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CASE SERIES

COVID-19-Associated Thromboembolism: A Case Series of Venous, Arterial, and Cardiac Manifestations

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ABSTRACT

Introduction: SARS-CoV-2 infection is frequently associated with coagulation disorders and an increased risk of thromboembolic complications that may involve multiple vascular territories, with potentially fatal consequences. **Case Series:** We present six patients with confirmed COVID-19 who developed confirmed or suspected venous, arterial, or cardiac thromboembolic complications. Patients ranged in age from 54 to 79 years (mean, 64.4 years). All exhibited progressive increases in ferritin levels during hospitalization, while elevated inflammatory markers and dynamic D-dimer changes were observed in association with thromboembolic events. Despite prophylactic or therapeutic anticoagulation, severe complications still occurred. Three patients died from suspected intra-atrial mass, acute femoral and popliteal artery occlusion, and fulminant massive pulmonary thromboembolism, respectively. **Conclusions:** This case series highlights the systemic and heterogeneous nature of COVID-19-associated thromboembolism, which may occur despite anticoagulant prophylaxis, affect multiple vascular territories, and develop even in the absence of traditional thrombotic risk factors.

Keywords: COVID-19, D-dimer, thrombotic events, hypercoagulability

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INTRODUCTION

Coronavirus disease 2019 (COVID-19) is associated with a wide range of systemic complications, among which thromboembolic events represent one of the most serious extrapulmonary manifestations. Both venous and arterial thromboembolism have been reported in patients with COVID-19 and are associated with increased morbidity and mortality.[1,2]

The prothrombotic state associated with COVID-19 results from a complex interplay of systemic inflammation, endothelial dysfunction, direct endothelial injury, and activation of the coagulation cascade. Excessive production of pro-inflammatory cytokines, particularly interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-α), and interleukin-1 (IL-1), together with dysregulated type I interferon responses, contributes to hypercoagulability and thrombus formation.[3–5] Additional mechanisms, includ-

ing antiphospholipid antibodies, immobilization, hypoxia, and potential direct viral effects on coagulation factors, may further increase thromboembolic risk.[6,7]

Thromboembolic complications may occur during both the acute phase of COVID-19 and the convalescent period, with potentially fatal consequences. Despite growing recognition of these complications, important questions remain regarding their clinical spectrum and optimal management. In this context, case series continue to provide valuable insights into uncommon presentations and emerging clinical patterns.

The aim of this study is to present six cases of COVID-19-associated thromboembolism involving venous, arterial, and cardiac manifestations, highlighting their clinical heterogeneity and emphasizing the need for increased clinical vigilance.

CASE SERIES

This retrospective case series presents six adult patients hospitalized with RT-PCR-confirmed COVID-19 at the Infectious Diseases Clinic I in Târgu Mureș, Romania, between January 2021 and March 2022, who developed thromboembolic complications. Depending on the clinical presentation, the diagnosis was confirmed by pulmonary or abdominopelvic computed tomography angiography, vascular Doppler ultrasonography, or transthoracic echocardiography. Demographic characteristics, comorbidities, laboratory findings, imaging results, treatment, and clinical outcomes were collected. Patients with known thrombophilia or without confirmed SARS-CoV-2 infection were excluded.

CASE 1 – SUSPECTED INTRA-ATRIAL MASS

A 54-year-old man with obstructive sleep apnea syndrome and class I obesity presented with cough, fever, myalgia, and progressive dyspnea. At admission, he was diagnosed with severe COVID-19. Laboratory tests revealed mild thrombocytopenia, lymphopenia with neutrophilia, and normal D-dimer levels. Chest computed tomography (CT) revealed extensive ground-glass opacities involving the entire left lung and scattered lesions in the right upper and lower lobes. The patient received antiviral treatment, corticosteroids, an IL-6 inhibitor, a third-generation cephalosporin, and prophylactic anticoagulation with enoxaparin (60 mg once daily). Despite treatment, his condition deteriorated, with a marked increase in D-dimer levels. On the fourth day of hospitalization, he developed hypoxemic respiratory failure requiring intensive care admission and orotracheal intubation, and anticoagulation was intensified to enoxaparin 80 mg twice daily because of respiratory deterioration.

