Case Report

Post COVID-19 hemorrhagic pericardial effusion; A case report with literature review

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ABSTRACT

Introduction: Pericardial effusion (PE) related to COVID-19 has rarely been observed, with most reported cases being non-hemorrhagic. This study aims to present a rare case of post-COVID-19 hemorrhagic PE.

Case report: A 44-year-old male presented with shortness of breath upon exertion, palpitation, and left-sided chest tightness. He was conscious, oriented, tachypneic, and tachycardic. Chest examination revealed a mixture of fine and coarse crackles along with muffled heart sounds. He had elevated D-dimer, C-reactive protein, prothrombin time, and aPTT. Computed tomography pulmonary angiogram showed acute pulmonary thromboembolism involving the posterior segmental lobar branch of the left lower lobe with concomitant pulmonary infarction. Echocardiography showed severe PE without diastolic collapse. Pericardial drainage was performed and by the 2nd day, there was no more effusion. On the 7th day, the patient developed severe complications which led to cardiac arrest.

Discussion: PE is a collection of fluid in the pericardium. It has been rarely observed in relation to COVID-19, both during and after the viral infection. If PE is suspected, Echocardiography can be used to confirm its diagnosis. There is no standard management for these cases and only non-hemorrhagic patients with mild to moderate effusion can be treated using conservative measures.

Conclusion: Hemorrhagic PE can be a rare but possible post-COVID-19 sequel, and echocardiography can be used to confirm its diagnosis. Drainage is necessary to resolve the effusion.

1. Introduction

In late 2019, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that caused novel coronavirus 2019 disease (COVID-19) initially appeared in Wuhan, China [1]. Soon after its emergence, the viral infection was recognized as a global pandemic by the World Health Organization (WHO) on March 11, 2020 [2]. Even though COVID-19 cases usually have mild or no symptoms, a more life-threatening condition called acute respiratory distress syndrome (ARDS) can develop in hypertensive, elderly, and diabetic patients [3]. Despite COVID-19 being initially considered as a pulmonary disease, it has been reportedly associated with a wide spectrum of systemic complications - both during and after the resolution of COVID-19 [4,5]. It is suggested that these systemic disorders are the result of proinflammatory effects, activation of coagulation pathways, and endothelial cell damage which are caused by SARS-CoV-2 [6]. Recent studies have revealed cardiovascular involvement in COVID-19, with the most common manifestations being acute coronary syndrome, myocarditis, myocardial infarction, arrhythmias, and heart failure [7]. On rare occasions, pericardial effusion (PE) has also been documented in association with COVID-19, in which it is mostly non-hemorrhagic [8,9].

The current study aims to present a rare case of post-COVID-19 hemorrhagic PE, with a brief review of the literature. In the writing of this paper, SCARE guidelines were followed [10].

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2. Case presentation

2.1. Patient information

A 44-year-old male working as a construction worker presented with shortness of breath upon exertion, palpitation, and left-sided chest tightness. He had no relevant medical history. However, he tested positive for COVID-19 four weeks prior to his complications. He recovered from COVID-19 within 2 weeks of symptoms via home medications and supplements. They included the followings: vitamin D 1000 IU, vitamin C 500 mg, zinc tab 50 mg, paracetamol tab 1000 mg, and enoxaparin 4000 IU every 12 hours.

2.2. Clinical findings

Upon further clinical evaluation, the patient was conscious, oriented, tachypneic with a respiratory rate of 34 b/m, tachycardic with a pulse rate of 130–140 b/m, and had a blood pressure of 130/82 mmHg. Chest examination revealed a mixture of fine and coarse crackles. He had muffled heart sounds (S1 and S2) with no added sound. Systemic examination was normal.

2.3. Diagnostic approach

His COVID-19 recovery were confirmed via negative RNA-PCR testing. During his active infection, he had an initial D-dimer of more than 1000 ng/mL, and his electrocardiogram (ECG) showed sinus tachycardia. In addition, high-resolution computed tomography (CT) scan revealed bilateral, multifocal, and ill-defined ground-glass opacification (GGO), mainly involving the lower lobes with absence of PE (Fig. 1).

His laboratory findings showed normal hemoglobin levels (14.6 g/dL), elevated white blood cell (WBC) count (15.7*10^9/L), normal platelet count (243*10^3/μL), and creatinine level (0.57 mg/dL), highly elevated D-dimer (4733 ng/mL), mildly elevated C-reactive protein (CRP) level (1.84mg/dL), increased prothrombin time (17 seconds) and aPTT (50 seconds), and an INR of 1.4. CT pulmonary angiogram revealed acute pulmonary thromboembolism involving the posterior segmental lobar branch of the right lower lobe with concomitant pulmonary infarction (Fig. 2). Echocardiography (Echo) and CT showed severe PE without diastolic collapse (Fig. 3).

