C-reactive protein and clinical outcomes in patients with COVID-19

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Background
A systemic inflammatory response is observed in coronavirus disease 2019 (COVID-19). Elevated serum levels of C-reactive protein (CRP), a marker of systemic inflammation, are associated with severe disease in bacterial or viral infections. We aimed to explore associations between CRP concentration at initial hospital presentation and clinical outcomes in patients with COVID-19.

Methods and results
Consecutive adults aged ≥18 years with COVID-19 admitted to a large New York healthcare system between 1 March and 8 April 2020 were identified. Patients with measurement of CRP were included. Venous thromboembolism (VTE), acute kidney injury (AKI), critical illness, and in-hospital mortality were determined for all patients. Among 2782 patients hospitalized with COVID-19, 2601 (93.5%) had a CRP measurement [median 108 mg/L, interquartile range (IQR) 53–169]. CRP concentrations above the median value were associated with VTE [8.3% vs. 3.4%; adjusted odds ratio (aOR) 2.33, 95% confidence interval (CI) 1.61–3.36], AKI (43.0% vs. 28.4%; aOR 2.11, 95% CI 1.76–2.52), critical illness (47.6% vs. 25.9%; aOR 2.83, 95% CI 2.37–3.37), and mortality (32.2% vs. 17.8%; aOR 2.59, 95% CI 2.11–3.18), compared with CRP below the median. A dose response was observed between CRP concentration and adverse outcomes. While the associations between CRP and adverse outcomes were consistent among patients with low and high D-dimer levels, patients with high D-dimer and high CRP have the greatest risk of adverse outcomes.

Conclusions
Systemic inflammation, as measured by CRP, is strongly associated with VTE, AKI, critical illness, and mortality in COVID-19. CRP-based approaches to risk stratification and treatment should be tested.
Keywords
C-reactive protein • Coronavirus • COVID-19 • Critical illness • Inflammation • Mortality

Introduction
The systemic inflammatory response to the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection is a hallmark of the 2019 coronavirus disease (COVID-19), and most hospitalized patients with COVID-19 have abnormal inflammatory biomarkers. C-reactive protein (CRP), an acute-phase protein first described by Tillet and Francis, is synthesized by the liver in response to interleukin-6 (IL-6) and is a widely available biomarker of inflammation. Elevated CRP concentrations are associated with cardiovascular disease and acute kidney injury (AKI) in surgical patients, with inflammatory rheumatic diseases such as rheumatoid arthritis and gout, and with incident venous thromboembolism (VTE) in community cohorts. C-reactive protein has also been associated with severe disease in patients with H1N1 influenza pneumonia, and a number of recent series have reported an association between higher CRP concentrations and greater disease severity in COVID-19. However, most studies were small, and evaluated neither a dose response nor heterogeneity across demographics. Moreover, the association between initial CRP concentration and VTE and AKI in COVID-19 is uncertain. The relationship between CRP concentrations and D-dimer, a fibrin degradation product that is associated with thrombosis in COVID-19, has not been explored. The aim of this study is to explore the associations between CRP concentrations at initial hospital presentation and clinical outcomes, including VTE and AKI, in patients with COVID-19 who were hospitalized at a large health-care system in New York.

Methods
Study participants and data collection
The study was approved by the New York University (NYU) Grossman School of Medicine Institutional Review Board and performed with a waiver of informed consent. We identified consecutive adults age ≥18 years with a nucleic acid amplification test positive for SARS-CoV-2 between 1 March 2020 and 8 April 2020 who were admitted to NYU Langone Health (NYULH), a multi-hospital health system in New York. At all inpatient facilities, CRP surveillance was standard of care for individuals with suspected or confirmed COVID-19 diagnoses because it was included in the electronic admission order sets during the pandemic. Elevated CRP concentrations (Siemens Dimension C-Reactive Protein, Siemens, Washington DC; Abbot Architect C-Reactive Protein, Chicago, IL) was defined as a measurement above the median value among patients hospitalized with COVID-19 at NYU. Patients were also divided into subgroups by quartile of initial CRP concentration. Demographics, comorbidities, outpatient medications, clinical presentations, and other laboratory data recorded for clinical purposes were abstracted from the electronic health record. Only the initial in-hospital measurements for each laboratory test were recorded. Relevant comorbidities, including hypertension, hyperlipidaemia, diabetes mellitus, heart failure, coronary artery disease, atrial fibrillation, cancer, and chronic kidney disease, were defined by International Classification of Diseases 10th Revision (ICD-10) codes.

