Received: 2 June 2020 | Revised: 15 August 2020 | Accepted: 18 August 2020

DOI: 10.1002/rth2.12435

CASE REPORT

rpth research & practice in thrombosis & haemostasis

Left ventricular thrombus and pulmonary embolism: A case series of thrombosis in COVID-19 in Thai patients

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Handling Editor: Susan Kahn

Abstract

Thrombosis in COVID-19 is increasingly recognized and is generally associated with a high mortality rate. The key clinical question of this report was whether COVID-19 could be complicated with cardiac thrombus and pulmonary embolism in Asian population. We demonstrated the case series of thrombosis in Thai patients with confirmed severe acute respiratory syndrome coronavirus 2 infection. One patient had the first case of a large left ventricular thrombus, and three other patients had pulmonary embolism. All patients were male and had low absolute lymphocyte count, while lactate dehydrogenase level and D-dimer were markedly elevated, especially at the time when the thrombosis was diagnosed. All patients had severe COVID-19 with pneumonia. Two patients who needed mechanical ventilation were successfully extubated. After hospitalization for 13-49 days, pneumonia and thrombosis improved and all of them could be discharged from the hospital. Thrombosis is common in COVID-19 and could present in both arterial and venous sites even in Asian populations. D-dimer is a strong marker to predict thrombosis and could be a prognostic predictor for severity of COVID-19.

KEYWORDS

blood clot, coronavirus, COVID-19, pulmonary embolism, thrombosis

Essentials

- We present the first reported case of left ventricular thrombus in coronavirus disease 2019 (COVID-19).
- Three consecutive cases of pulmonary embolism in Thai patients with COVID-19 are also demonstrated.
- D-dimer is a strong marker to predict thrombosis.
- Thromboprophylaxis is essential in Asian patients hospitalized with COVID-19.

1 | INTRODUCTION

There is a high prevalence of venous thromboembolism (VTE) among patients with severe coronavirus disease 2019 (COVID-19).[1] According to recent studies, the incidence of pulmonary embolism (PE) in patients with COVID-19 pneumonia in the intensive care unit (ICU) ranged from 22% to 69%.[2-5] However, thrombosis in Thai patients with COVID-19 has not been reported. Some reports have shown that thrombosis in COVID-19 could present in various sites—for instance, pulmonary, mesenteric, and intracranial vessels—and was associated with a high mortality rate.[4,6] A lower incidence of VTE was reported in the Asian population than in the White population.[7]

The key clinical question of this report was whether COVID-19 could be complicated with cardiac thrombus and PE in Asian populations. Here, we report a case series of patients with severe COVID-19 pneumonia who developed thrombosis in our cohort of 144 patients admitted in our hospital.[8] We defined severe COVID-19 according to the World Health Organization definition (fever or suspected respiratory infection plus one of the following: respiratory rate >30/min or oxygen saturation ≤93% on room air).[9] All of them survived.

To our knowledge, case 1 is the first case report of large left ventricular (LV) thrombus in COVID-19. Three other consecutive cases with PE are also demonstrated. As far as we know, there is no report of PE in patients with COVID-19 in other hospitals in Thailand. Also, there are only few reports of PE in the Asian population with COVID-19. There are challenges in the diagnosis of PE in COVID-19 patients (ie, the clinical signs and symptoms of acute PE can overlap with the symptoms of COVID-19 infection, and diagnostic examinations cannot be easily performed). Therefore, PE could be underdiagnosed. We would like to raise awareness that PE also occurs in Asian patients with COVID-19 and pharmacological thromboprophylaxis is essential in Asian patients hospitalized with COVID-19.

2 | CASE SERIES

2.1 | Case 1

A 71-year-old man presented with acute watery diarrhea for 10 days and was found to have COVID-19. His underlying health risks were hypertension, dyslipidemia, and a history of coronary artery disease (CAD) with balloon angioplasty for 10 years. He had taken clopidogrel and an angiotensin-converting enzyme inhibitor daily for secondary prevention of CAD. His vital signs at admission were normal and oxygen saturation was 95% on room air. Baseline chest radiograph revealed ground-glass opacities at both lower lung fields. His complete blood count showed a low absolute lymphocyte count (ALC) of 1020/mm^3, elevated lactate dehydrogenase (LDH) level of 317 U/L (135-225) and high D-dimer of 1548 ng/mL fibrinogen equivalent units (FEU). He was treated with hydroxychloroquine and darunavir/ritonavir on the first day of admission (the 10th day of his illness). On the 11th day of illness, the patient's symptoms progressed and oxygen saturation decreased to 93% on room air as a result of severe pneumonia. His symptoms and oxygen saturation improved after a complete course of favipiravir and supportive oxygen therapy.