Transthoracic echocardiography performed on the 16th day of hospitalization because of severe hemodynamic instability revealed right heart dilatation, interventricular septum displacement, and a dilated inferior vena cava without inspiratory collapse, consistent with acute right heart overload suggestive of pulmonary thromboembolism. An oval-shaped mass (18 × 11 mm) attached to the anteroseptal wall of the left atrium raised suspicion of an intracardiac mass, although its nature could not be definitively established. Further imaging could not be performed because of the patient's rapidly deteriorating condition. The patient developed pulseless electrical activity cardiac arrest and died despite advanced resuscitation. Histopathological examination was not performed.

CASE 2 – PULMONARY THROMBOEMBOLISM

A 68-year-old man with hypertension presented with fever, cough, and generalized weakness and was admitted with moderate COVID-19. His respiratory status progressively deteriorated from the fourth day of hospitalization, requiring intensive care admission and initiation of prophylactic low-molecular-weight heparin (enoxaparin 60 mg once daily). Laboratory evaluation showed a marked increase in ferritin levels (4,296 ng/ml vs. 1,333 ng/ml at admission). D-dimer levels remained within normal limits during the initial phase of hospitalization.

On the 20th day of hospitalization, sudden worsening of dyspnea accompanied by tachypnea and a rise in D-dimer levels prompted CT angiography, which revealed filling defects in the bilateral superior subsegmental pulmonary arteries and bilateral ground-glass opacities in the upper lobes, some with early fibrotic changes. Anticoagulation was intensified to enoxaparin 80 mg twice daily following the diagnosis of pulmonary thromboembolism, with a favorable hemodynamic outcome. At discharge, the patient required home oxygen therapy because of persistent partial respiratory failure and was prescribed oral anticoagulation.

CASE 3 – PULMONARY THROMBOEMBOLISM COMPLICATED BY PULMONARY INFARCTION AND ABSCESS FORMATION

A 79-year-old woman with hypertension, heart failure, and depression was admitted with severe COVID-19. Chest CT showed near-complete consolidation of the left lower lobe and a circumferential pericardial effusion of 8 mm. Treat-

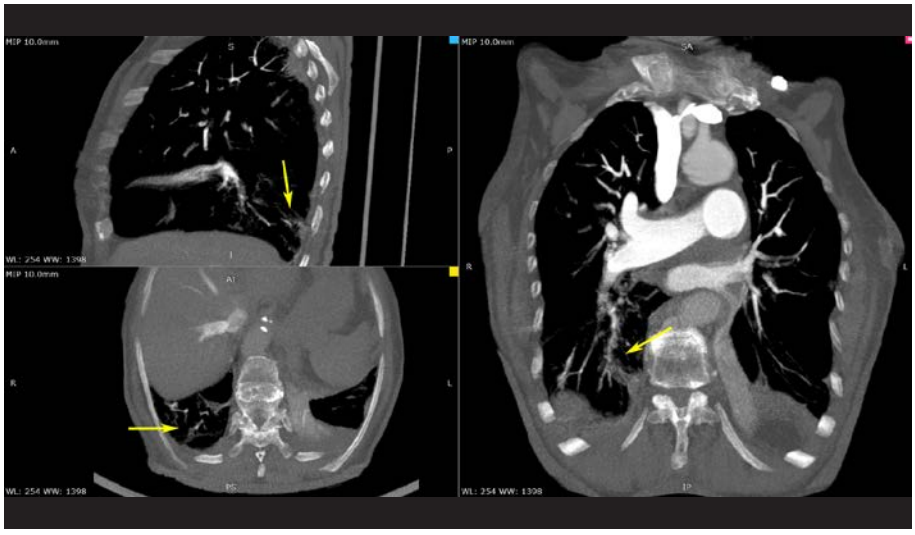

FIGURE 1. Filling defects in the pulmonary arteries supplying the basal lateral and basal posterior segments of the right lower lobe.

ment included remdesivir, corticosteroids, a third-generation cephalosporin, and anticoagulation with enoxaparin (60 mg twice daily), initiated on admission and mantained at the same dose throughout hospitalization.

On the eighth day of hospitalization, worsening hypoxemia, tachypnea, and chest pain prompted pulmonary CT angiography, which confirmed pulmonary thromboembolism with filling defects in the segmental arteries of the right lower lobe (Figure 1).

