

Incidence and implications of atrial fibrillation in patients hospitalized for COVID compared to non-COVID pneumonia: A multicenter cohort study

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BACKGROUND Atrial fibrillation (AF) has been reported to occur with coronavirus disease 2019 (COVID-19), but whether it is related to myocarditis or lung injury is unclear.

OBJECTIVES The purpose of this study was to compare incident AF in patients with pneumonia/adult respiratory distress syndrome (ARDS) with and without COVID.

METHODS This retrospective multicenter cohort study from 17 hospitals (March 2020 to December 2021) utilizing the University of California COVID Research Data Set (CORDS) included patients aged ≥ 18 years with primary diagnosis of pneumonia or ARDS during hospitalization. Patients with a history of AF were excluded. All subjects had documented COVID test results. Cohorts were compared using the χ^2 test for categorical variables and the Wilcoxon rank test for continuous variables. Multivariable logistic regression models were used to investigate the association between COVID and development of new AF.

RESULTS Of the 39,415 subjects, 12.2% had COVID. The COVID+ cohort consisted predominantly of younger males with more comorbidities. Incident AF was lower in the COVID+ group than in the

non-COVID group (523 [10.85%] vs 4899 [14.16%]; odds ratio [OR] 0.74; $P < .001$), which remained significant after adjustment for demographics and comorbidities (OR 0.71; $P < .001$). Patients had normal cardiac troponin levels. AF was related to intensive care unit care, pressor support, and mechanical ventilation, and was associated with higher mortality (26.2% vs 10.21%; $P < .001$) and longer hospitalization (22.5 vs 15.1 days; $P < .001$) in the COVID+ group compared to the controls.

CONCLUSION Incident AF is lower in COVID+ compared to non-COVID pneumonia/ARDS patients and seems to be related to severity of illness rather cardiac injury. AF was associated with higher mortality and prolonged hospitalization.

KEYWORDS Atrial fibrillation; Coronavirus disease 2019 (COVID-19); Lung disease

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Introduction

Atrial fibrillation (AF) is the most common cardiac rhythm disorder and is associated with significant morbidity and mortality.¹ The development and sustainment of AF are multifactorial, with greater risk given increasing age and comorbidities such as hypertension, coronary artery disease, cerebrovascular disease, and diabetes.¹ Increased sympathetic tone and circulating inflammatory factors also are associated with the development of AF.^{2,3}

Coronavirus disease 2019 (COVID-19), which is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has resulted in a worldwide pandemic during the first year of the pandemic affecting more than 500 million people worldwide and 80 million people in the United States.⁴ Infec-

tion can lead to illnesses of varying severity, ranging from mild upper respiratory infection to severe pneumonia and adult respiratory distress syndrome (ARDS) requiring mechanical ventilation. Although COVID-19 is primarily considered a lung disease driven by an inflammatory immune response,^{5,6} cardiovascular involvement also has been reported.⁷ Cardiac manifestations of COVID-19 include myocarditis, heart failure, arrhythmia, and acute coronary syndrome, and can be associated with higher mortality.^{7,8}

A high incidence of AF has been seen in patients with COVID-19 and is associated with significantly worse outcomes.^{9,10} Partially because of the multiple shared risk factors, little is known about the incidence of AF in COVID-19 patients, and current studies are limited to relatively small sample sizes without controls. In addition, whether the high rates of AF seen in patients with severe COVID-19 infection are directly related to inherent virus virulence or are due to a nonspecific response to severe respiratory illness, resultant hypoxia, and/or therapeutic interventions is uncertain.

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KEY FINDINGS

- The incidence of new-onset atrial fibrillation (AF) in patients hospitalized for coronavirus disease 2019 (COVID-19) pneumonia was approximately 10%.
- Compared to patients with non-COVID pneumonia and acute respiratory distress syndrome, the incidence of new-onset atrial fibrillation is less in patients with COVID pneumonia.
- AF is not directly associated with COVID-19, but development of incident AF is a poor prognostic factor, associated with higher mortality and longer hospitalization.

We sought to measure the incidence of new-onset AF in patients with COVID-19 and directly compare it to the incidence in a cohort of subjects with non-COVID-19 pneumonias and ARDS in order to assess whether COVID-19 is a risk factor for the development of AF. We hypothesize that the high rates of AF seen in COVID-19 patients is attributable to severe respiratory illness and not COVID-19 itself; therefore, we expect to see similar rates of incident AF in a non-COVID-19 comparison group having a similar pulmonary disease process.