Outcomes
Venous thromboembolism was defined by deep vein thrombosis or pulmonary embolism. Thrombotic events were identified from
radiology reports and clinical documentation using a natural-language processing tool (simpleNLP) with sensitivity and specificity >95%, ICD-10 diagnosis codes assigned during hospitalization, and chart review of echocardiogram reports, as described previously. All thrombotic events were confirmed by manual medical record review. Acute kidney injury was defined as an increase in serum creatinine by \( \geq 0.3 \) mg/dL within a 48 h period or a 50% increase in serum creatinine compared with the baseline value. Critical illness was defined by treatment in an intensive care unit, need for mechanical ventilation, transfer to a hospice, or in-hospital death. All-cause in-hospital mortality was determined for all patients. Follow-up was complete through 13 May 2020.

**Statistical analysis**

Categorical variables are reported as frequencies and percentages, and were compared by \( \chi^2 \) tests. Continuous variables are presented as mean (SD) and median (interquartile range (IQR)) and compared using t-tests or non-parametric Mann–Whitney test for all non-normally distributed data. Patients were categorized by the median CRP concentration, or the quartile of CRP concentration. Logistic regression models were generated to estimate the odds of the clinical endpoints, adjusted for demographics and clinical comorbidities. Covariates included in the multivariable models included age, sex, race/ethnicity, body mass index, tobacco use, hypertension, hyperlipidaemia, chronic kidney disease, coronary artery disease, heart failure, malignancy, and baseline laboratory values. Subgroup analyses were performed to evaluate the consistency of the study findings by age, sex, race, and the presence or absence of obesity [body mass index (BMI) \( \geq 30 \) kg/m\(^2\)]. To evaluate trends in CRP over time, CRP trajectory plots were generated and stratified by clinical outcomes. Differences in CRP values by group were compared by repeated measures analysis of variance (ANOVA). Since D-dimer is associated with thrombosis in patients with COVID-19, subgroup analyses were also performed to evaluate relationships between CRP and outcomes by D-dimer level (above vs. below the median value of 384 ng/mL). Finally, we investigated the relationship between IL-6 and outcomes in patients with both CRP and IL-6 measured at hospital presentation. Statistical analyses were performed using R (R Foundation for Statistical Computing, Vienna, Austria). Statistical tests are two-sided, and \( P \)-values <0.05 were considered to be statistically significant.

**Results**

**Patient characteristics**

A total of 2782 consecutive adults with COVID-19 were admitted to NYULH between 1 March and 8 April 2020, and 2601 (93.5%) had a measurement of CRP. The median initial CRP concentration was 108 mg/L (IQR 53–169) (Figure 1). Only 58 patients (2.2%) had an initial normal CRP <5 mg/L (the upper reference limit for the assay). Clinical characteristics of patients stratified by quartile of initial CRP concentration are shown in Table 1. Patients with CRP concentrations in the highest quartiles were more likely to be men (71.3% in the highest CRP quartile vs. 52.6% in the lowest CRP quartile, \( P < 0.001 \)) and of Hispanic ethnicity (30.4% in the highest quartile vs. 20.5% in the lowest quartile, \( P < 0.001 \)) than patients with lower CRP concentrations. Fewer patients with the highest CRP values had a history of heart failure (9.6% in the highest quartile vs. 16.4% in the lowest quartile, \( P = 0.0004 \)), coronary artery disease (18.5% vs. 27.5%, \( P = 0.0002 \)), or chronic kidney disease (16.8% vs. 23.9%, \( P = 0.0019 \)). Patients with the highest CRP concentrations were also less likely to have been prescribed statin therapy (11.6% in the highest quartile vs. 17.4% in the lowest quartile, \( P = 0.0035 \)) or a beta-blocker (10% vs. 15.3%, \( P = 0.005 \)) prior to hospital admission. Elevated CRP concentrations at presentation were associated with higher temperatures and lower oxygen saturation at presentation, as well as higher initial white blood cell and platelet counts, higher initial D-dimer levels, and higher initial ferritin concentrations (Figure 1). Clinical characteristics of patients stratified by median initial CRP concentration are shown in Supplementary material online Table S1. In a sensitivity analysis of patients who were admitted and discharged alive with a hospital length of stay \( \leq 1 \) day, the median initial CRP was 45.9 mg/L (IQR 21.8–107.4).

