Pulmonary Embolism in Hospitalized Patients with COVID-19: A Multicenter Study

Sadjad Riyahi, MD • Hreedi Dev • Ashkan Behzadi, MD • Jinhye Kim, MD • Hanieh Attari, MD • Syed I. Raza, MD • Daniel J. Margolis, MD • Ari Jonisch, MD • Ayah Megahed, MD • Anas Bamashmos, MD • Kareem Elfatairy, MD • Martin R. Prince, MD, PhD

From the Departments of Radiology of Weill Cornell Medicine, 416 E 55th St, New York, NY 10022 (S.R., H.D., J.K., H.A., S.I.R., D.J.M., A.J., M.R.P.); Bridgeport Hospital, Yale New Haven Health System, Bridgeport, Conn (A. Behzadi, A.M., A. Bamashmos, K.E.); and Columbia College of Physicians and Surgeons, New York, NY (M.R.P.). Received March 22, 2021; revision requested April 15; revision received June 18; accepted June 25. Address correspondence to M.R.P. (e-mail: map2008@med.cornell.edu).

Supported by New York-Presbyterian Hospital and Weill Cornell Medical College, including the Clinical and Translational Science Center (grant UL1 TR000457) and Joint Clinical Trials Office.

Conflicts of interest are listed at the end of this article.

See also the editorial by Ketai in this issue.

RadioLOGY 2021; 301:E426–E433 • https://doi.org/10.1148/radiol.2021210777 • Content code: CH

Background: Pulmonary embolism (PE) commonly complicates SARS-CoV-2 infection, but incidence and mortality reported in single-center studies, along with risk factors, vary.

Purpose: To determine the incidence of PE in patients with COVID-19 and its associations with clinical and laboratory parameters.

Materials and Methods: In this HIPAA-compliant study, electronic medical records were searched retrospectively for demographic, clinical, and laboratory data and outcomes among patients with COVID-19 admitted at four hospitals from March through June 2020. PE found at CT pulmonary angiography and perfusion scintigraphy was correlated with clinical and laboratory parameters. The d-dimer level was used to predict PE, and the obtained threshold was externally validated among 85 hospitalized patients with COVID-19 at a fifth hospital. The association between right-sided heart strain and embolic burden was evaluated in patients with PE undergoing echocardiography.

Results: A total of 413 patients with COVID-19 (mean age, 60 years ± 16 [standard deviation]; age range, 20–98 years; 230 men) were evaluated. PE was diagnosed in 102 (25%; 95% CI: 21, 29) of 413 hospitalized patients with COVID-19 who underwent CT pulmonary angiography or perfusion scintigraphy. PE was observed in 21 (29%; 95% CI: 19, 41) of 73 patients in the intensive care unit (ICU) versus 81 (24%; 95% CI: 20, 29) of 340 patients who were not in the ICU (P = .37). PE was associated with male sex (odds ratio [OR], 1.74; 95% CI: 1.1, 2.8; P = .02); smoking (OR, 1.86; 95% CI: 1.0, 3.4; P = .04); and increased d-dimer (P < .001), lactate dehydrogenase (P < .001), ferritin (P = .001), and interleukin-6 (P = .02) levels. Mortality in hospitalized patients was similar between patients with PE and those without PE (14% [13 of 102] vs 13% [40 of 311]; 95% CI: 9, 17; P = .98), suggesting that diagnosis and treatment of PE were not associated with excess mortality. The d-dimer levels greater than 1600 ng/mL [8.761 nmol/L] helped predict PE with 100% sensitivity and 62% specificity in an external validation cohort. Embolic burden was higher in patients with right-sided heart strain among the patients with PE undergoing echocardiography (P = .03).

Conclusion: Pulmonary embolism (PE) incidence was 25% in patients hospitalized with COVID-19 suspected of having PE. A d-dimer level greater than 1600 ng/mL [8.761 nmol/L] was sensitive for identification of patients who needed CT pulmonary angiography.

©RSNA, 2021

Online supplemental material is available for this article.