Methods

This was a multicenter retrospective cohort study utilizing the University of California COVID Research Data Set (CORDS). This database includes all patients tested for SARS-CoV-2 and treated at any of the 17 University of California (UC) Health hospitals across 5 academic medical centers (Davis, Irvine, Los Angeles, San Diego, San Francisco). Data were extracted for the period from March 2020 to December 2021.

Subjects were included if they were admitted to an inpatient facility and had a diagnosis of pneumonia or ARDS associated with their admission, determined using the *International Statistical Classification of Diseases, Tenth Revision (ICD-10)* codes in the patient electronic health record (EHR). If patients had multiple hospital admissions, only data from the first admission were included. Subjects were excluded if they had a previous diagnosis of AF.

Subjects were then divided into 2 cohorts based on their SARS-CoV-2 test results. Patients were placed in the COVID pneumonia cohort (COVID+) if they had a positive polymerase chain reaction COVID test confirmation within 10 days or during an inpatient admission. Patients without such a positive COVID test were placed in the non-COVID pneumonia cohort (non-COVID). All patients included in the database had an associated COVID test.

Patient characteristics, including race/ethnicity, sex, comorbidities, inpatient medications, and clinical outcomes, were collected. Ages were estimated from patient birth years because birth dates were removed from the database for de-identification. Self-identified race/ethnicity was categorized into Asian, White, Hispanic/Latino, Black/African American, American Indian or Alaska Native (AIAN), Native Hawaiian or Other Pacific Islander (NHPI), Other, and Unknown. The following comorbidities (using ICD-10 classification obtained from patient EHRs) were identified: hypertension, diabetes mellitus, coronary artery disease, previous history of coronary artery bypass graft, history of prosthetic heart valve, peripheral vascular disease, congestive heart failure, obstructive sleep apnea, chronic obstructive pulmonary disease, end-stage renal disease, cirrhosis, and thyroid disease. Comorbidities were considered present if the subject had a documented history of that condition before admission. Medications given during the hospitalization were queried from the EHR and grouped by general class of medication, including vasopressor drugs and

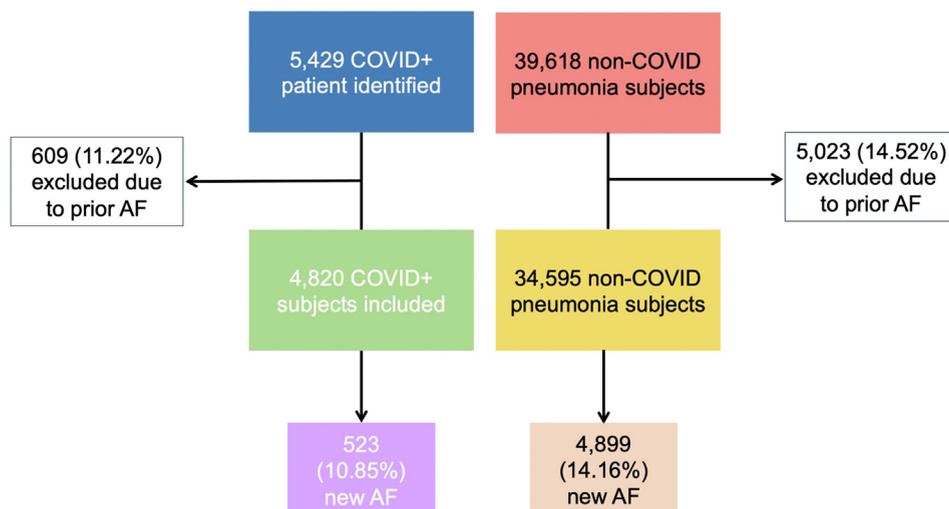


Figure 1 Incidence of new-onset atrial fibrillation (AF) among patients hospitalized for coronavirus disease 2019 (COVID-19) vs non-COVID pneumonia.