Eleven days later, repeat chest CT demonstrated a wedge-shaped consolidation in the right lower lobe, together with two fluid-containing cavitary lesions in the basal segments, suggestive of pulmonary abscesses or cavitated pulmonary infarcts. Empirical antibiotic therapy was modified despite the absence of a documented bacterial pathogen. The patient gradually improved and was discharged after 32 days of hospitalization.

CASE 4 – ACUTE FEMORAL AND POPLITEAL ARTERY OCCLUSION

A 58-year-old man with no significant medical history presented with cough, fever, and ageusia and was diagnosed with moderate COVID-19. Chest CT revealed diffuse bilateral interstitial infiltrates. Treatment included favipiravir and prophylactic low-molecular-weight heparin (enoxaparin 60 mg once daily). Despite the addition of dexamethasone and remdesivir following respiratory deterioration on the third day of hospitalization, his clinical condition remained guarded.

On the ninth day of hospitalization, the patient developed sudden pain in the right lower limb, predominantly in the hallux, accompanied by distal hypothermia and mild cyanosis of the right foot. Laboratory tests showed persistently elevated inflammatory markers (C-reactive protein 187.4 mg/L, ferritin 1,420 ng/ml, fibrinogen 499 mg/dl) and markedly elevated D-dimer levels (>3 mg/L), compared with normal values at admission. Arterial Doppler ultrasound demonstrated absent flow in the right common femoral artery and at the origin of the superficial femoral artery. Peripheral CT angiography subsequently confirmed acute occlusion of the right common femoral artery, the proximal two-thirds of the superficial femoral artery with distal reperfusion, and the right popliteal artery. Following confirmation of acute arterial occlusion, anticoagulation was intensified to enoxaparin 60 mg twice daily. Pulmonary CT angiography showed severe COVID-19-related lung involvement without evidence of pulmonary thromboembolism (Figure 2). Because of the patient's critical condition and high operative risk, surgical revascularization was not feasable. The patient developed multiple organ failure and died.

CASE 5 – SUSPECTED PULMONARY THROMBOEMBOLISM

A 60-year-old woman with no significant medical history was admitted with fever, productive cough, ageusia, anosmia, and asthenia due to COVID-19. Laboratory tests showed normal D-dimer levels and only mildly elevated inflammatory markers. Chest CT demonstrated diffuse bilateral interstitial infiltrates. Treatment included remdesivir, dexamethasone, prophylactic-dose anticoagulation with nadroparin (0.4 ml once daily), a third-generation cephalosporin, and symptomatic therapy.

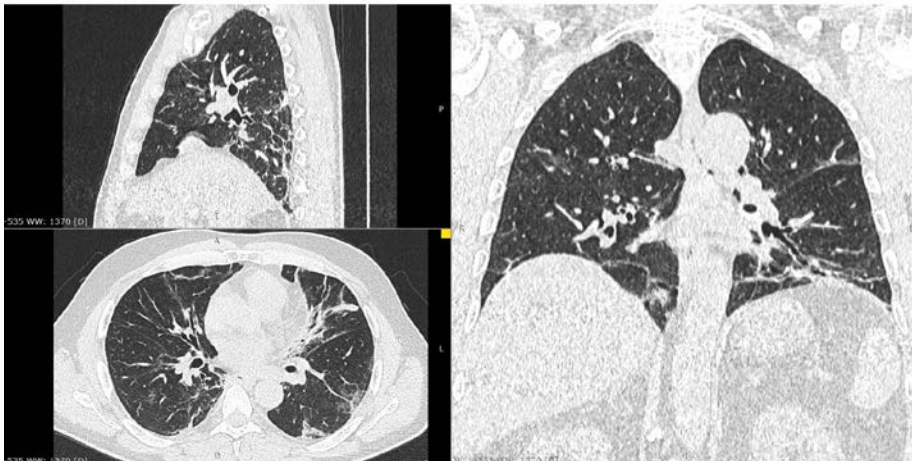

FIGURE 2. CT angiogram showing severe pulmonary involvement, with extensive bilateral ground-glass opacities involving nearly the entire lung parenchyma and no evidence of central pulmonary embolism.

The patient's condition gradually improved, and discharge was planned. However, on the fifth day of hospitalization, one day before the planned discharge, she developed sudden severe respiratory deterioration with hypoxemia, tachypnea, tachycardia, and hemodynamic instability, requiring intensive care admission. D-dimer levels increased from 0.9 mg/L to 10.8 mg/L within 48 hours. Because of patient's rapidly deteriorating condition, imaging confirmation could not be obtained, and the diagnosis of suspected massive acute pulmonary thromboembolism was established on the basis of clinical and laboratory findings. Despite resuscitation, she died less than 24 hours later from acute cardiorespiratory decompensation.