**Clinical outcomes**

Initial CRP concentrations were associated with clinical outcomes in patients with COVID-19. An initial CRP value above the median measurement was associated with VTE [8.3% vs. 3.4%; adjusted odds ratio (aOR) 2.33, 95% confidence interval (CI) 1.61–3.36], AKI (43.0% vs. 28.4%; aOR 2.11, 95% CI 1.76–2.52), critical illness (47.6%...
vs. 25.9%; aOR 2.83, 95% CI 2.37–3.37), and in-hospital mortality (32.2% vs. 17.8%; aOR 2.59, 95% CI 2.11–3.18) compared with patients with an initial CRP value below the median (Figure 2). Patients with the highest quartiles of CRP measured had the greatest likelihood of VTE, AKI, critical illness, and mortality (Figure 2). Associations between CRP concentration and adverse outcomes were consistent in subgroups defined by age, sex, race, and obesity (Figure 3).

Levels of D-dimer increased concordantly with higher quartiles of initial CRP concentration (Table 1). Associations between CRP concentration and VTE, AKI, critical illness, and mortality were consistent in patients with low and high D-dimer (data not shown). In a subgroup analyses by D-dimer level, patients with low values of both CRP and D-dimer were at low risk for in-hospital adverse events. In contrast, the incidences of VTE (12.3% vs. 2.8%, P < 0.001), AKI (52.1% vs. 23.7%, P < 0.001), critical illness (58.2% vs. 20.2%, P <

