

Left Ventricular Thrombus Formation in a COVID-19 Patient with a Complex Course of Pericarditis and Myocardial Infarction.

Jalil Golshani¹, Zahra Kalantari¹, Hassan Ahangar¹, Saman Ameri¹, Reza Madady¹, and Vahid Khosroshahi¹

¹Zanjan University of Medical Sciences

August 12, 2023

Left Ventricular Thrombus Formation in a COVID-19 Patient with a Complex Course of Pericarditis and Myocardial Infarction.

Abstract:

Background: Coronavirus Disease 2019 (COVID-19) has grown significantly in scale as a result of the development of the novel Zoonotic Severe Acute Respiratory Syndrome Coronavirus-2. Despite the fact that respiratory symptoms predominate, cardiovascular problems have also been reported. Pericarditis, inflammation of the pericardium, is the most prevalent pericardial disease and a prominent cause of acute chest discomfort in young people which is most commonly caused by viral infections. Individuals with COVID-19 may be hypercoagulable, which could accelerate the onset of left ventricular thrombus (LVT).

Case Presentation: A 38-year-old COVID-19 patient with ST elevation MI and LV thrombus and the signs and symptoms of pericarditis with no past medical history of any heart conditions was represented.

Conclusions: The case demonstrated that thrombotic complications such as coronary thrombosis and LV clot could occur even in COVID-19 patients with non-specific symptoms and gradually progressed and led to clot formation and coronary involvement. This indicates the mysterious face of COVID-19. This complex process highlights the necessity of screening patients for COVID-19 disease even with non-specific cardiac symptoms.

KEYWORDS: Pericarditis, Myocardial Infarction, Left Ventricular Thrombus Formation, COVID-19

Background:

Pericarditis, inflammation of the pericardium, is the most prevalent pericardial disease and a prominent cause of acute chest discomfort in young people (1). It is mostly caused by viral infections as well as rheumatic illnesses, uremia, or neoplasms (2). A pericardial friction rub, concave ST-segment elevations, pleuritic chest discomfort, and pericardial effusion are all common indications and symptoms (1). Given that pericarditis is generally self-limited, the therapy is usually supportive, including nonsteroidal anti-inflammatory medications or colchicine. Other causes of pericarditis, on the other hand, may have different prognosis and therapeutic implications (3, 4).

Up to 15% over all acute myocardial infarction (AMI) patients and 25% of individuals with anterior myocardial infarction suffer from a left ventricular thrombus (LVT) (5, 6). With the introduction of early percutaneous coronary intervention (PCI) as a widely used treatment of AMI, the incidence of LVT decreased dramatically. LVT is most typically seen in patients with late-onset MI in clinical practice. It is infrequently seen in patients who report early and receive fast revascularization, especially in the first 24 hours after an AMI (7).

Coronavirus Disease 2019 (COVID-19) has grown significantly in scale as a result of the development of the novel Zoonotic Severe Acute Respiratory Syndrome Coronavirus-2. In addition to the predominance of respiratory symptoms, cardiovascular problems have also been reported(8).

A COVID-19 patient with ST elevation MI and LV thrombus with signs and symptoms of pericarditis was introduced.

Timeline:

Timing	Event
-10 days	History of upper respiratory infections (URI)
Presentation	Pleuritic chest pain, nausea, diarrhea, and cough
+1 day	Pleuritic chest pain Electrocardiography: diffuse ST-segment elevation Echocardiography: reduced ejection fraction (LVEF:45%) Serum troponin: within normal limits
+2 day	Increased pleuritic chest pain positive Real-time RT-PCR test ECG: Increased ST-segment elevation in precordial leads Echocardiography: left ventricle (LV) clot and a decreased EF (35%) Serum troponin(hsTn): 27185 (normal range: 34(
+1 month	Angiography: heavy thrombotic lesion on the left anterior descending artery (LAD) Resolution of chest pain Electrocardiography: T wave inversion in anterior and lateral leads, ST elevation in inferior leads and Q wave in lateral leads Computed Tomography Angiography (CTA): reduction of thrombus burden
+3 month	chest pain relieved stable vital signs Echocardiography: reduced-sized LV clot EF=45-50%

Case Presentation:

A 38-year-old man with a history of URI 10 days before admission and no risk factor for cardiac disease was presented with pleuritic chest pain that radiates to the scapula and shoulders, exacerbated by deep inspiration and in supine position, nausea, diarrhea and cough starting three days prior to admission.