CASE 6 – ACUTE-ON-CHRONIC MESENTERIC ISCHEMIA

A 65-year-old man with insulin-dependent type 2 diabetes mellitus, hypertension, and ischemic heart disease was admitted with moderate COVID-19. Initial laboratory tests showed elevated inflammatory markers and D-dimer levels. Treatment included dexamethasone, therapeutic-dose anticoagulation with enoxaparin (60 mg twice daily), a third-generation cephalosporin, and symptomatic therapy.

On the ninth day of hospitalization, worsening hyperglycemia required insulin pump therapy. Two days later, sudden severe abdominal pain prompted contrast-enhanced abdominopelvic CT, which revealed segmental stenoses of the superior and inferior mesenteric arteries associated

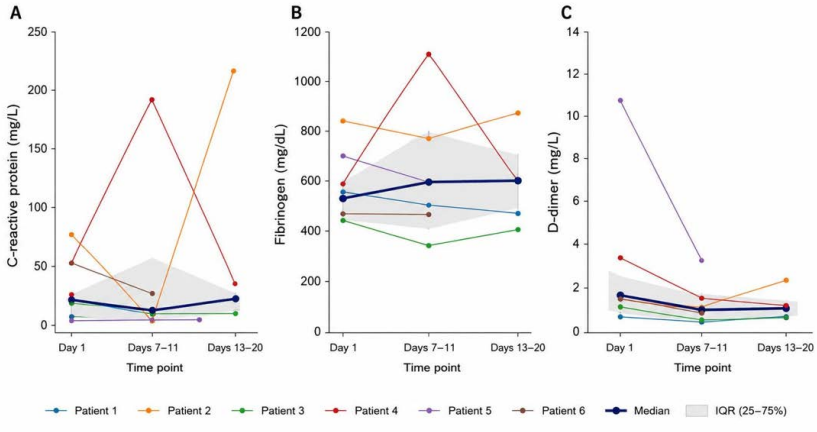

FIGURE 3. Temporal evolution of inflammatory and coagulation biomarkers in the six patients. **A.** CRP levels. **B.** Fibrinogen levels. **C.** D-dimer levels. Individual trajectories are represented by thin lines, while the thick line denotes the median value at each time point. Measurements were performed on day 1, days 7–11, and days 13–20. Data for patients 5 and 6 were available only up to days 7–11.

TABLE 1. Bloodwork evolution during inpatient stay

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Age (years)	54	68	79	58	60	65
Sex	Male	M	F	M	F	M
BMI (kg/m^2)	37	29.39	23.8	26.77	22.4	31.8
Severity of COVID-19 at admission	Severe	Moderate	Severe	Moderate	Severe	Moderate
No. of days from onset of illness to admission	5	3	10	4	9	13
Thromboembolic event	Suspected intracardiac thrombotic involvement	Pulmonary thromboembolism	Pulmonary thromboembolism with abscessed pulmonary infarction	Acute femoral and popliteal artery occlusion	Suspected pulmonary thromboembolism	Acute-on-chronic mesenteric ischemia
Time of onset of complication from admission (days)	16	20	8	9	8	11
SpO_2 on room air at admission	78%	93%	89%	94%	90%	98%
White blood cell count at admission (× 10^3/µl)	6.8	4.0	4.94	6.25	6.98	4.49
Platelet count at admission (× 10^3/µl)	124	124	215	163	306	185
D-dimer on admission (mg/L)	<0.5	<0.5	>0.5	<0.5	<0.5	>0.5
Maximum D-dimer (mg/L)	1.88	1.92	2.05	>3	10.8	>0.5
Maxim CRP (mg/L)	0.4	127	197.8	187.4	97	56.2
Fibrinogen (mg/dl)						
Min	127	189	196	480	345	–
Max	304	545	447	619	416	427
INR	0.91	1.29	1.30	1.44	1.06	1.13
APTT (s)	24.1	31.6	20.9	138.5	27.4	28.4
IL-6 (pg/ml)	48.09	53.7	NA	175.52	NA	NA
Max ferritin (ng/ml)	667.4	4296	206.9	1977.7	1002	651.8
Imaging method	Echocardiography					
Chest CT	Pulmonary CT angiography	Pulmonary CT angiography	Pulmonary CT angiography and Doppler echocardiography	NA	Abdominopelvic CT angiography	
Anticoagulation upon admission	Prophylactic	Prophylactic	Therapeutic	Prophylactic	Prophylactic	Therapeutic
Admission to intensive care	Yes	Yes	Yes	Yes	Yes	No
Evolution	Death	Discharged	Discharged	Death	Death	Discharged