Due to the extremely high level of D-dimer (40 560 ng/mL FEU) on 19th day of illness (Table 1), PE was suspected, and computed tomography pulmonary angiography (CTPA) was performed. PE was not detected. However, a large 4.1 × 2.0 cm. thrombus was seen in the left ventricle (Figure 1A). Echocardiogram confirmed a large LV

TABLE 1 Clinical characteristics and laboratory findings of 4 cases

Patients	Case 1	Case 2	Case 3	Case 4
Sex	Male	Male	Male	Male
Age	71	44	50	44
BMI (kg/m^2)	21.8	34	21	24.8
Medical history	CAD, HT	Gout	DM	HT, DLP
Day of illness at diagnosis of thrombosis	19	20	13	14
Disease severity	Severe pneumonia	Severe pneumonia with ARDS	Severe pneumonia with ARDS	Severe pneumonia
Need for ICU	No	Yes	Yes	Yes
Anticoagulant prophylaxis	No	Yes[a]	No	No
Chest radiograph	GGO, bilateral pulmonary infiltrates	GGO, bilateral pulmonary infiltrates	GGO, bilateral pulmonary infiltrates	GGO, bilateral pulmonary infiltrates
Sites of thrombus by CTPA	LV	Bifurcation of the main and multiple bilateral branches of PA	Extensive bilateral interlobar and subsegmental PA	Bilateral lobar and segmental PA
Antiphospholipid antibodies[b]	Negative	Negative	Negative	Negative

(Continues)

TABLE 1 (Continued)

Patients	Case 1	Case 2	Case 3	Case 4
IL-6 (pg/mL)	NA	52	92	NA
D-dimer (ng/mL FEU)				
- At admission	814	410	5678	800
- At thrombosis	40 560	39 400	15 320	67 820
- After treatment with anticoagulant	6240	6860	3030	9270
ALC (mm^3)				
- At admission	1020	1579	693	1073
- At thrombosis	2233	869	1452	1818
- After treatment with anticoagulant	1532	1240	2076	1236
Platelet count (mm^3)				
- At admission	309 000	158 000	246 000	233 000
- At thrombosis	542 000	153 000	228 000	302 000
- After treatment with anticoagulant	367 000	138 000	293 000	271 000
PT (s)				
- At admission	NA	12.8	12.0	NA
- At thrombosis	12.4	12.7	12.8	12.4
- After treatment with anticoagulant	12.4	12.5	12.4	12.9
aPTT (s)				
- At admission	NA	28.4	30.0	NA
- At thrombosis	23.3	23.9	23.4	26.3
- After treatment with anticoagulant	24.3	45.7[c]	24.8	27.1
Fibrinogen (mg/dL)				
- At admission	NA	363	NA	NA
- At thrombosis	414	283	582	558
- After treatment with anticoagulant	363	441	607	516
LDH (U/L)				
- At admission	362	234	440	582
- At thrombosis	262	706	444	340
- After treatment with anticoagulant	222	440	333	262

Abbreviations: ALC, absolute lymphocyte count; aPTT, activated partial thromboplastin; ARDS, acute respiratory distress syndrome; BMI, body mass index; CAD, coronary artery disease; CTPA, computed tomography pulmonary angiography; DLP, dyslipidemia; DM, diabetes mellitus; DVT, deep vein thrombosis; GGO, ground-glass opacity; HT, hypertension; ICU, intensive care unit; IL-6, interleukin-6; LDH, lactate dehydrogenase; NA, not available; PA, pulmonary artery; PE, pulmonary embolism; PT, prothrombin time.

[a]Patient received prophylaxis anticoagulant but was discontinued due to upper gastrointestinal hemorrhage.

[b]Antiphospholipid antibodies (lupus anticoagulant, IgG and IgM anticardiolipin, IgG and IgM anti–β_2-glycoprotein.

[c]Treatment with heparin.

thrombus (Figure 1B,C) and mildly impaired LV function, likely from the patient's history of CAD. His electrocardiogram demonstrated a normal sinus rhythm. He was treated with therapeutic low-molecular-weight heparin (LMWH) and underwent bridging to oral warfarin for the next 3 days. Eventually, he was discharged from the hospital with warfarin on the 34th day of his illness.