SARS-CoV-2 binds the angiotensin-converting enzyme-2 receptors on endothelial cells, especially within the kidneys, heart, lungs, and liver (1). Endothelial cell damage leads to thrombosis, which can be a defense mechanism that compartmentalizes infection and prevents further dissemination (2). However, with COVID-19, widespread thrombosis has been reported to cause numerous thrombotic complications, including deep vein thrombosis, pulmonary embolism (PE), myocardial infarction, stroke, and disseminated intravascular coagulation (3,4).

Grillet et al (5) reported a 23% positive rate for CT pulmonary angiography studies. In New York, Kaminetzky et al (6) reported that when CT pulmonary angiography was performed, it was positive for PE in 37% of patients with COVID-19 compared with 14.5% of patients before the pandemic. A meta-analysis of 4382 patients hospitalized with COVID-19 showed a 17.6% incidence of PE, with a substantially higher rate among patients with severe (ie, admitted to the intensive care unit [ICU]) versus general (ie, not admitted to the ICU) disease (21.7% vs 12.5%) (7). Thus, the incidence of PE varies widely in the literature, and uncertainty remains about who should be imaged. A high prevalence of right-sided heart strain has also been noted (8), suggesting that PE may be more fatal in patients with COVID-19; however, the risk factors for PE in COVID-19 are not well established.

The purpose of this study was to determine the multicenter incidence of PE in COVID-19 and its associations with COVID-19 suspected of having PE. A d-dimer level greater than 1600 ng/mL [8.761 nmol/L] helped predict PE with 100% sensitivity and 62% specificity in an external validation cohort. Embolic burden was higher in patients with right-sided heart strain among the patients with PE undergoing echocardiography (P = .03).

This copy is for personal use only. To order printed copies, contact reprints@rsna.org
with clinical and laboratory parameters. Secondary objectives were to assess the predictive value of D-dimer level and the relationship between right-sided heart strain and clot burden. We evaluated patients with COVID-19 admitted to four hospitals and developed a predictive model to identify patients with COVID-19 at high risk for PE. The model was validated with data from another institution.

Materials and Methods

Patients
The institutional review boards of Weill Cornell Medicine and Bridgeport Hospital approved this retrospective, Health Insurance Portability and Accountability Act–compliant review of existing medical records and waived the requirement for informed consent. This project used data extracted from the electronic medical records of four large New York-Presbyterian hospitals for all admitted patients who met the following inclusion criteria: age of at least 18 years, COVID-19 diagnosis confirmed with a positive result on a SARS-CoV-2 reverse transcription polymerase chain reaction (RT-PCR) assay (obtained via nasopharyngeal swab), and chest CT pulmonary angiography or perfusion scintigraphy performed after admission for COVID-19. Patients with PE before the first RT-PCR test were excluded. Data were extracted primarily by 4th-year medical students and confirmed by research fellows for COVID-19 admissions from March 3 to June 5, 2020. For external validation, data were extracted manually according to the same criteria for an additional cohort of patients with COVID-19 hospitalized and undergoing CT pulmonary angiography for suspected PE at Bridgeport Hospital in Connecticut (hereafter called the external validation cohort).

This patient population overlaps somewhat with a patient population described in a published letter: In that report, 41 patients with PE from the same COVID-19 database were evaluated for risk and D-dimer cutoff of venous thromboembolism (VTE) (9).
Figure 1: Patient flowchart. CTPA = CT pulmonary angiography, PE = pulmonary embolism, RT-PCR = reverse transcription polymerase chain reaction.

(IQR). The Student t test or Mann-Whitney U test was used to compare differences between groups for these variables. Categorical variables are reported as frequency and percentage, and the significance of between-group comparisons was assessed by using the χ² test.