2.4. Therapeutic intervention

The patient was transferred to the intensive care unit (ICU) for necessary treatment. Pericardial drainage was performed. Within 18 hours, 900 CC of blood was drained (through 11 French central venous line). The result of the fluid analysis showed low glucose and protein, with full of RBC. On the second day, Echo showed no more effusion, and the drain was removed.

2.5. Follow-up and outcome

In the ICU, the patient developed severe thrombocytopenia, minor oral bleeding, and hemoptysis. In the subsequent days, he developed more tachypnea and desaturation. On the 7th day, he suddenly developed severe chest pain, and within a few minutes, developed cardiac arrest and died.

3. Discussion

As the global pandemic of COVID-19 progressed and more people got infected with the virus, associated extra-pulmonary complications have become increasingly reported.

Meanwhile, cardiac involvement of various types has been observed throughout the literature [11]. PE, which is the buildup of fluid in the pericardium, is one of the cardiovascular complications of COVID-19 which has rarely been reported - both during and after the viral infection [12,13]. According to the existing literature, most reports of COVID-19 related PE were non-hemorrhagic, with only a few hemorrhagic cases being present [9,14]. This makes COVID-19 the second viral infection that can lead to hemorrhagic PE, in addition to Coxsackievirus [15].

The most frequent etiology of PE in developing countries is thought to be viral infections; however, tuberculosis tends to be a more common etiologic agent [13]. Even though the exact pathologic mechanism of PE due to COVID-19 is yet to be known, it has been suggested that SARS-CoV-2 can trigger an excessive systemic inflammatory response and induce cytotoxic effects which in turn can lead to injury in many organs of the body, including the heart and its surrounding tissues [11]. Normally, the pericardium contains up to 50 mL of pericardial fluid; however, in PE, capillary leakage can substantially increase the amount of fluid to up to 2000 mL in severe conditions [7].

Generally, the previous cases of COVID-19 associated PE presented with dyspnea, chest pain, and orthopnea [13,16]. Amongst these cases,

Fig. 1. Axial section of high-resolution computed tomography of the chest showing patch of ground glass opacity in the apical segment of the left lower lobe.
most of them were reported in positive COVID-19 patients and usually presented as a late finding [17]. However, PE as a post-COVID-19 complication has been observed only a few times in the literature [18, 19]. Tachycardia, tachypnea, raised jugular venous pressure, muffled heart sound, pulsus paradoxus, and hypotension are important indicators of PE upon clinical examination [17].

There are currently no specific blood biomarkers for the diagnosis of PE; however, it is often associated with elevated inflammatory markers, including CRP, WBC, D-dimer, and erythrocyte sedimentation rate [16]. If PE is suspected, Echo can be used to confirm its diagnosis which shows electrical alternans, low QRS voltage, and tachycardia; it can also be used to estimate the PE size [7]. Nonspecific changes in ST-T and elevation of concave diffuse ST with PR depression can indicate myocarditis and pericarditis, respectively. Similar results were noted in this study. In addition, on chest CT examination of COVID-19 cases, PE can be an incidental finding [17].

To date, there is no standard management approach regarding PE that is related to COVID-19 [9]. Medications such as nonsteroidal anti-inflammatory drugs (NSAID), colchicine, and steroids, are used in the treatment of the disease. However, their effectiveness is generally dependent upon the severity of the disease, as only cases with mild to moderate effusion can be treated using these medications [11]. Meanwhile, the use of NSAIDs is not recommended during active COVID-19 infection as it can worsen the condition [13]. Medications are ineffective to relieve large or hemorrhagic PE; hence, both require drainage to rapidly resolve the effusion, either through percutaneous
pericardiocentesis or surgical drainage which can be guided by Echo [17].

In conclusion, hemorrhagic PE can be a rare but possible post-COVID-19 complication. Upon suspicion, Echo can be used to confirm the diagnosis of the condition. Drainage is necessary to resolve the effusion as medications are ineffective. It is important to consider post-COVID-19 extrapulmonary conditions even if the respiratory symptoms have subsided so that early diagnosis and treatment can be performed.

Ethical approval

The manuscript approved by ethical committee of the University of Sulaimani.

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Author contributions

BNar J. Hama Amin: physician managing the case, final approval of the manuscript.

Fahmi H. Kakamad, Razhan K. Ali, Bestoon KH. Salih, Muhammed Gh. Hamasaeed, Ahmed G. Hamasaeed: literature review, writing the manuscript, final approval of the manuscript.

Abdulwahid M. Salih: major contribution of the idea, literature review, final approval of the manuscript.

Registration of research studies

registration is not necessary for case report.

Guarantor

Fahmi Hussein Kakamad is Guarantor of this submission.

Consent

Consent has been taken from the family of the patients for publication of this report.

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Declaration of competing interest

There is no conflict to be declared.

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