2.2 | Case 2

A 44-year-old man presented with fever for 7 days and was diagnosed COVID-19. He had a history of gouty arthritis and obesity (body mass index 34 kg/m^2). Chest radiograph showed ground-glass opacities at both lower lung fields. He was prescribed hydroxychloroquine,

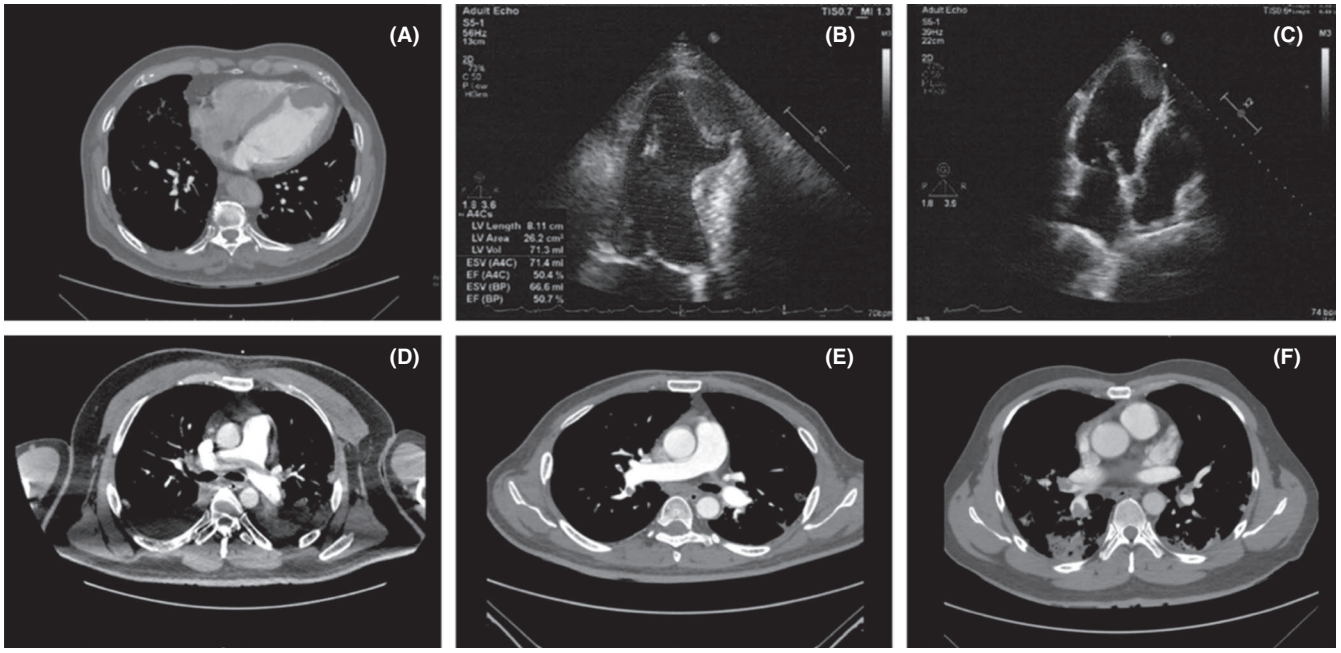

FIGURE 1 Computed tomography pulmonary angiography (CTPA) and echocardiography illustrating a large bland thrombus within left ventricle (A, B, and C) in case 1. CTPA showing acute pulmonary embolism involving bifurcation of the main pulmonary artery and multiple bilateral lobar and segmental branches in case 2 (D). CTPA showing extensive acute pulmonary embolism interlobar and subsegmental pulmonary arteries of all lobes in case 3 (E). CTPA showing acute pulmonary embolism involving bilateral lobar and segmental pulmonary arteries in case 4 (F)

darunavir/ritonavir, azithromycin, and favipiravir on the day of admission (the 7th day of illness) for the diagnosis of severe pneumonia. He also received enoxaparin 40 mg daily for VTE prophylaxis.

On the 15th day of illness, his pneumonia progressed to respiratory failure. Thus, he was intubated and transferred to the ICU. Three days later, the patient developed a nonmassive upper gastrointestinal hemorrhage. Enoxaparin was then discontinued. On the 20th day of illness, he developed acute severe hypoxemia, and PE was suspected. Investigations showed the level of D-dimer sharply rising to 39 400 ng/mL FEU (Table 1). CTPA revealed acute PE involving bifurcation of the main pulmonary artery (Figure 1D). Therapeutic dose of unfractionated heparin (UFH) was administered. Despite optimal mechanical ventilation and prone positioning, he still had worsening hypoxemia. Venovenous extracorporeal membrane oxygenation (VV-ECMO) was initiated on the 21st day of illness. His symptoms then gradually improved.

On the 30th day of illness, tension hemothorax was detected, and a percutaneous drainage tube was inserted. VV-ECMO was later removed, and UFH was changed to LMWH. Finally, he was extubated on the 39th day of illness and discharged on the 54th day of his illness with warfarin.