We used a subsample of 408 patients who underwent chest CT pulmonary angiography to build a random forest model for predicting acute PE. We did not include the five patients with perfusion scintigraphy in this analysis to maintain consistency in the method of outcome detection. Missing values were imputed in variables with less than 10% missing data points by using the missForest method of imputation. The total study sample was randomly divided into a training set that maintained the outcome proportion, including two-thirds of the patients, and a test set (ie, the remaining one-third) for model building and internal validation, respectively. Because acute PE was a rare event, the synthetic minority oversampling technique, or SMOTE, was applied in the training set to balance the data by upsampling the minority group. A random forest model was fitted on the balanced training set to predict acute PE. The independent variables were age; sex; body mass index; smoking history; history of chronic lung disease, cardiovascular disease, and VTE or hypercoagulability; interval from COVID-19 symptoms to CT pulmonary angiography (in days); duration of hospital stay (in days); thromboprophylaxis at least 48 hours before CT pulmonary angiography; number of days on a ventilator; intubation; need for supplemental oxygen at arrival; noninvasive ventilation; heart rate; systolic blood pressure; percentage of oxygen saturation; D-dimer, C-reactive protein, fibrinogen, and ferritin levels; platelet count; and PT and activated partial thromboplastin time. Variable importance was determined by using permutation importance (ie, mean decrease in accuracy). A 10 × 10 cross-validation technique was used to train the model on the training set. Predictions were then made by using the random forest model on the test set. The area under the receiver operating characteristic curve was calculated to assess the model performance on the validation set. The Youden index was then used to establish a threshold for the predictions for diagnosis of acute PE.

Univariate logistic regression was used to assess the predictive performance of D-dimer level as a continuous variable. Subsequently, sensitivity analysis was performed by using the Youden J index to determine the optimal cutoff point in D-dimer levels for PE prediction by using D-dimer level alone. This cutoff was then externally validated by using the external validation cohort described earlier.

The significance level was set to .05, and statistical analysis was performed with R software, version 4.0.2 (R Core Team).

Results

From March 3 to June 5, 2020, 25,335 SARS-CoV-2 RT-PCR tests at four hospitals served to identify 8,460 patients with...
positive results; of these patients, 413 were admitted (Fig 1).
The research data repository had detailed information, including laboratory data and vital signs data within 72 hours of the ordering of imaging were collected. CT re-

| Variable                        | PE Present (n = 102) | PE Absent (n = 311) | P Value |
|---------------------------------|----------------------|---------------------|---------|
| Platelet count (cells/μL)       | 308 ± 163            | 305 ± 1744          | .86     |
| Partial thromboplastin time (sec)* | 32.4 (29.5–36.7)     | 32.5 (29.5–36.4)    | .86†    |
| Prothrombin time (sec)*         | 14.6 (13.3–15.9)     | 14 (13–15.3)        | .07†    |
| Fibrinogen level (mg/dL)        | 607.9 ± 273          | 569 ± 218           | .26     |
| Alanine aminotransferase level (U/L)* | 45 (22–65)         | 31 (17–60.5)        | .06†    |
| Albumin level (g/dL)*           | 3.1 (2.3–3.6)        | 3 (2.4–3.6)         | .68†    |
| Aspartate aminotransferase level (U/L)* | 37 (24–58)          | 33 (22–55)          | .23†    |
| Lactate dehydrogenase level (U/L) | 552 ± 475           | 410 ± 249           | <.001   |
| Creatinine level (mg/dL)        | 1.06 ± 0.8           | 1.02 ± 1.21         | .78     |
| Lymphocytes (absolute) (×10³/μL)* | 1.09 (0.7–1.64)     | 1.1 (0.64–1.55)     | .70†    |
| Creatine kinase level (U/L)*    | 93 (53–191)          | 73 (39–181)         | .06†    |
| Ferritin level (ng/mL)*         | 809 (490–1434)       | 595 (300–1167)      | .001†   |
| B-type natriuretic protein level (pg/mL)* | 62 (14–395)      | 60 (20–196)         | .76†    |
| Troponin I level (ng/mL)*       | 0.04 (0.03–0.25)     | 0.03 (0.03–0.07)    | .71     |
| Erythrocyte sedimentation rate (mm/h) | 78.5 ± 37.9           | 75.9 ± 37           | .54     |
| C-reactive protein level (mg/dL)* | 22.4 (8.2–131.8)    | 16.1 (4.4–92)       | .17     |
| D-dimer level (ng/mL)*          | 4262 (1839–13246)    | 1200 (496–3410)     | <.001†  |
| Interleukin-6 level (pg/mL)*    | 42 (13.3–93.5)       | 13 (5.5–40.5)       | .02     |