Table 1 Baseline characteristics of COVID patients compared to non-COVID patients with respiratory illness

Characteristic	COVID+ (n = 4820)	Non-COVID (n = 34,595)	P value
Female	2022 (41.95)	15,535 (44.91)	.001
Age group (y)			<.001
18–29	275 (5.71)	2134 (6.17)	
30–39	463 (9.61)	2842 (8.22)	
41–49	625 (12.97)	3329 (9.62)	
51–59	1001 (20.77)	5942 (17.18)	
61–69	1050 (21.78)	8532 (24.66)	
71–78	685 (14.21)	6311 (18.24)	
78+	721 (14.96)	5505 (15.91)	
Race			<.001
Asian	559 (11.60)	4247 (12.28)	
Black or AA	384 (7.97)	2854 (8.25)	
White	1051 (21.80)	14,019 (40.52)	
Hispanic or Latino	929 (19.27)	4569 (13.21)	
AIAN	14 (.29)	132 (.38)	
NHPI	50 (1.04)	208 (.60)	
Other	1135 (23.55)	5074 (14.67)	
Unknown	698 (14.48)	3492 (10.09)	
Comorbidity			
Hypertension	1910 (39.63)	12,392 (35.82)	<.001
DM	2031 (42.14)	12,425 (35.92)	<.001
Coronary artery disease	836 (17.34)	6264 (18.11)	.197
Previous CABG	112 (2.32)	799 (2.31)	.951
Peripheral vascular disease	338 (7.01)	2600 (7.52)	.213
Heart failure	491 (10.19)	3660 (10.58)	.405
Sleep apnea	425 (8.82)	2917 (8.43)	.368
COPD	297 (6.16)	2521 (7.29)	.004
ESRD	385 (7.99)	1940 (5.61)	<.001
Cirrhosis	154 (3.20)	1790 (5.17)	<.001
Thyroid disease	460 (9.54)	3587 (10.37)	.077
Hospital course			
ICU	1615 (33.51)	13,288 (38.41)	<.001
Pressor use	934 (19.38)	10,519 (30.41)	<.001
Mechanical ventilation	637 (13.22)	2576 (7.45)	<.001
Outcome			
Death	400 (8.22)	1679 (4.85)	<.001
Length of stay (d)	8 [4–16]	6 [3–12]	<.001

Values are given as n (%) or median [interquartile range] unless otherwise indicated.

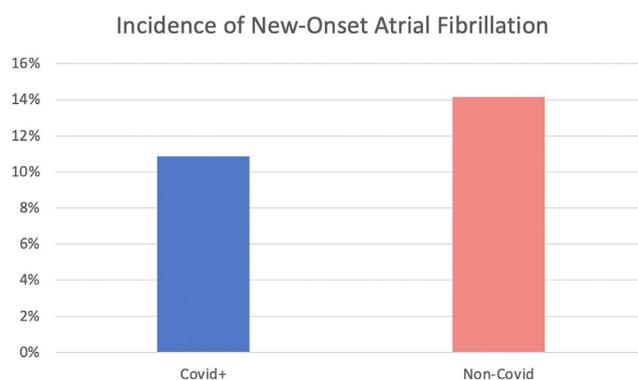
AA = African American; AIAN = American Indian or Alaska Native; CABG = coronary artery bypass graft; COPD = chronic obstructive pulmonary disease; COVID = coronavirus disease; DM = diabetes mellitus; ESRD = end-stage renal disease; ICU = intensive care unit; NHPI = Native Hawaiian or other Pacific islander.

antiarrhythmic drugs. Laboratory values represent the first laboratory value drawn during the subject's admission.

These Health Insurance Portability and Accountability Act of 1996 (HIPAA)-limited data were determined to be exempt from human subject protection and patient consent by UC Davis IRB under protocol 1879428-1. The research reported in this paper adhered to Helsinki Declaration guidelines.

Statistical analysis

Values are given as number (%) and continuous variables as median [interquartile range]. Baseline characteristics are given for patients diagnosed with new-onset AF, comparing

**Figure 2** Incidence of new-onset atrial fibrillation in coronavirus disease 2019 (COVID)+ and non-COVID patients.

those with COVID pneumonia to those with non-COVID pneumonias. *P* values were calculated, using the χ^2 test for categorical variables and the Wilcoxon rank test for continuous variables.

Univariate analysis was performed first to identify the significant variables associated with development of AF. Multivariate logistic regression analysis then was performed to identify significant predictors. Candidate variables for model inclusion were those that were statistically and clinically relevant variables¹ associated with AF and those with *P* < .05 on univariate analysis.