### Table 1  Characteristics of patients with COVID-19 based on the quartile of initial CRP

| Quartile 1  | Quartile 2  | Quartile 3  | Quartile 4  | P-value |
|------------|------------|------------|------------|---------|
| (<53 mg/L) | (>53 to ≤108 mg/L) | (>108 to ≤169 mg/L) | (>169 mg/L) |         |
| (n = 648)  | (n = 655)   | (n = 650)  | (n = 648)  |         |
| Age, years; median (IQR) | 64 (51–75) | 64 (51–75) | 64 (53–74) | 62 (52–72) | 0.451   |
| Male sex | 341 (52.62%) | 389 (59.39%) | 432 (66.46%) | 462 (71.30%) | <0.001  |
| Race/ethnicity |         |         |         |         | 0.036   |
| Non-Hispanic white | 278 (42.90%) | 247 (37.71%) | 260 (40.00%) | 252 (38.89%) |         |
| African American | 107 (16.51%) | 94 (14.35%) | 87 (13.38%) | 85 (13.12%) |         |
| Hispanic | 133 (20.52%) | 181 (27.63%) | 179 (27.54%) | 197 (30.40%) |         |
| Asian | 50 (7.72%) | 50 (7.63%) | 48 (7.38%) | 31 (4.78%) |         |
| Other/multiracial | 58 (8.95%) | 59 (9.01%) | 49 (7.54%) | 55 (8.49%) |         |
| Unknown | 22 (3.40%) | 24 (3.66%) | 27 (4.15%) | 28 (4.32%) |         |
| Tobacco use |         |         |         |         | <0.001  |
| Current | 56 (8.64%) | 32 (4.89%) | 23 (3.54%) | 30 (4.63%) |         |
| Former | 140 (21.60%) | 128 (19.54%) | 133 (20.46%) | 131 (20.22%) |         |
| Never | 452 (69.75%) | 495 (75.57%) | 494 (76.00%) | 487 (75.15%) |         |
| BMI, kg/m², mean (SD) | 28.5 (25.1–32.9) | 29.0 (25.5–34.0) | 29.3 (25.8–34.5) | 28.98 (25.5–33.0) | 0.011   |
| Clinical comorbidities |         |         |         |         |         |
| Hypertension | 412 (63.58%) | 413 (63.05%) | 425 (65.38%) | 374 (57.72%) | 0.029   |
| Hyperlipidaemia | 284 (43.83%) | 286 (43.66%) | 264 (40.62%) | 284 (43.83%) | 0.575   |
| Diabetes mellitus | 245 (37.81%) | 254 (38.78%) | 272 (41.85%) | 229 (35.34%) | 0.114   |
| Heart failure | 106 (16.36%) | 85 (12.98%) | 85 (13.08%) | 62 (9.57%) | 0.004   |
| Coronary artery disease | 178 (27.47%) | 146 (22.29%) | 133 (20.46%) | 120 (18.52%) | 0.001   |
| Atrial fibrillation | 57 (8.91%) | 70 (10.75%) | 48 (7.41%) | 50 (7.78%) | 0.137   |
| Cancer | 70 (10.80%) | 80 (12.12%) | 72 (11.08%) | 61 (9.41%) | 0.446   |
| Chronic kidney disease | 155 (23.92%) | 143 (21.83%) | 145 (22.31%) | 109 (16.82%) | 0.012   |
| Medications at baseline |         |         |         |         |         |
| Statin | 113 (17.44%) | 85 (12.98%) | 90 (13.85%) | 75 (11.57%) | 0.017   |
| Beta-blocker | 98 (15.31%) | 90 (13.82%) | 71 (10.96%) | 64 (9.95%) | 0.012   |
| ACE-I or ARB | 107 (16.72%) | 114 (17.51%) | 115 (17.75%) | 88 (13.69%) | 0.176   |
| Anticoagulation | 52 (8.12%) | 61 (9.37%) | 54 (8.33%) | 53 (8.24%) | 0.844   |
| Clinical characteristics at presentation |         |         |         |         |         |
| Temperature at presentation (°C) | 37.3 (36.8–38.1) | 37.4 (36.9–38.2) | 37.5 (37–38.2) | 37.5 (37–38.3) | 0.001   |
| Oxygen saturation (%) | 96 (94–98) | 94 (91–96) | 93 (89–95) | 91 (86–95) | <0.001  |
| Initial laboratory markers [median (IQR)] |         |         |         |         |         |
| First creatinine, mg/dL | 1.0 (0.8–1.4) | 1.0 (0.8–1.3) | 1.0 (0.8–1.4) | 1.0 (0.8–1.4) | 0.694   |
| First WBC | 2.0 (1.0–7.0) | 3.0 (1.0–8.5) | 4.0 (2.0–12.3) | 5.0 (2.0–12.5) | <0.001  |
| First CRP | 27.5 (13.4–40.9) | 80.6 (67.5–94.0) | 136.0 (123.0–151.0) | 218.0 (191.1–267.9) | <0.001  |
| First haemoglobin, g/dL | 13.2 (11.8–14.4) | 13.2 (11.9–14.4) | 13.3 (12–14.4) | 13.3 (12–14.3) | 0.94    |
| First platelet | 180 (142–230) | 189 (152–244.5) | 200 (160–255) | 229 (175–285) | <0.001  |
| First D-dimer | 301 (197.96–563) | 335 (213–630) | 404 (261–740) | 484 (296–963) | <0.001  |
| First ferritin | 379 (167–730.6) | 658 (355.4–1310.2) | 811 (433.5–1646) | 1173.6 (637.35–1903.5) | <0.001  |
| First lymphocyte | 1 (0.7–1.4) | 0.8 (0.6–1.2) | 0.8 (0.6–1.1) | 0.8 (0.5–1.1) | <0.001  |

ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; WBC, white blood cell count.
0.001), and mortality (39.8% vs. 13.0%, P < 0.001) were substantially higher in patients with concomitant elevations in CRP and D-dimer compared with those with low CRP and D-dimer values (Figure 4A). While both CRP and D-dimer increased the adjusted odds of an adverse outcome, patients with elevation of both CRP and D-dimer concentrations had the greatest risk of adverse events (Figure 4B).

Among 2601 adults with COVID-19 and CRP concentration measured at presentation, 2224 (85.5%) had serial CRP measurements, with a median of 5 (IQR 3–10) CRP measurements reported during hospital admission. The median peak CRP concentration was 164 mg/L (91.09–251.5), measured on median hospital day 2 (IQR 0–5). Higher peak CRP concentrations were reported among patients with fatal COVID-19 (248.1 mg/L, IQR 169.0–395.0 vs. 141.0 mg/L, IQR 74.7–216.8 in patients who survived, P < 0.001). Levels of CRP were significantly higher over time in patients who developed AKI, critical illness, or who died during hospitalization. Levels of CRP over time are shown in patients with and without fatal disease, critical illness, VTE, and AKI (Figure 5A–D).