Note.—Unless otherwise noted, data are mean ± standard deviation. To convert alanine aminotransferase level to SI units (μkat/L), multiply by 0.0167. To convert aspartate aminotransferase level to SI units (μkat/L), multiply by 0.0167. To convert lactate dehydrogenase level to SI units (nmol/L), multiply by 5.476. PE = pulmonary embolism.

* Data are the median, with the interquartile range in parentheses.
† Mann-Whitney U test was used because of nonnormal distribution.

Bivariate Analysis in Patients Undergoing CT Pulmonary Angiography
Comparison of patients with PE-positive (n = 102) and PE-negative (n = 311) findings showed that men and smokers had 74% (odds ratio, 1.74; 95% CI: 1.1, 2.8; P = .02) and 86% (odds ratio, 1.86; 95% CI: 1.01, 3.4; P = .04) (Table 1) greater odds of having acute PE. Levels of alanine aminotransferase, lactate dehydrogenase, ferritin, D-dimer, and interleukin-6; heart rate; respiratory rate; and diastolic blood pressure were significantly higher in the PE group, whereas percentage of oxygen saturation was significantly lower (P < .05) (Tables 1, 2). Patients with PE-positive findings had a shorter interval between their first RT-PCR assay and imaging compared with patients with findings negative for PE (median, 1 day [IQR, 0–10 days] vs 3 days [IQR, 0–16 days]; P = .01). Incidence of acute deep vein thrombosis was higher in patients with PE-positive findings than in those with PE-negative findings (22 of 102 [22%] vs 22 of 311 [7%]; P < .001) (Table 3). There was a trend toward significantly higher PT and creatine kinase levels among those with PE.
Right-sided Heart Strain and Embolic Burden

Of 102 patients with findings positive for PE, 30% (n = 31) underwent echocardiography within 24 hours after PE diagnosis. Right-sided heart strain was present in eight of the 31 (26%). Mann-Whitney U test showed higher embolic burden for patients with right-sided heart strain (median Qanadli score, 11 [IQR, 8.3–13.3] vs 6 [IQR, 2–7.5]; P = .03) among patients with findings positive for PE (Figs 2, 3).

D-Dimer Level

Figure 4 shows the relative importance of the predictor variables calculated by mean decrease in accuracy in the final random forest model, trained on the training set. In general, the greater the mean decrease in accuracy when the variable was omitted from the model, the more important the variable. The d-dimer level was the dominant predictor in this model. The results of the sensitivity analysis of the final random forest model on the test set are presented in Table E2 (online).

Subsequently, we analyzed the utility of d-dimer level alone in the prediction of PE in 383 patients without missing d-dimer measurement (PE-positive, 98 patients; PE-negative, 285 patients; d-dimer measurement was missing in 30 patients [7%]). The univariable logistic regression model showed an area under the receiver operating characteristic curve of 0.75 (95% CI: 0.69, 0.80), a sensitivity of 83% (95% CI: 73, 89 [true-positive rate, 80 of 98]), a specificity of 56% (95% CI: 51, 62 [true-negative rate, 161 of 285]), and accuracy of 63% (95% CI: 58, 68), with d-dimer level used as a continuous variable. The optimal cutoff point for d-dimer was 1600 ng/mL (8.761 nmol/L), with the Youden index used in the entire internal sample.

Application of the d-dimer cutoff to the external validation cohort showed a sensitivity of 100% (95% CI: 89, 100 [true-positive rate, 32 of 32]), a specificity of 62% (95% CI: 48, 73 [true-negative rate, 20 of 53]), and an accuracy of 76% (95% CI: 66, 85) for diagnosis of PE.