2.3 | Case 3

A 50-year-old man presented at the emergency department with acute respiratory failure on the 8th day of illness. Chest radiograph showed bilateral lung infiltrations. The reverse transcriptase PCR for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) later revealed a positive result. He was then treated with darunavir/ritonavir, favipiravir, and hydroxychloroquine for COVID-19 pneumonia. Diabetes mellitus was also newly diagnosed.

On the 13th day of illness, he developed sinus tachycardia and desaturation. Bedside echocardiography illustrated signs of right ventricular overload (dilated D-shaped right ventricle). CTPA was performed and demonstrated extensive acute PE involving interlobar and subsegmental pulmonary arteries of all lobes (Figure 1E). Laboratory results showed markedly elevated D-dimer levels (15 320 ng/mL FEU), low ALC, and elevated LDH and interleukin-6. He was treated with therapeutic LMWH. Eventually, he was extubated on the 28th day of illness and discharged on the 42nd day of his illness with warfarin.

2.4 | Case 4

A 44-year-old guitarist presented with fever and progressive dyspnea for 12 days and was confirmed to have COVID-19. He had a history of hypertension and dyslipidemia. Chest radiograph demonstrated bilateral ground-glass opacities of both lower lung zones. He received hydroxychloroquine, darunavir/ritonavir, azithromycin, and favipiravir for COVID-19 pneumonia.

On the 14th day of illness, the patient developed impending respiratory failure. During that period, his D-dimer level was extremely high (67 820 ng/mL FEU). (Table 1) CTPA revealed acute PE involving bilateral lobar and segmental pulmonary arteries (Figure 1F).

Therapeutic doses of LMWH were given. His condition improved, and he was discharged home with warfarin on the 29th day of illness.

3 | DISCUSSION

We have demonstrated four cases of thrombosis in Thai patients with severe COVID-19, one case with a large thrombus in the left ventricle and three cases with PE. All patients had severe pneumonia and received the same regimen of antiviral agents. All were males and were characterized by having baseline lymphopenia with markedly elevated D-dimer levels. Table 1 illustrates the sequential values of D-dimer during the course of hospitalization. The D-dimer levels of all patients were >1500 ng/mL FEU, continuously rose over time, and peaked on the day of diagnosis of thrombosis or PE. D-dimer levels dropped rapidly after initiating the anticoagulant. The results of antiphospholipid antibodies (lupus anticoagulant, IgG and IgM anticardiolipin, IgG and IgM anti–β_2-glycoprotein) were negative in all patients. All four patients had no personal or family history of thrombosis. Because all patients had no symptoms and signs of deep vein thrombosis, we did not perform screening compression ultrasonography of the lower extremities. Three patients did not receive pharmacological thromboprophylaxis because they were hospitalized in the early period of the COVID-19 pandemic and there was no routinely recommended pharmacological thromboprophylaxis for medical patients, especially in the Asian population, during that time. The other one (case 2) received prophylactic LMWH after the thromboprophylaxis protocol was implemented for all patients with severe COVID-19. Unfortunately, prophylactic LMWH was withheld for 2 days as a result of the upper gastrointestinal hemorrhage before the development of PE. At that time there were still limited data on the optimal dose of LMWH prophylaxis in patients with COVID-19. Therefore, the standard fixed dose of 40 mg of enoxaparin was used. After this case, we have changed our thromboprophylaxis protocol from fixed-dose LMWH prophylaxis to weight-based LMWH prophylaxis.

All patients had severe pneumonia and received the same regimen of antiviral agents. Three patients who had PEs were admitted to the ICU. Recent study has shown that critically ill patients with COVID-19 were at a higher risk for the development of a PE than patients with influenza.[3] Global data have also shown that disease severity and mortality from COVID-19 are higher among men than women. Multiple dimensions of biological sex, including sex steroids, sex chromosomes, and genomic and epigenetic differences between men and women impact immune responses and may affect responses to SARS-CoV-2 infection. In addition, underlying preexisting comorbidities (such as ischemic heart disease, diabetes, or hypertension) and high-risk behaviors (such as smoking and alcohol use) are more frequent in men. Sex differences in angiotensin-converting enzyme 2 (ACE2) receptors and ACE2 enzyme activity have also been proposed.[10,11]