Results

A total of 45,047 subjects were included based on diagnosis of pneumonia or ARDS. Of these patients, 5632 (12.5%) were excluded because of previous AF history. Of the 39,415 remaining subjects, 4820 (12.23%) had an associated positive COVID test and were placed in the COVID+ cohort, compared to 34,595 (87.77%) with negative COVID test who were placed in the non-COVID cohort (Figure 1). A total of 1558 subjects (3.95%) had ARDS in addition to pneumonia. Patients in the COVID+ cohort were more likely to be <60 years old (49.1% vs 41.2%; *P* < .001), male (58.1% vs 55.1%; *P* = .001), and non-White (78.2% vs 59.5%; *P* < .001), and to have hypertension (39.6% vs 35.8%; *P* < .001) and diabetes (42.1% vs 35.9%; *P* < .001) (Table 1). COVID+ patients were more likely to require mechanical ventilation but required less hemodynamic support or intensive care unit care (Table 1).

Incident AF

A total of 5422 subjects developed new-onset AF, particularly male patients (61.6%) and older (>60 years) patients (81.3%), with a lower incidence of 523 (10.85%) in the COVID+ cohort vs 4899 (14.16%) in the non-COVID cohort (OR 0.738; *P* < .001) (Figure 2). Patients with incident AF in the COVID+ cohort AF were significantly more likely to have diabetes and hypertension than those without COVID-19 (Table 2).

Significant predictors of development of new-onset AF in COVID+ patients included male sex (OR 1.33; *P* < .001),

Table 2 Characteristics of patients with new-onset AF during hospitalization for pneumonia, comparing COVID+ to non-COVID patients

Characteristic	COVID+ (n = 523)	Non-COVID (n = 4899)	P value
Female	178 (34.03)	1905 (39.52)	.03
Age group (y)			.029
18–29	7 (1.34)	40 (.82)	
30–39	11 (2.10)	91 (1.86)	
41–49	25 (4.78)	196 (4.00)	
51–59	74 (14.15)	569 (11.61)	
61–69	117 (22.37)	1234 (25.19)	
71–78	108 (20.65)	1270 (25.92)	
78+	181 (34.61)	1499 (30.60)	
Race			<.001
Asian	90 (17.21)	698 (14.25)	
Black or AA	25 (4.78)	286 (5.84)	
White	153 (29.25)	2348 (47.93)	
Hispanic or Latino	83 (15.90)	476 (9.71)	
AIAN	3 (.57)	18 (.37)	
NHPI	5 (.96)	52 (1.10)	
Other	106 (20.27)	590 (12.04)	
Unknown	58 (11.09)	429 (8.79)	
Comorbidity			
Hypertension	199 (38.05)	1425 (29.09)	<.001
DM	192 (36.71)	1151 (23.49)	<.001
Coronary artery disease	89 (17.02)	913 (18.64)	.364
Previous CABG	14 (2.68)	120 (2.45)	.750
Prosthetic valve	4 (.76)	81 (1.65)	.088
Peripheral vascular disease	40 (7.65)	228 (4.65)	.003
Heart failure	55 (10.52)	542 (11.06)	.704
Sleep apnea	35 (6.69)	297 (6.06)	.568
COPD	32 (6.12)	280 (5.72)	.707
ESRD	39 (7.46)	244 (4.98)	.016
Cirrhosis	10 (1.91)	158 (3.23)	.099
Hospital course			
ICU	324 (61.95)	2854 (58.26)	.103
Antiarrhythmic drug use	315 (60.23)	3066 (62.58)	.291
Pressor use	259 (49.52)	2356 (48.09)	.534
Mechanical ventilation	133 (25.43)	754 (15.39)	<.001
Outcome			
Death	137 (26.20)	500 (10.21)	<.001
Length of stay (d)	15 [7–30]	9 [5–18]	<.001

Values are given as n (%) or median [interquartile range] unless otherwise indicated.

AF = atrial fibrillation; other abbreviations as in Table 1.

diabetes (OR 2.02; $P < .001$), level of care in the intensive care unit (OR 3.79; $P < .001$), vasopressor use (OR 5.26; $P < .001$), and mechanical ventilation (OR 2.57; $P < .001$) (Table 3). Predictors for development of AF in COVID+ patients also predicted development of AF in non-COVID patients. In multivariate logistic regression, this difference in incident AF remained statistically significant (OR 0.709; $P < .001$).