Table 3: Clinical Outcomes for 413 Hospitalized Patients with COVID-19 Evaluated for Pulmonary Embolism with Chest CT (n = 408) or Perfusion Scintigraphy (n = 5)

| Variable                     | PE Present (n = 102) | PE Absent (n = 311) | P Value |
|-----------------------------|----------------------|---------------------|---------|
| Duration of hospital stay (d)* | 11 (5–25)            | 12 (4–35)           | .72     |
| Interval from RT-PCR assay to imaging (d)** | 1 (0–10)            | 3 (0–16)           | .01     |
| Intensive care unit         | 21 (21)              | 52 (17)             | .37     |
| Intubation                  | 25 (25)              | 71 (23)             | .73     |
| Death                       | 13 (14)              | 40 (13)             | .98     |
| Acute renal failure         | 24 (24)              | 59 (19)             | .26     |
| Acute respiratory distress syndrome | 26 (25)          | 78 (25)             | .93     |
| Deep vein thrombosis        | 22 (22)              | 22 (7)              | <.001   |
| Anticoagulation             | 36 (35)              | 107 (34)            | .87     |

Note.—Unless otherwise indicated, data are number of patients, with the percentage in parentheses. PE = pulmonary embolism; RT-PCR = reverse transcription polymerase chain reaction.

* Data in parentheses are the range.

** Period between the first SARS-CoV-2 RT-PCR assay to contrast material–enhanced CT or perfusion scintigraphy.
Discussion

In this multicenter study of 413 patients hospitalized with COVID-19 and suspected of having a pulmonary embolism (PE), PE was found in 25% of patients (95% CI: 21, 29). Although use of CT pulmonary angiography and perfusion scintigraphy in patients with COVID-19 can be cumbersome because of the complex logistics of transporting sick patients infected with COVID-19 to the radiology department, there may be a benefit because we observed no excess mortality when PE was diagnosed and treated. Our random forest model demonstrated d-dimer level was the dominant predictor; a d-dimer level greater than 1600 ng/mL (8.761 nmol/L) had 100% sensitivity and 62% specificity for diagnosis of PE on external validation when used alone. Patients with PE-positive findings and right-sided heart strain confirmed with echocardiography had higher embolic burden than those without right-sided heart strain.

The 25% incidence of PE in all hospitalized patients with COVID-19 is higher than the 17.6% (95% CI: 12.3, 23.5) overall PE incidence and is closer to the 21.7% (95% CI: 14.8, 29.3) incidence in the severe group reported by Liu et al in a systematic review and meta-analysis (7). We also did not find significantly higher PE incidence in patients in the ICU. This might reflect that hospitalized patients in our multicentric cohort had severe illness. The large range in PE incidence reported by Liu et al reflects the heterogeneity and small sample size of the studies included, with most studies having fewer than 100 patients from single institutions.

We found that male sex was significantly associated with PE, in line with previous reports (7,13,14). A similar sex bias regarding COVID-19 and other infectious diseases was previously reported. In a meta-analysis including more than 3 million reported global COVID-19 cases, men were almost three times more likely to require admittance to an intensive treatment unit and had 40% higher odds of death than women (15). This female advantage in COVID-19 may be explained by the sex differences in the immune system (15). The effects of androgen on endothelial function have also been suspected to be a contributing factor, making male sex a potential risk factor for VTE, more so in the context of COVID-19 (7).

The d-dimer level was higher in patients with COVID-19 with PE than in those without PE (16), and this has been confirmed in nearly every publication on this topic (9,17). The d-dimer measures fibrin breakdown products and has been used for prediction and prognosis of VTE among patients with COVID-19 (7,9,17–21). One study showed that d-dimer level peaks as the measurements approached the day of US examination for deep vein thrombosis, following an inverse U shape (19). It is thought that the sensitivity of d-dimer measurement for diagnosis of VTE diminishes with time (17,22). The 1600-ng/mL (8.761 nmol/L) d-dimer threshold we found is lower than most thresholds previously reported for PE or VTE prediction, ranging from 2600 to 7500 ng/mL (14.2–41.0 nmol/L) (9,16,23) and is closer to the 1500-ng/mL (8.21-nmol/L) cutoff proposed by Cui et al (24) in 81 patients.

