

Chronic intracardiac thrombus, a long-term complication of COVID-19: Case reports

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Abstract

Inflammation and thrombogenic effects of coronavirus disease 2019 (COVID-19) can lead to cardiovascular complications in patients even after recovery from COVID-19. Intracardiac thrombus is life-threatening and can cause sudden death. Our study describes two patients who recovered from COVID-19 and presented with chronic intracardiac thrombus.

KEYWORDS

COVID-19 infection, intracardiac thrombus, open-heart surgery

1 | INTRODUCTION

Patients with coronavirus disease 2019 (COVID-19) have an increased risk of thromboembolism, which can lead to life-threatening cardiovascular complications, such as acute myocardial infarction and intracardiac thrombus.^{1,2} Even in patients recovering from COVID-19, these complications pose potential risks to the cardiovascular system in the long term. Due to the severity of the inflammation and the tendency to develop thromboembolism, sudden deaths may occur due to the presence of a large thrombus.³

2 | CASE REPORTS

The clinical, laboratory, and demographic characteristics of two patients previously infected with COVID-19 when admitted to the hospital are presented in Table 1.

2.1 | Case 1

An 80-year-old patient was diagnosed with COVID-19 two months ago and her hematological, biochemical, and inflammatory

parameters at that time were as described in Table 2. A telecardiogram, thoracic CT scan, and radiological examination revealed diffuse bilateral consolidation and peripheral ground-glass opacification, thereby confirming COVID-19 infection. The COVID-19 positive patient, whose oxygen saturation values decreased, was hospitalized and taken to intensive care. The patient was given breathing exercises and pressurized oxygen therapy, but without intubation. In addition, enoxaparin 6000 IU, favipiravir 200 mg, hydroxychloroquine 200 mg, ascorbic acid 1000 mg, and methylprednisolone 80 mg ampoule treatments were initiated. After 1 week of treatment, the oxygen saturation values of the patient increased and he was discharged on the 10th day. During the discharge phase, D-Dimer, ferritin, and fibrinogen levels decreased, although not at normal values. A CT performed due to an old COVID-19 infection revealed fibrotic changes in the lungs and a mass in the right ventricular cavity of the heart (Figure 1). Echocardiography revealed an immobile mass, approximately 3 × 5 × 6 cm in diameter, in the right atrium that was obstructing the flow of the vena cava inferior. T-PA infusion was given to dissolve the thrombus, but the thrombus did not shrink. During surgery, a mass covering one-third of the cavity, extending into the vena cava inferior, was demonstrated at the right atriotomy (Figure 2A,B). The mass was excised from the right atrial wall. In the postoperative period, the patient could not be taken off

TABLE 1 Demographic/clinical data and laboratory findings

Demographic parameters	Case 1	Case 2
Sex (M/F)	F	F
Age (years)	80	78
Additional disease	Hypertension, DM	DM
ECG	Sinus rhythm	Sinus rhythm
Clinical findings	Palpitations, dyspnea, and lower limb edema	Transient ischemic attack
Taking anticoagulants	No	No
Cardiac thrombosis localization	Right atrium	Left atrium
White blood cell (μ /ml)	5810	7500
Hemoglobin (g/dL)	13.1	12.7
Platelet (μ /ml)	146	194
Ferritin (ng/ml)	331	351
Fibrinogen, mg/dL	583	516
D-Dimer, mg/L	5913	412
Troponin-I (pg/ml)	12.7	30
BUN (mg/dl)	33	41
Creatinine (mg/dL)	1	1.01
AST	34	82
ALT	21	76
Albumin (g/dL)	2.14	3.22
INR	1.52	1.48
CRP	27.1	8.6

Abbreviations: ALT, alanine aminotransferase; AST, aspartate transaminase; CRP, C-reactive protein; DM, diabetes mellitus; INR, international normalized ratio, LDH, lactate dehydrogenase.

the ventilator. Oxygen saturation was low and hemodynamic parameters were insufficient. The patient developed cardiopulmonary arrest on the fifth postoperative day and died.