Physical examinations; vital signs included blood pressure of 110/70 mmHg, pulse rate of 87 beats/min, respiratory rate of 23/min, oximetry of 90% on room air, and a temperature of 38.5 °C (101 degF).

The patient's chest pain improved on the first day of admission and electrocardiography revealed diffuse ST-segment elevation (Figure 1).

Echocardiography showed a reduced ejection fraction (LVEF=45%) and the serum troponin (hsTn) was equal to 36 (normal range: 34). An initial plan for medical treatment of acute pericarditis was in place when the patient was admitted to the hospital.

Infectious diseases consultant added Vancomycin 2gr twice a day and Meropenem 1gr three times a day to the treatment.

A spiral lung CT scan showed no evidence in favor of covid and infectious diseases.

Laboratory evaluations such as viral, inflammatory, and coagulation markers were normal. All hypercoagulability tests (activated protein C resistance, beta-2 glycoprotein antibodies, protein S activity, and

homocysteine) were normal.

On the next day of admission, the patient's pleuritic chest pain changed to typical pain and felt as a feeling of pressure. In addition, serum troponin reached 27185, and SARS-CoV-2 PCR became positive.

ECG showed diffuse ST-segment elevation and increased ST elevation in precordial leads (Figure 2).

Echocardiography indicated a large left ventricle (LV) apical clot size (3.1cm*1.5cm) and moderate LV systolic dysfunction (LVEF 35%).

Due to the rise of serum troponin and LV clots, an intravenous injection of unfractionated heparin was started and the patient underwent angiography. The results disclosed a heavy thrombotic lesion on the left anterior descending artery (Figure 3).

On the 7th day of admission; the patient's symptoms disappeared gradually, Anticoagulation was switched to Rivaroxaban, and the patient was discharged with aspirin 80 mg, clopidogrel 75mg, and rivaroxaban 15 mg once a day.

During a one-month follow-up, electrocardiography showed T wave inversion in anterior and lateral leads, ST elevation in inferior leads, and Q wave in lateral leads (Figure 4). CTA revealed reduction of thrombus burden in left anterior descending artery (Figure 5).

Based on clinical status and echocardiographic findings, continuation of rivaroxaban 15 mg daily for three months was prescribed.

Additionally, after three months, echocardiography detected a reduced-sized LV clot (2.2cm*0.7cm) with an increased LV systolic function (Figure 6).

In addition, the patient had a favorable evolution and showed stable vital signs, pain improvement, no angina, no dyspnea, and good work ability.

Discussion:

The patient in this case report was recovering from COVID-19 infection when he developed acute pericarditis symptoms, elevated cardiac injury biomarkers, and LV clots.

Pericarditis is an inflammatory and non-ischemic condition that affects the pericardium (1, 9). The clinical appearance varies greatly and may be preceded by coryzal symptoms or generalized symptoms including lethargy, diarrhea, or malaise. Symptomatic arrhythmias, heart failure, myocardial infarction, cardiogenic shock, or sudden cardiac death may be caused by cardiac inflammation (10). Pleuritic chest discomfort, pericardial friction rub, and suggestive abnormalities in electrocardiography are the clinical hallmarks of acute pericarditis (4). The most frequent cause is infection, with viral infections being the most frequently recognized in developed nations (11). Various cardiovascular conditions, such as myocarditis, pericarditis, or more generally, elevated cardiac injury biomarkers, have all been reported as cardiac involvement in COVID-19, according to a growing body of research (12). Acute coronary syndrome may be caused by localized inflammation at the plaque level, which could destabilize coronary atheroma (13).

Our case demonstrated thrombotic complications such as coronary thrombosis. LV clots can occur even in COVID-19 patients with non-specific symptoms, and gradually progress and lead to clot formation and coronary involvement, which shows the mysterious face of COVID-19.

This complex process indicates the necessity of screening patients for COVID-19 disease even with non-specific cardiac symptoms (14).

Moreover, the early initiation of antiviral treatment and most importantly, the initiation of anticoagulants after diagnosis will stop the progressive course of the disease.

These patients are at risk of cardiac re-thrombosis and cerebral thrombosis. Therefore, it is recommended to continue anticoagulant treatment for at least three months (15).

Conclusions:

Our case demonstrated that thrombotic complications such as coronary thrombosis and LV clots can occur even in well-appearing, COVID-19 patients with non-specific symptoms which indicates the necessity of screening for COVID-19 disease.

Further studies are also required to analyze the role of routine anticoagulation upon the diagnosis of COVID-19 and preventing further threatening complications.