COVID+ patients who developed incident AF had significantly longer length of stay compared to both non-COVID pneumonia patients who developed AF (median 15 vs 9 days; mean 22.45 vs 15.14 day; $P < .001$ by

Table 3 Univariate logistic regression analysis of predictors of incident AF in COVID+ patients

	OR (95% CI)	P value
Male	1.33 (1.25–1.42)	<.001
Comorbidity		
Hypertension	0.93 (0.77–1.12)	.435
DM	2.02 (1.88–2.17)	<.001
Coronary artery disease	1.04 (0.96–1.12)	.335
Previous CABG	1.08 (0.90–1.30)	.398
Prosthetic valve	0.97 (0.34–2.73)	.949
Peripheral vascular disease	1.11 (0.79–1.57)	.547
Heart failure	1.04 (0.77–1.40)	.792
Sleep apnea	0.72 (0.50–1.03)	.071
COPD	0.99 (0.70–1.45)	.965
ESRD	0.92 (0.65–1.30)	.636
Cirrhosis	0.56 (0.29–1.07)	.081
Thyroid disease	1.03 (0.76–1.40)	.864
Hospital course		
ICU	3.79 (3.14–4.58)	<.001
Pressor use	5.26 (4.35–6.37)	<.001
Mechanical ventilation	2.57 (2.06–3.19)	<.001

AF = atrial fibrillation; CI = confidence interval; OR = odds ratio; other abbreviations as in Table 1.

Mann-Whitney and t test) and COVID patients who did not develop AF (median 7 days; mean 12.19 days; $P < .001$ by Mann-Whitney and t test). Use of antiarrhythmic drugs for treatment of new-onset AF was similar in the 2 cohorts: 60.23% in COVID+ patients and 62.58% in non-COVID patients. Other characteristics of the patients who developed incident AF are listed in Table 2.

Laboratory values

Median values of troponin I, troponin T, brain natriuretic peptide (BNP), N-terminal pro-brain natriuretic peptide (NT-pro BNP), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) are listed in Table 4. Overall, the values were in the normal range with no difference between the 2 groups. More patients who developed AF had troponin data available compared to those who did not develop AF (41.8% vs 27.8%, respectively). There was no significant elevation in troponin I or T level in those with incident AF (Table 5).

Table 4 Comparison of median laboratory values for the 2 cohorts

	COVID+		Non-COVID		P value
	Median [interquartile range]	n	Median [interquartile range]	n	
Troponin I	.04 [.04-.04]	1451	.04 [.04-.12]	7106	.561
Troponin T	19 [8-49.9]	433	34.5 [14-104]	2720	.623
BNP	76.5 [37-196.5]	1500	197 [70-673]	6471	.666
NT-proBNP	330 [87-1417]	331	1009 [234-4917]	1603	.641
ESR	60 [37-82]	896	48 [22-86]	961	.441
CRP	9.9 [5-16.73]	1994	4.9 [1.18–13.8]	2635	.364

BNP = brain natriuretic peptide; COVID = coronavirus disease; CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; NT-proBNP = N-terminal pro-brain natriuretic peptide.

Table 5 Comparison of median laboratory values for patients who developed AF

	COVID+		Non-COVID		P value
	Median [interquartile range]	n	Median [interquartile range]	n	
Troponin I	.041 [.04-.17]	169	.05 [.04-.26]	1449	.0412
Troponin T	52.5 [18.75-145.25]	71	52 [25-149.1]	576	.508

AF = atrial fibrillation; COVID = coronavirus disease.

Mortality

The COVID+ cohort had significantly higher in-hospital mortality compared to the non-COVID cohort (8.22% vs 4.85%; $P < .001$) (Figure 3). Incident AF was associated with significantly higher in-hospital mortality in both cohorts (COVID+: OR 5.53; 95% CI 4.39–6.98; $P < .001$; non-COVID: OR 2.75; 95% CI 2.46–3.07; $P < .001$).

Discussion

In our analysis of 39,415 diverse subjects across the state of California, there was less incident AF in COVID+ subjects compared to the non-COVID pneumonia comparison group. Several surrogates of severe illness were associated with the development of AF, including vasopressor use, mechanical ventilation, admission to the intensive care unit, and longer length of stay. The lack of significant elevation in troponin T and I levels in either cohort among AF patients favors severity of illness and lung injury as an association with AF rather than myocardial injury. However, cardiac biomarkers were ordered for only a subset of patients, likely those with pre-existing cardiovascular disease, so these data are inherently biased.