with parietal atherosclerotic plaques. Preserved distal arterial flow and the absence of segmental occlusive defects were suggestive of chronic mesenteric ischemia, possibly complicated by an acute-on-chronic ischemic episode. Following surgical consultation, conservative management with close clinical monitoring was recommended.

The main clinical, biological, and imaging characteristics of the six patients are summarized in Table 1, and the temporal evolution of inflammatory and coagulation biomarkers is presented in Figure 3. No patient had severe renal impairment requiring dose adjustment of anticoagulant therapy.

DISCUSSION

SARS-CoV-2 infection is associated with a prothrombotic state resulting from the interplay of systemic inflammation, endothelial dysfunction, platelet activation, and activation of the coagulation cascade. Excessive production of pro-inflammatory cytokines, direct endothelial injury mediated through ACE2 receptors, complement activation, and neutrophil extracellular trap (NET) formation contribute to the development of immunothrombosis and increase the risk of both venous and arterial thromboembolic events.[2,7–12]

The present case series illustrates the broad spectrum of COVID-19-associated thromboembolic complications, involving the venous and arterial systems as well as the heart. The six patients had a mean age of 64 years, with thromboembolic events developing a mean of 19.3 days after symptom onset. Most events occurred during the second week of illness, corresponding to the period of peak systemic inflammation described in COVID-19 and supporting the relationship between severe systemic inflammation, hypercoagulability, and thromboembolism.

The laboratory findings were consistent with severe COVID-19. Although total white blood cell counts remained within the normal range in most patients, lymphopenia common. Among the biomarkers evaluated, progressive ferritin elevation was the most consistent finding across all cases, reflecting intensification of the inflammatory response. Elevated IL-6 levels, when measured, together with increased C-reactive protein concentrations further supported the role of cytokine-mediated inflammation. Fibrinogen showed a biphasic pattern, with declining levels in some patients despite clinical deterioration, suggesting consumption coagulopathy rather than reduced thrombotic risk.[13]

Case 1 highlights the diagnostic challenges posed by intracardiac masses in patients with severe COVID-19. The nature of the left atrial mass remained uncertain in the absence of histopathological confirmation, as imaging mimics such as atrial myxoma or prominent trabeculations could not be completely excluded. The combination of severe right-sided chamber enlargement, inferior vena cava dilation without inspiratory collapse, interventricular septal shift, and a marked prothrombotic state was suggestive of acute right heart overload and possible pulmonary thromboembolism. Although neither the nature of the atrial mass nor pulmonary thromboembolism could be definitively established, a thromboembolic process was considered plausible in the context of severe COVID-19 Hypercoagulability, endothelial dysfunction, and venous stasis, are recognized contributors to COVID-19-associated thrombosis, consistent with Virchow's triad.[14,15]

A major finding of this series is the occurrence of thromboembolic complications despite anticoagulant prophylaxis. Conventional venous thromboembolism risk scores were developed before the COVID-19 pandemic and do not account for the thombo-inflammatory state induced by SARS-CoV-2 infection.[1,16] Severe systemic inflammation, endothelial dysfunction, complement activation, and NET formation may exceed the predictive capacity of these models.[9,10] Although D-dimer is a useful marker of coagulation activation, its interpretation remains challenging because it also reflects systemic inflammation.[10] Dynamic increases in ferritin, IL-6, and D-dimer have been associated with increased thrombotic risk despite standard thromboprophylaxis.[3,17,18] However, any decision to intensify anticoagulation must be balanced against the potential risk of bleeding.