Since IL-6 is upstream of CRP in the inflammatory cascade, we investigated the relationship between IL-6 and outcomes in 1693 patients with COVID-19 who had CRP and IL-6 measured at hospital presentation. Elevated initial IL-6 and CRP concentrations in COVID-19 were each associated with increased odds of mortality [IL-6 above the median concentration (>13 pg/mL), aOR 2.29, 95% CI 1.75–2.98; CRP above the median, aOR 1.93, 95% CI 1.50–2.49] and critical illness (IL-6, aOR 3.05, 95% CI 2.43–3.84; CRP, aOR 1.98, 95% CI 1.57–2.49) after adjustment for clinical covariates. When stratified by initial CRP concentration, an initial IL-6 above the median value (>13 pg/mL) was associated with critical illness and mortality among patients with CRP above (aOR for critical illness, 3.04, 95% CI 2.27–4.06; aOR for mortality, 1.91, 95% CI 1.39–2.62) and below (aOR for critical illness, 3.16, 95% CI 2.15–4.64; aOR for mortality, 2.51, 95% CI 1.58–4.00) the median concentration. These data suggest that IL-6 may provide additional prognostic information in patients hospitalized with COVID-19.

Discussion
In an analysis of COVID-19 patients hospitalized in a large New York health system, nearly all patients had evidence of a systemic inflammatory response to SARS-CoV-2 infection with a median CRP concentration of 108 mg/L, a value nearly 40-fold higher than the laboratory upper limit of normal. Patients with elevated CRP concentrations above the median value at the time of initial presentation were more likely to have VTE, AKI, critical illness, and in-hospital mortality during hospitalization.
Figure 3: Associations between CRP and all-cause mortality, critical illness, venous thromboembolism, and acute kidney injury in subgroups by age, sex, race, and body mass index. Odds ratios adjusted for age, sex, race/ethnicity, body mass index, tobacco use, hypertension, hyperlipidaemia, chronic kidney disease, atrial fibrillation, coronary artery disease, heart failure, malignancy, initial ferritin, absolute lymphocyte count, D-dimer, and baseline use of statins, beta-blockers, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, and anticoagulants.
Figure 4. Associations between CRP and all-cause mortality, critical illness, venous thrombo-embolism, and acute kidney injury stratified by initial D-dimer measurement. The incidence (A) and adjusted odds (B) of adverse outcomes are shown. (low CRP <108 mg/dL; high CRP ≥108 mg/dL; low D-dimer ≤384 ng/mL; high D-dimer >384 ng/mL). (A) *p for trend <0.001 for all outcomes. (B) Odds ratios adjusted for age, sex, race/ethnicity, body mass index, tobacco use, hypertension, hyperlipidaemia, chronic kidney disease, atrial fibrillation, coronary artery disease, heart failure, malignancy, initial ferritin, absolute lymphocyte count, baseline use of statins, beta-blockers, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, and anticoagulants.
the subsequent hospital stay than those with lower initial measurements, and patients with the highest CRP values had the worst clinical outcomes.

C-reactive protein is well established as a marker of systemic inflammation and severe infection. As an acute-phase reactant, CRP binds to phosphocholine in pathogens and membranes of host cells, and acts as an opsonin to enhance phagocytosis and facilitate clearance. Ligand-bound CRP also efficiently activates the classical pathway of the complement system, an important component of innate host defence.14 Prior to the COVID-19 global pandemic, up to 90% of all marked elevations in CRP concentration were attributed to an infectious aetiology, most often from bacterial pathogens.15,16 Elevated CRP concentrations have also been reported in severe viral infections, including H1N1 influenza pneumonia, and now in SARS-CoV-2 infection.6–9 In a prior study of 298 patients with COVID-19, patients who died had an initial CRP that was 10-fold higher than that of survivors (100.0 vs. 9.7 mg/L, \( P < 0.001 \)), and CRP concentrations were associated with mortality, with an area under the receiver operating characteristic curve (AUC) of 0.896.9 Recent reports also identified associations between CRP concentrations and respiratory failure requiring mechanical ventilation, with a nearly five-fold greater risk of acute respiratory distress syndrome (ARDS) reported in patients with high-sensitivity CRP >5 mg/L compared with those with lower CRP values.12,17 CRP is associated with extra-pulmonary disease in COVID-19, and correlations between CRP concentrations and myocardial injury have been reported in multiple series.18–21 In contrast, prior studies have not reported on the relationship between CRP and AKI and VTE in COVID-19, nor on serial measures of CRP over time.