Figure 3: Contrast-enhanced CT scans in a 63-year-old woman with COVID-19 and a history of antiphospholipid syndrome. (A) Axial image shows paucity of vessels at lung bases with flattening of the interventricular septum (+). (B–D) Axial images show aortic mural clot (arrows).
with severe COVID-19 admitted to the ICU. Tentative diagnosis and provisional prophylactic or therapeutic anticoagulation in patients with severe disease might have contributed to identification of a lower d-dimer threshold.

Previous studies have found that about half of the patients with PE have some degree of right ventricular compromise (25) and increased right-sided heart strain is associated with PE and syncope, as well as higher mortality (26). Among 31 PE-positive patients with COVID-19 with an available echocardiographic report, right-sided heart strain was associated with higher embolic burden. Because an estimated 25% of the pulmonary vasculature must be occluded to result in pulmonary hypertension and because acute right-sided heart failure requires more than 50% occlusion (27), it is not surprising to see higher rates of right ventricular strain in these patients, whose pulmonary vasculature is already compromised by infection. Right ventricular systolic dysfunction was previously reported in patients with COVID-19 without PE (28,29) due to the release of vasoactive mediators, such as serotonin, thromboxane, and histamine, in response to the acute hypoxic injury and platelet-rich clots (30), which may also contribute to right-sided heart strain in these patients.

The main limitation of this study was the retrospective data collection, which made it difficult to control for factors influencing the outcomes. These factors include severity of the disease, treatment protocols, and regular laboratory and clinical data collection. Furthermore, inclusion of only those patients undergoing imaging for suspected PE might not reflect the true incidence of PE in hospitalized patients with COVID-19. Several variables were excluded from the random forest analysis (including partial pressure of oxygen, erythrocyte sedimentation rate, and interleukin-6, C-reactive protein, brain natriuretic peptide, fibrinogen, creatine kinase, and troponin I levels) for having more than 10% missing values; we used imputation for those with missing values below this threshold. Prophylactic anticoagulation may have influenced the d-dimer level in patients suspected of having PE. Furthermore, because of the limited data on prophylactic anticoagulation dosage and a lack of randomization in treatment assignment, our analysis of the effect of prophylactic anticoagulation dosage is limited. Finally, lack of echocardiography within 24 hours in all PE-positive patients prevented us from calculating incidence of right-sided heart strain among all patients.

In conclusion, our results indicate a high incidence of pulmonary embolism (PE) in hospitalized patients with COVID-19 undergoing CT pulmonary angiography or perfusion scintigraphy, which is even higher in men and smokers. A d-dimer level greater than 1600 ng/mL (8.761 nmol/L) is useful in the identification of patients likely to have PE. Right ventricular strain was associated with higher embolic burden.

**Author contributions:** Guarantors of integrity of entire study, S.R., A. Behzadi, H.A., S.I.R., M.R.P.; study concepts/study design or data acquisition or data analysis/interpretation, all authors; manuscript drafting or manuscript revision for important intellectual content, all authors; approval of final version of submitted manuscript, all authors; agrees to ensure any questions related to the work are appropriately resolved, all authors; literature research, S.R., A. Behzadi, J.K., H.A., S.I.R., D.J.M., A. Bamashmos, K.E.; clinical studies, H.D., A. Behzadi, J.K., H.A., S.I.R., A. Bamashmos, K.E., M.R.P.; statistical analysis, S.R., H.A., S.I.R., M.R.P.; and manuscript editing, S.R., H.D., J.K., H.A., S.I.R., D.J.M., A.J., A. Bamashmos, M.R.P.