2.2 | Case 2

A 78-year-old female patient was hospitalized with the diagnosis of COVID-19 3 months ago and the blood values at that time were as presented in Table 3. The thorax CT showed ground-glass opacity that indicated bilateral lung involvement. In addition to oxygen therapy, medical treatment was initiated for COVID-19 infection (enoxaparin, antiviral agent, hydroxychloroquine, levofloxacin, and methylprednisolone). The patient was closely monitored for any thrombotic and hypercoagulation; after 10 days of treatment, the patient's clinical parameters and blood values began improving and

TABLE 2 Blood parameters of the first patient at the time of COVID-19 infection

Parameters	Results
CRP (mg/dL)	51 (<3.11)
Sedimentation (mm/h)	33 (0–20)
Serum procalcitonin (ng/ml)	11 (0.5–2)
LDH (U/L)	188 (90–250)
WBC (μ /L)	3.0×10^3 ($3.9\text{--}10.8 \times 10^3$)
Lymphocyte count (μ l)	0.87×10^3 ($1.16\text{--}3.61 \times 10^3$)
Lymphocyte (%)	16.6 (18–47)
Platelet count (μ l)	106×10^3 ($145\text{--}345 \times 10^3$)
ALT (10–40 (U/L)	71
AST (15–42 (U/L)	77
After COVID-19 test (+)	
Ferritin (ng/ml)	891 (23.9–336.2)
Fibrinogen (mg/dL)	1056 (245–400)
D-Dimer (ng/ml)	1613 (0–500)
Oxygen saturation (%)	89–93

Abbreviations: ALT, alanine aminotransferase; AST, aspartate transaminase; COVID-19, coronavirus disease 2019; CRP, C-reactive protein; LDH, lactate dehydrogenase; WBC, white blood cell.

she was discharged. Three months later, the patient was admitted to the neurology clinic due to a transient ischemic attack. Echocardiography revealed a mobile mass in the left atrium (Figure 3), which was believed to be a thrombus. Coronary angiography was normal and the patient was given thrombolytic therapy to resolve the thrombus. However, the thrombus did not shrink and a thrombus excision by surgery was decided upon. The mobile mass in the left atrium was removed (Figure 4). The patient, who did not develop complications in the postoperative period, was discharged with Enoxaparin sodium 0.4 ml twice a day and then oral anticoagulant treatment 1 week later.

3 | DISCUSSION

The two patients we present in this article represent two different cases of intracardiac thrombus due to underlying COVID-19 infection. Conditions associated with vascular endothelial dysfunction—including advanced age, hypertension, diabetes, smoking, severe inflammatory response, lung parenchyma disease, hypoxia, additional vascular diseases, immobilization, and coronary artery disease—all have high thromboembolic risk and poor prognostic cardiovascular disease in patients with COVID-19.⁴ While the presence of HT and DM in our patients are considered effective thrombotic risk factors, the presence of COVID-19 infection increased the susceptibility to thrombotic processes in the subsequent period.