Similar to previous studies, we demonstrated that patients with COVID-19 infection requiring hospitalization have high rates of cardiovascular comorbidities, are older, more frequently are male, and are disproportionately of non-White race.¹¹⁻¹³ Our findings of incident AF are largely consistent with the few existing studies that have measured rates of 13%–21% for historical or new AF and 5.4%–11.5% for new-onset AF in COVID-19 patients.^{14–18}

Similar rates of new-onset AF in patients with non-COVID ARDS have been reported.¹⁹

Few studies have sought to determine whether this high rate of AF is the result of direct cardiovascular involvement or simply is associated with severity of illness. One study demonstrated similar rates of incident AF in patients with COVID-19 compared to those with influenza, correlating to markers of inflammation and disease severity.¹⁶ To the best of our knowledge, our study is the first to compare COVID vs non-COVID pneumonia/ARDS patients in a large diverse population. Our findings are consistent with previous studies demonstrating increased mortality in COVID-19 patients who develop AF, and likely to be a marker of severe illness driving mortality.^{14,15}

Several hypotheses exist for the mechanism of AF in COVID-19, including systemic inflammation, myocardial injury, increased sympathetic tone, and oxidative stress.²⁰ Hypoxia and hypotension likely play a causal role in the development of the mechanistic triggers for the development of AF. The implications of incident AF during hospitalization for a severe COVID-19 infection are unknown, and long-term follow-up is needed to determine the risk of recurrent AF and cardioembolic stroke in these patients.

Unique strengths of this study include its extremely large and diverse sample size and comparison to a non-COVID pneumonia cohort. The CORDS database from which this study was derived includes all adult patients tested for COVID-19 across the entire UC Health system, which provides care to 1.8 million unique patients annually across California or approximately 4.6% of the state population.^{21,22}

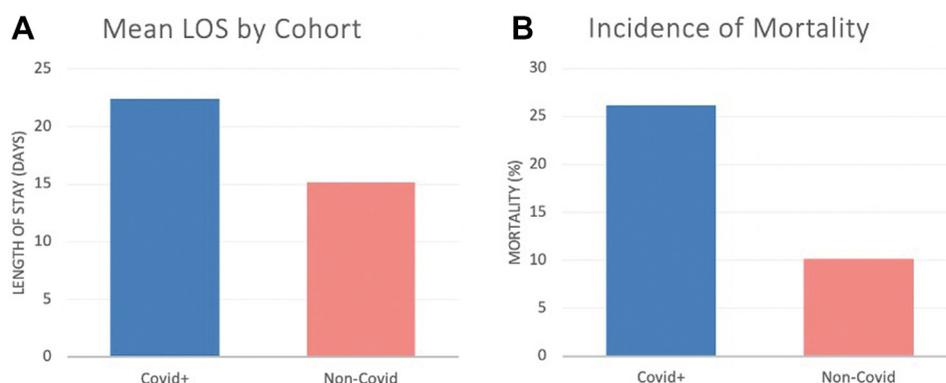


Figure 3 Comparison of outcomes of mortality (B) and length of stay (LOS) (A) in patients with coronavirus disease 2019 (COVID) pneumonia vs non-COVID pneumonia.

Study limitations

The large sample size allowed for a diverse and representative population; however, individual chart review and long-term follow-up beyond index hospitalization were not available. In addition, the hospital day on which subjects developed AF is not known, making hazard proportion analysis unavailable. The COVID+ cohort had significantly longer length of stay, which can be an indicator of more severe disease. However, this increased disease severity should increase the risk of development of AF and promote the null hypothesis that AF is not associated with COVID-19 infection. This study included only patients with COVID-19 infection severe enough to warrant inpatient hospital admission and cannot be extrapolated to individuals with mild infection who likely are younger and healthier.

Conclusion

Incident AF is lower in COVID compared to non-COVID hospitalized patients with pneumonia/ARDS. AF seems to be associated with severity of illness and higher mortality rather than myocardial injury, as indicated by normal troponin levels in our study cohort of diverse California patients.

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Patient Consent: These HIPAA-limited data were determined to be exempt from human subject protection and patient consent by UC Davis IRB under protocol 1879428-1.

Ethics Statement: The research reported in this paper adhered to Helsinki Declaration guidelines.

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