List of Abbreviations:

- AMI Acute Myocardial Infarction
- COVID-19 Coronavirus Disease 2019
- CTA Computed Tomography Angiography
- ECG Electrocardiogram
- hsTn High-sensitivity Troponin
- LVEF Left Ventricular Ejection Fraction
- LVT Left Ventricular Thrombus
- RT-PCR Real Time Polymerase Chain Reaction
- URI Upper Respiratory Infection

References:

1. Imazio M, Gaita F, LeWinter M. Evaluation and Treatment of Pericarditis: A Systematic Review. *Jama*. 2015;314(14):1498-506.
2. Little WC, Freeman GL. Pericardial disease. *Circulation*. 2006;113(12):1622-32.
3. Troughton RW, Asher CR, Klein AL. Pericarditis. *Lancet*. 2004;363(9410):717-27.
4. Salarda EM, Ocazonez-Trujillo D, Prakash SK. An unusual cause of acute pericarditis: a case report. *Eur Heart J Case Rep*. 2021;5(2):ytaa535.
5. Solheim S, Seljeflot I, Lunde K, Bjornerheim R, Aakhus S, Forfang K, et al. Frequency of left ventricular thrombus in patients with anterior wall acute myocardial infarction treated with percutaneous coronary intervention and dual antiplatelet therapy. *Am J Cardiol*. 2010;106(9):1197-200.
6. Delewi R, Nijveldt R, Hirsch A, Marcu CB, Robbers L, Hassell ME, et al. Left ventricular thrombus formation after acute myocardial infarction as assessed by cardiovascular magnetic resonance imaging. *Eur J Radiol*. 2012;81(12):3900-4.
7. Sharma H, George S. Early Left Ventricular Thrombus Formation in a COVID-19 Patient with ST-Elevation Myocardial Infarction. *Case Rep Cardiol*. 2020;2020:8882463.
8. Panigada M, Bottino N, Tagliabue P, Grasselli G, Novembrino C, Chantarangkul V, et al. Hypercoagulability of COVID-19 patients in intensive care unit: A report of thromboelastography findings and other parameters of hemostasis. *J Thromb Haemost*. 2020;18(7):1738-42.
9. Caforio AL, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J*. 2013;34(33):2636-48, 48a-48d.
10. Eckart RE, Shry EA, Burke AP, McNear JA, Appel DA, Castillo-Rojas LM, et al. Sudden death in young adults: an autopsy-based series of a population undergoing active surveillance. *J Am Coll Cardiol*. 2011;58(12):1254-61.
11. Breinholt JP, Moulik M, Dreyer WJ, Denfield SW, Kim JJ, Jefferies JL, et al. Viral epidemiologic shift in inflammatory heart disease: the increasing involvement of parvovirus B19 in the myocardium of pediatric

cardiac transplant patients. *J Heart Lung Transplant*. 2010;29(7):739-46.

12. Buckley BJR, Harrison SL, Fazio-Eynullayeva E, Underhill P, Lane DA, Lip GYH. Prevalence and clinical outcomes of myocarditis and pericarditis in 718,365 COVID-19 patients. *Eur J Clin Invest*. 2021;51(11):e13679.

13. Madjid M, Safavi-Naeini P, Solomon SD, Vardeny O. Potential Effects of Coronaviruses on the Cardiovascular System: A Review. *JAMA Cardiol*. 2020;5(7):831-40.

14. Bhurint Siripanthong, Recognizing COVID-19-related myocarditis Heart Rhythm. 2020 Sep;17(9):1463-1471. Doi: 10.1016/j.hrthm.2020.05.001.

15. Chandra A, Chakraborty U, Ghosh S, et al Anticoagulation in COVID-19: current concepts and controversies *Postgraduate Medical Journal* 2022;98:395-402.

Figure Legends

Figure 1

Electrocardiogram presenting diffuse ST-segment elevations.

Figure 2

ECG showing diffused ST-segment elevation, and increased ST elevation in precordial leads.

Figure 3 (a,b)

Angiogram showing non-occlusive and occlusive thrombus in the left anterior descending artery.

Figure 4

Electrocardiogram at 1 month showing T wave inversion in anterior and lateral leads, ST elevation in inferior leads and Q wave in lateral leads.

Figure 5

CT-Angiogram revealed reduction of thrombus burden.

Figure 6

Echocardiogram indicating a reduced-sized LV clot (EF=45-50%).

Hosted file

Pericarditis Figure File TIFF.docx available at <https://authorea.com/users/651933/articles/659661-left-ventricular-thrombus-formation-in-a-covid-19-patient-with-a-complex-course-of-pericarditis-and-myocardial-infarction>