We have demonstrated the first case report of a large LV thrombus in a patient with COVID-19 pneumonia (case 1). There are several known conditions associated with cardiac thrombi such as atrial fibrillation or ischemic cardiomyopathy.[12] However, the exact cause of thrombus in the left ventricle in this patient was unknown. Cardiac thrombi in ischemic cardiomyopathy usually develops in patients with severely impaired left ventricular ejection fraction.[12,13] Our patient only had mildly impaired cardiac function and did not have atrial fibrillation. Thus, other causes may contribute to the etiology of the large thrombus in this patient. COVID-19–related severe hypercoagulability could be the culprit of thrombosis in the cardiac chamber or might aggravate the thrombus to become larger. In this case, the risk of complications from surgical thrombectomy was considerably high; hence, therapeutic anticoagulation was used in the first instance. Anticoagulants are used to reduce the risk of stroke or systemic embolism. A recently published multicenter cohort study of anticoagulation strategies for LV thrombi showed that off-label use of direct oral anticoagulants was associated with a higher risk of systemic embolism compared with warfarin use. Prospective randomized clinical trials are needed to determine the most effective treatment strategies for LV thrombi.[14] This patient was successfully treated with warfarin, and we plan to do follow-up echocardiogram at 3 months after anticoagulant therapy.

Patients with COVID-19 have increased risk of thromboembolic events, and several mechanisms may be involved in the pathogenesis of these complications. Most of the reports on a high incidence of thrombosis are in relation to venous thrombosis, while the evidence about arterial thrombosis in patients with COVID-19 is limited. The rate of ischemic stroke and acute coronary syndrome was 2.5% and 1.1%, respectively, in one study.[4] There were also reported cases of aortic thrombi.[15,16]

The mechanism of the hypercoagulability leading to arterial thrombosis is yet to be fully elucidated. The proposed mechanism of SARS-CoV-2 internalization into human host cells assumes that the viral particles bind to ACE2 receptors, which abundantly present at multiple tissues, especially lung and endothelial cells. SARS-CoV-2 infection can produce endothelial dysfunction due to direct invasion of endothelial cells by the virus or mediated by the presence of cytokines and other acute-phase reactants. Severe COVID-19 leads to an overwhelming release of proinflammatory cytokines (eg, interleukin-6), and activates endothelial cells, neutrophils, mononuclear cells, and platelets, leading to tissue factor–mediated activation of coagulation. Various changes in circulating prothrombotic factors have been reported in patients with severe COVID-19. Local thrombosis could be due to inflammation and hypoxia, leading to endothelial cell activation and tissue factor expression, downregulation of thrombomodulin, and loss of heparan sulfate.[17] Ventricular thrombus is a rare complication of stress cardiomyopathy. Although the specific mechanism of ventricular thrombus in COVID-19 is not fully understood, it could be due to hypercoagulability and local myocardial inflammation creating an area of relatively static blood that is prone to thrombus formation.

D-dimer has been proposed as a prognostic marker of disease severity and mortality in patients with COVID-19.[18,19] Although D-dimer level can rise in many conditions, it is considered a sensitive indicator for identifying

the risk of VTE.[2,4,20] One study from China suggested that the acceptable cutoff value of D-dimer levels to predict VTE was 1500 ng/mL FEU with the sensitivity and specificity of 85% and 88.5%, respectively.[20]

One study from China demonstrated that the prevalence of VTE was 25% in patients who did not receive anticoagulant prophylaxis.[20] All had a lower extremity venous thrombosis. PE was not reported. Studies from Europe revealed that 20%-69% of patients with COVID-19 experienced breakthrough symptomatic VTE even on prophylactic thromboprophylaxis.[2-5] The optimal dose of LMWH thromboprophylaxis is controversial. A higher dose of LMWH may be required for preventing VTE in this situation.[2-5,21] Several randomized controlled trials looking at the appropriate intensity of LMWH prophylaxis are still ongoing in patients with COVID-19.

In conclusion, this report addresses the problem of thrombosis in COVID-19 in the Asian population. Awareness of thrombosis and prompt diagnosis and treatment are crucial in COVID-19 patients. Moreover, this report supports the recommendation that thromboprophylaxis is required in Asian patients with severe COVID-19 pneumonia.

RELATIONSHIP DISCLOSURE

The authors report nothing to disclose.

AUTHOR CONTRIBUTIONS

NN, SP, SS, PC, and PA collected and interpreted the clinical data and wrote the article. NN and KT treated the patients. All critically reviewed the article and gave final approval.

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How to cite this article: Nanthatanti N, Phusanti S, Chantrathammachart P, Thammavaranucupt K, Angchaisuksiri P, Sungkanuparph S. Left ventricular thrombus and pulmonary embolism: A case series of thrombosis in COVID-19 in Thai patients. *Res Pract Thromb Haemost* 2020;4:1224–1229. https://doi.org/10.1002/rth2.12435