The present series illustrates the phenomenon of breakthrough thrombosis. Four patients initially received prophylactic enoxaparin, whereas two (Cases 3 and 6) received therapeutic anticoagulation from admission. Nevertheless, severe thromboembolic complications still occurred. Case 1 developed a suspected intracardiac mass despite escalation of anticoagulation, suggesting that thrombotic risk may persist despite anticoagulant therapy in severe COVID-19. Case 5 further illustrates this phenomenon: despite no significant medical history, apparent clinical improvement, and ongoing prophylactic anticoagulation, the patient developed fatal suspected massive pulmonary thromboembolism one day before the planned discharge, accompanied by a rapid increase in D-dimer levels from 0.9 to 10.8 mg/L. Together, these findings emphasize the need for continued clinical and laboratory vigilance throughout hospitalization and support further research to optimize anticoagulation strategies in COVID-19.

Two patients in our series developed arterial ischemic complications, involving acute femoral and popliteal artery occlusion and acute-on-chronic mesenteric ischemia, respectively. Although less common than venous thromboembolism, arterial thrombotic events may have devastating consequences. Unlike venous thrombosis, which is primarily driven by hypercoagulability and stasis, arterial thrombosis in COVID-19 appears to depend more on endothelial injury and platelet activation following SARS-CoV-2 infection of vascular endothelial cells through ACE2 receptors.[7,15] Both patients developed arterial ischemic complications on the 11th hospital day, accompanied by elevated inflammatory and coagulation markers. However, D-dimer and fibrinogen levels did not correlate with the vascular territory involved. Case 5, with suspected pulmonary thromboembolism, had the highest D-dimer concentration (10.8 mg/L), whereas the two arterial cases showed more moderate elevations. These findings suggest that arterial thrombosis in COVID-19 is not determined solely by systemic hypercoagulability but also by local endothelial dysfunction and platelet activation.

The clinical presentation also differed between venous and arterial thromboembolic events. Venous thromboembolism evolved more insidiously, whereas arterial thrombosis presented abruptly with acute ischemic

symptoms. Case 4 developed acute lower-limb ischemia requiring urgent surgery that could not be performed because of the patient's critical condition. Case 6 illustrates the potential interaction between pre-existing vascular disease and the COVID-19-associated thrombo-inflammatory environment. The patient had insulin-dependent diabetes mellitus, hypertension, ischemic heart disease, and imaging findings suggestive of chronic mesenteric ischemia. Severe hyperglycemia requiring insulin pump therapy may have further amplified endothelial dysfunction, platelet activation, and inflammation, thereby increasing thrombotic risk.

The concept of 'multiple-hit thrombosis' provides a useful framework for understanding the thromboembolic complications observed in our series. Patients were simultaneously exposed to severe systemic inflammation, viral endotheliopathy, cytokine storm-associated hypercoagulability, NET formation, obesity, prolonged immobilization, invasive devices, and hypoxia. Although each factor alone may not necessarily result in thromboembolism, their cumulative and synergistic effects likely created a profoundly prothrombotic environment. This may explain why severe thromboembolic complications occurred despite anticoagulation, suggesting that the challenge in severe COVID-19 extends beyond hypercoagulability alone and reflects the interaction of multiple pathogenic mechanisms.

The main limitations of this study are the small sample size and the descriptive, retrospective design, which may have limited the availability of complete clinical and laboratory data. Nevertheless, the cases presented illustrate the diversity of thromboembolic manifestations associated with SARS-CoV-2 infection and highlight the importance of careful clinical and imaging evaluation of these patients.

CONCLUSIONS

This case series highlights the broad and heterogeneous spectrum of COVID-19-associated thrombembolic complications involving the cardiac, pulmonary, and arterial circulations. Progressive increases in ferritin levels, elevated IL-6 and C-reactive protein concentrations, together with dynamic changes in D-dimer levels, were observed in association with COVID-19-associated thromboembolic complications. However, no predictive conclusions regarding individual biomarkers can be darwn from this small case series. These findings highlight the importance of close clinical assessment, regular laboratory monitoring, and timely imaging in patients with suspected thromboembolic complications.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

INFORMED CONSENT

Informed consent was obtained from all subjects involved in the study.

DATA AVAILABILITY

Data supporting the results of this article will be provided on request, by the corresponding author.

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AUTHOR CONTRIBUTIONS

V.N. contributed to the study conception, data collection, patient management, literature review, data analysis, and manuscript drafting. A.V. contributed to the cardiology evaluation, data interpretation, and critical revision of manusript. A.A.V contributed to data collection and figure preparation. A.M.V conceived and supervised the study, and critically revised the manuscript. All authors have read and agreeed to the published version of the manuscript.

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