cardiovascular risk and echocardiographic data of our cohort of patients prior the SARS-CoV-2 infection allowed us to make two significant contributions in this area: First, we found that earlier forms of heart disease as manifested by cardiovascular risk factors and the Framingham score are associated with hospitalizations and the more severe form of COVID-19, ARDS. In contrast, others had reported that the presence of known cardiovascular diseases like coronary artery disease or heart failure were associated with mortality in COVID-19 patients [1,4,5]. Our findings may facilitate more effective preventive efforts. Second, we found that patients with early structural heart changes such as diastolic dysfunction and left ventricular hypertrophy are significantly more likely to be hospitalized for COVID-19. This is an important finding as the echocardiogram was performed several months before the COVID-19 diagnosis. This finding continues to support the growing literature that screening echocardiograms improve outcomes in senior a population. We have previously found that screening echocardiograms identify stage B heart failure and when this information was shared with the physician there were medication changes and improved outcomes and costs [18,19].

The mechanisms by which cardiovascular risk factors without known heart disease worsen outcomes are even less understood. Our prior work has revealed that patients without known heart disease but with conditions such as diabetes or hypertension had myocardial fibrosis, a known risk factor of ventricular arrhythmias, in cardiac magnetic resonance [17] Future studies should evaluate further the impact of cardiovascular risk factor control on COVID-19 morbidity and mortality. This data can strengthen models of care that mitigate COVID-19 risk among vulnerable patients. High-touch care with frequent preventative visits with primary care provider and specialists has been shown to result in higher use of cardiovascular medications, fewer hospitalizations and lower healthcare costs [8,18,19].

Our study also found that social determinants of health such as area deprivation index and household income are important predictors of both COVID-19 morbidity and mortality in our cohort of patients. This may explain the emerging evidence that COVID-19 is impacting minorities differentially [20]. Our data suggests that this may be mediated by economic disadvantage which is more prevalent in communities of color and not by biological factors. This is consistent with the growing evidence that social determinants of health are important predictors of racial disparities on cardiovascular diseases.

There are several limitations that deserve mention. First, we relied on a retrospective review of the medical record hence the inclusion of comorbidities using administrative codes could have led to information bias. Second, mortality data has a time delay preventing us from identifying all deaths. Third, our low incidence could be explained by the fact that at the time of this report widespread COVID-19 testing was not available and we only reported symptomatic patients who had a COVID-19 diagnostic test. Fourth, our sample size was limited and therefore could not construct fitted prediction models to adjust for all possible confounders. Fifth, we could not report any blood biomarkers that could influence in-hospital mortality.

In conclusion, this study provides evidence that early stages of cardiovascular disease and socio-economic disadvantage increase the probability of poor outcomes after a COVID-19 diagnosis. This data suggests that patients with good cardiovascular profiles, as measured by echocardiography and clinical evaluation, do better when diagnosed with COVID-19. Future studies must evaluate the biological mechanisms through which cardiovascular risk factors increase COVID-19 risk. COVID-19 prevention among vulnerable populations must consider the improvement of their cardiovascular risk profile as a key strategy.

Disclosures

No conflicts to disclose.

Credit author statement

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ajpc.2020.100090>.

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