Other inflammatory markers are also associated with adverse outcomes in patients with high and low initial CRP concentrations. In a separate analysis of 1400 patients hospitalized with COVID-19, IL-6 and tumour necrosis factor (TNF)-\( \alpha \) cytokine levels at the time of hospitalization were associated with survival after adjustment for clinical comorbidities and CRP, D-dimer, and ferritin concentrations.22 These data confirm the observed relationships between markers of inflammation and adverse clinical outcomes in COVID-19. Ultimately, CRP may be preferred as a biomarker since it is inexpensive and widely available at most medical centres, facilitating rapid implementation of routine biomarker measurement into clinical care of patients with COVID-19.

In contrast to CRP measurement for cardiovascular risk stratification, in which inflammation may contribute to accelerated atherosclerosis and instability of atherosclerotic plaque, CRP concentrations in COVID-19 infection reflect disease severity and the magnitude of the acute inflammatory response. The role of systemic inflammation in the pathogenesis of COVID-19 remains incompletely understood, and causal relationships between inflammation measured by CRP and adverse clinical outcomes are speculative. However, the detrimental inflammatory response observed in some individuals with COVID-19 parallels secondary haemophagocytic lymphohistiocytosis (also known as macrophage activation syndrome), and may independently contribute to multiorgan damage in COVID-19.23,24 Earlier studies of patients with ARDS prior to COVID-19 demonstrated that glucocorticoid administration reduces CRP concentrations.25 In a recent randomized trial of dexamethasone immunosuppression in patients with severe COVID-19, steroid therapy reduced the incidence of death among those who required supplemental oxygen and in critically ill patients requiring invasive mechanical ventilation. The clinical benefit of immunosuppression further supports the hypothesis that inflammation in response to viral

![Figure 5](https://example.com/figure5.png)  
*Figure 5* Trajectory of CRP levels over time among patients with and without fatal disease (A), critical illness (B), VTE (C), and AKI (D).
infection contributes to poor outcomes in COVID-19.24 Thrombo-inflammation has been proposed as a mediator of adverse events in COVID-19, with dysregulation of the normal antithrombotic function of the endothelium in the response to inflammatory stress, leading to leucocyte recruitment, complement and platelet activation, and enhanced coagulation in the microvasculature.27,28 This is supported by autopsy studies of decedents with COVID-19, in which platelets with fibrin microthrombi were identified within the pulmonary, renal, hepatic, and cardiac microcirculation.29 Thrombotic events, particularly pulmonary embolism, are common in COVID-19 and are associated with systemic inflammation.13,30 In the present analysis, CRP and D-dimer were each independently associated with adverse events. However, patients were at highest risk when they presented with concomitant elevations in CRP and D-dimer concentrations, providing additional support for the synergistic role of inflammation and thrombosis in the pathogenesis of disease associated with SARS-CoV-2 infection. Given the role of micro- and macrothrombosis in disease pathogenesis, an NIH-funded multicentre, adaptive, randomized clinical trial [Accelerating COVID-19 Therapeutic Interventions and Vaccines (ACTIV)-4] is ongoing to determine optimal antithrombotic dosing for patients hospitalized with COVID-19 (NCT04505774).

Limitations of this study include its retrospective observational study design. The first measured CRP concentration at hospital admission was used for the primary analyses. Patients were hospitalized at a single healthcare system in New York during a 6-week period of rapid viral spread, although a diverse cohort of COVID-19 patients across four inpatient sites is represented. The time period studied preceded the finding that immunosuppression associated with systemic inflammation. As measured by CRP, is strongly associated with VTE, AKI, critical illness, and in-hospital mortality in patients with COVID-19. Inflammatory biomarker-based approaches to risk stratification and treatment should be evaluated to improve outcomes of patients with SARS-CoV-2 infection.

Conclusions

Systemic inflammation, as measured by CRP, is strongly associated with VTE, AKI, critical illness, and in-hospital mortality in patients with COVID-19. Inflammatory biomarker-based approaches to risk stratification and treatment should be evaluated to improve outcomes of patients with SARS-CoV-2 infection.

Supplementary material

Supplementary material is available at European Heart Journal online.

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