**Disclosures of Conflicts of Interest:** S.R. disclosed no relevant relationships. H.D. disclosed no relevant relationships. A. Behzadi disclosed no relevant relationships. J.K. disclosed no relevant relationships. H.A. disclosed no relevant relationships. S.I.R. disclosed no relevant relationships. D.J.M. institution received in-kind research support from Siemens Healthineers for MRI development; ad hoc consulting consideration from Promase; honorarium for speaking at a patient support group from
the Prostate Cancer Research Institute. A.J. disclosed no relevant relationships. A.M. disclosed no relevant relationships. H.O.J. disclosed no relevant relationships. A. Ramachmos disclosed no relevant relationships. K.E. disclosed no relevant relationships. M.R.P. disclosed no relevant relationships.

References

1. Devaux CA, Rolain JM, Raoult D. ACE2 receptor polymorphism: Susceptibility to SARS-CoV-2, hypertension, multi-organ failure, and COVID-19 disease outcome. J Microbiol Immunol Infect 2020;53(3):425–435.
2. Varga Z, Flammer AJ, Steiger P, et al. Endothelial cell infection and endothelitis in COVID-19. Lancet 2020;395(10234):1417–1418.
3. Chi G, Lee JJ, Jamil A, et al. Venous thromboembolism among hospitalized patients with COVID-19 undergoing thromboprophylaxis: a systematic review and meta-analysis. J Clin Med 2020;9(8):2489.
4. Henrina J, Putra ICS, Cahyadi I, et al. Clinical characteristics and outcomes of venous thromboembolism in patients hospitalized for COVID-19: systematic review and meta-analysis. Thrombosis Update 2021;2:100037.
5. Grillot F, Behr J, Calame P, Aubry S, Delabrousse E. Acute pulmonary embolism associated with COVID-19: pneumonia detected with pulmonary CT angiography. Radiology 2020;296(3):E186–E188.
6. Kaminetzky M, Moore W, Fansiwala K, et al. Pulmonary embolism at CT angiography. Radiology 2020;296(3):E189–E191.
7. Liu Y, Cai J, Wang C, Jin J, Qu L. Incidence, prognosis, and laboratory indicators of venous thromboembolism in hospitalized patients with coronavirus disease 2019: a systematic review and meta-analysis. J Vasc Surg Venous Lymphatic Disord 2021. 10.1016/j.jvsv.2021.01.012. Published online January 30, 2021.
8. Ullah W, Saeed R, Sarwar U, Patel R, Fischman DL. COVID-19 complicated by acute pulmonary embolism and right-sided heart failure. JACC Case Rep 2020;2(9):1797–1802.
9. Chen J, Wehmer CR, Li HA, et al. D-dimer cut-off points and risk of venous thromboembolism in adult hospitalized patients with COVID-19. Thromb Res 2020;196:318–321.
10. Qanadli SD, El Hajjam M, Veilland-Baron A, et al. New CT index to quantify arterial obstruction in pulmonary embolism: comparison with angiographic index and echocardiography. AJR Am J Roentgenol 2001;176(6):1415–1420.
11. Steenkoven DJ, Buhlmann P. MissForest--non-parametric missing value imputation for mixed-type data. Bioinformatics 2012;28(1):112–118.
12. Chowla NV, Bowyer KW, Hall LO, Kegelmeyer WP. SMOTE: synthetic minority over-sampling technique. J Artif Intell Res 2002;16:321–357.
13. Chang H, Rockman CR, Jacobowitz GR, et al. Deep vein thrombosis in hospitalized patients with coronavirus disease 2019. J Vasc Surg Venous Lymphatic Disord 2021;9(3):597–604.
14. Fauvel C, Weizman O, Trinamille A, et al. Pulmonary embolism in COVID-19 patients: a French multicentre cohort study. Eur Heart J 2020;41(32):3058–3068.
15. Peckham H, de Gruijter NM, Raine C, et al. Male sex identified by global COVID-19 meta-analysis as a risk factor for death and ITU admission. Nat Commun 2020;11(1):6317.