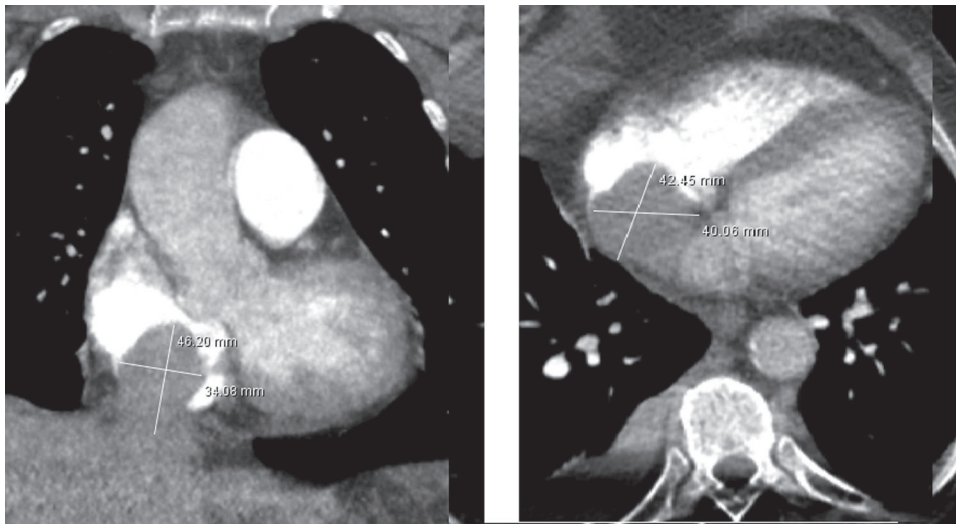


FIGURE 1 Thrombus in the right atrium, extending into the vena cava inferior

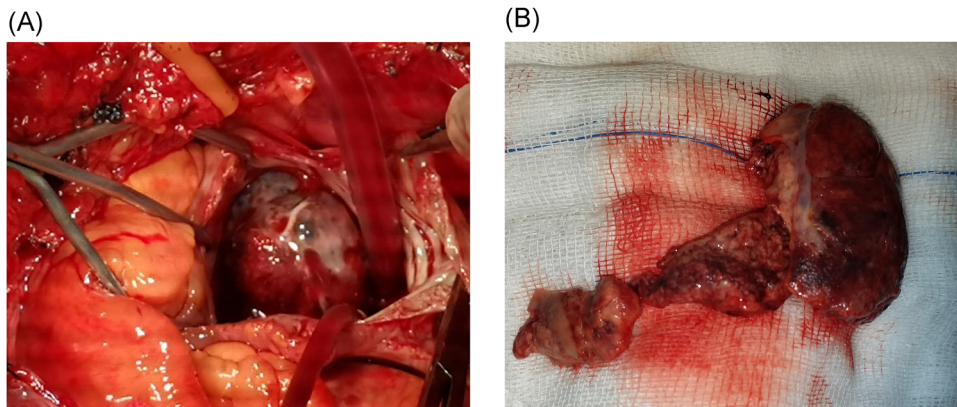


FIGURE 2 (A) Operational view of the thrombus in the right atrium. (B) Operational view of the thrombus extending into the vena cava inferior

It was found that thrombotic tendency was frequently detected in patients with COVID-19, which increased the severity of the disease and caused death. Pathophysiological mechanisms remain unclear, but may be associated with virus-induced endothelial dysfunction and immune activation.⁵ Inflammatory and coagulopathy markers—including ferritin, CRP, LDH, prothrombin time, and D-Dimer—were all elevated, thereby indicating a high risk of lung injury and thrombosis. Hematological and inflammatory parameters increased in active periods of infection in both the patients in our study. Involvement related to lung damage was observed and oxygen therapy was given in the intensive care unit. However, no thromboembolic event was observed in the early stages of infection, although the thrombotic parameters were high.

In numerous case series, high D-Dimer levels have been reported in 40%–45% of patients associated with disease severity and increased mortality.⁶ In our patients, D-Dimer levels increased in the first disease period, but the thrombotic process occurred after months. However, it is unclear when this thrombotic formation began

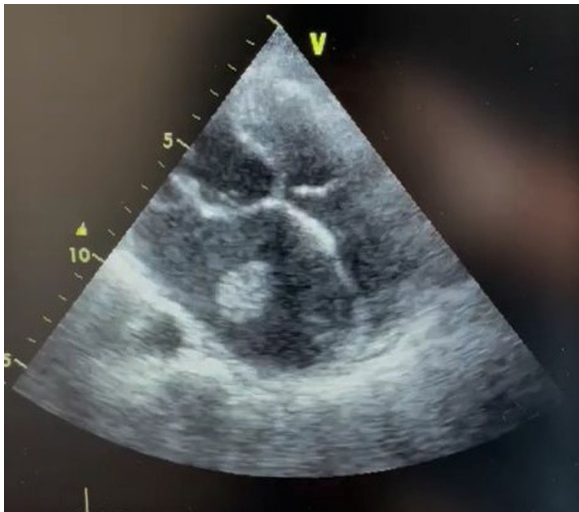
after the disease. Therefore, we believe that even if the D-Dimer values are close to normal and the COVID-19 test is negative, as in our second patient, anticoagulant therapy for a few months would be beneficial.

High D-Dimer levels are considered influential in the prognosis of patients. Although their effect on thrombosis and clot formation is debated, prophylactic anticoagulation protocols have been used in numerous hospitals.⁷ However, the role and doses of anticoagulants in preventing arterial and venous thromboembolism remain unknown. In the two patients included here, D-Dimer levels were found to be high during the period when they had COVID-19 infection. When the patients were discharged, they did not have any thromboembolic complaints and signs. However, during the periods when the intracardiac thrombus developed, D-Dimer values were very high in the first case, and D-Dimer values were not increased in the second case. Thus, we could not determine when the thrombosis occurred for either patient and at which D-Dimer values it caused thrombus. This result revealed that regardless of the range of D-Dimer levels, a

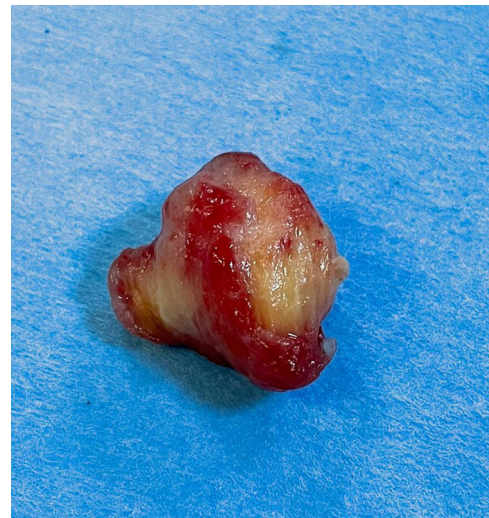
TABLE 3 Blood parameters of the second patient at the time of COVID-19 infection

Parameters	Results
CRP (mg/dL)	45 (<3.11)
Sedimentation (mm/h)	42 (0–20)
Serum procalcitonin (ng/ml)	14 (0.5–2)
LDH (U/L)	171 (90–250)
WBC (μ /L)	3.8×10^3 (3.9 – 10.8×10^3)
Lymphocyte count (μ l)	0.91×10^3 (1.16 – 3.61×10^3)
Lymphocyte (%)	15.4 (18–47)
Platelet count (μ l)	111×10^3 (145 – 345×10^3)
ALT (10–40 (U/L)	84
AST (15–42 (U/L)	79
After COVID-19 test (+)	
Ferritin (ng/ml)	501 (23.9–336.2)
Fibrinogen (mg/dL)	942 (245–400)
D-Dimer (ng/ml)	1277 (0–500)
Oxygen saturation (%)	85–90

Abbreviations: ALT, alanine aminotransferase; AST, aspartate transaminase; COVID-19, coronavirus disease 2019; CRP, C-reactive protein; LDH, lactate dehydrogenase; WBC, white blood cell.

**FIGURE 3** Echocardiography image of the thrombus in the left atrium

thrombotic process can occur in patients with COVID-19. Therefore, if D-Dimer levels increased during the first infection period, a thromboembolic prophylaxis must be conducted for a period of at least three months. However, when the thrombosis occurs remains unknown. Certain publications report that it can occur between 5 and 25 days.⁸ Ferritin, fibrinogen, and D-Dimer values increased during infection in both our patients, but a thrombotic condition did not occur. Thrombotic findings occurred in the second and third months

**FIGURE 4** Operational view of the thrombus in the left atrium

after the disease and required surgery. Although ferritin and fibrinogen values were approximately the same in the thrombotic event period in both cases, D-Dimer values were much higher in the thrombotic process that occurred in the second month in the first case study as compared with the second one. The first patient died in the hospital after the operation. This explains the prognostic importance of D-Dimer.

4 | CONCLUSION

To date, reports of intracardiac thrombosis in COVID-19 patients with normal cardiac anatomy and function are rare and may cause major cardiovascular complications. Therefore, patients are recommended standard prophylactic anticoagulation doses for at least 3 months during and after the illness. In addition, patients must follow up with monthly echocardiography to check for cardiac thrombus.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

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