



INTRA CARDIAC FREE-FLOATING MICROTHROMBI AND PERICARDITIS IN THE SETTING OF COVID 19: AN UNUSUAL MIX!

Cardiology

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ABSTRACT

Background: The coronavirus disease 2019 (COVID-19), which is caused by severe acute respiratory syndrome coronavirus 2 (sarscov2), has been shown to cause conventional respiratory distress, in addition to that, there has been recent reports suggesting the cardiovascular system involvement during the course of the infection, including microthrombosis and cardiopulmonary serosal layers infection and inflammation although being separately described. The present paper demonstrates the first reported case of the concomitant presence of intra-cardiac free-floating microthrombi and pericardial effusion secondary to COVID-19 infection, beyond the casual clinical presentation.

Case report : A 51 year-old man with no medical history, presented to the emergency department, with a progressively worsening dyspnea, he had an oxygen saturation of 85% on first medical contact. The chest radiography and the baseline ECG showed respectively multifocal bilateral patchy opacities and a sinus tachycardia with low QRS voltage and t wave inversion in all leads. On further assessment, the echocardiography revealed the presence of free-floating microthrombi in the right atrium and a moderate pericardial effusion. Biological findings and pulmonary computed tomography were suggestive of sarscov2 infection. Interestingly, there was no evidence of pulmonary embolism. The patient, thus, received heparin therapy and colchicine. We noted a rapid improvement in the following seven days, the hospital discharge was, hence, deemed warranted.

Conclusions: This case highlights an unusual presentation of COVID-19 infection, the diagnostic and therapeutic challenges we are facing in this setting. Moreover, it raises the question about the emergent need of a therapeutic regimen, in order to better manage this unique condition and a fortiori mitigate the COVID-19 complications.

KEYWORDS

microthrombi, covid-19, Pericardial effusion, anticoagulation

INTRODUCTION:

Since December 2019, Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is responsible for the global pandemic of coronavirus disease 2019 (COVID-19). Around 14% of cases are reported to have severe symptoms and 5% of patients developed respiratory failure, shock, and multi-organ failure (1).

Hypercoagulopathy has been strongly demonstrated in COVID-19 patients, being attributed to a myriad of pathogenesis mechanisms namely: endothelial injury, stasis, and coagulation abnormalities such as elevated fibrinogen, D-dimer, factor VIII activity, and von Willebrand factor (VWF) antigen. (2).

The pathophysiological mechanism of myocardial injury in COVID-19 is still unclear, but it is hypothesized that hypoxia, hemodynamic instability, systemic inflammation and micro-thrombosis may be causative events. (3) Conversely, very few attention has been paid to pericardial effusion (PE) in the setting of COVID-19. Presumably rare, only very few case reports described PE, revealed by chest pain or a deterioration of general condition. (4)

To our knowledge, this is the first case describing the uncommon presentation of free-floating intracardiac microthrombi and viral pericarditis, induced both by the infection of sarscov2, thus demonstrating the challenges of its management, and the uncertain prognosis it clearly confers.

CASE REPORT :

A 51 year-old man with 15-day history of dry cough, dyspnea and trepopnea, was admitted during the COVID-19 outbreak, to the emergency department. The patient claimed having no history of other preexisting pathological condition. At the first physical exam, he was tachypneic, hypoxic with an oxygen saturation of 85% on room air, we also noted muffled cardiac sounds and an ewart sign.

Chest radiograph demonstrated multifocal bilateral patchy opacities and the electrocardiogram showed a sinus rhythm at 112 bpm, a low QRS voltage and t wave inversion in all leads. A point-of-care ultrasonography was performed, revealing the presence of a moderate circumferential pericardial effusion with porridge-like appearance (Figure 1), combined with bilateral pleural effusion. It also demonstrated the presence of free-floating microthrombi in the right atrium (Figure 2).

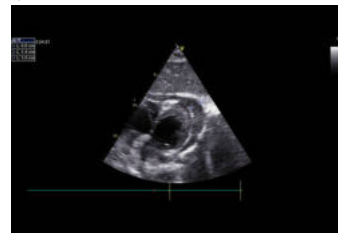


Figure 1: transthoracic echocardiogram subcostal view visualizing a porridge-like pericardial effusion



Figure 2: Transthoracic echocardiogram: Free-floating Microthrombi running through the right atrial, evidenced on subcostal 4-chamber view.

Computed tomography (CT) pulmonary angiogram demonstrated bilateral peripherally located ground-glass opacities without evidence of pulmonary embolism.

Laboratory tests revealed leukopenia, thrombocytopenia, elevated d-dimers and fibrinogen, raised markers of inflammation, and negative troponin test results. Serologies (Human Immunodeficiency Virus, Hepatitis B Virus, Hepatitis C Virus) were all negative. Tuberculosis testings have been performed to rule out a mycobacterium tuberculosis infectious disease.

Although, Combined nasopharyngeal and oropharyngeal swabs were taken and tested negative for SARS-CoV-2, clinical symptoms, typical COVID-19 findings at chest CT, and epidemiological criteria were highly suggestive of COVID-19. Therefore, we completed by blood serology that revealed positive SARS-CoV-2 IgG.

Treatment with colchicine 0.5 mg once a day and anticoagulation with low molecular weight heparin (LMWH) were initiated on day 2, we then observed a rapid improvement based on clinical, biological and echocardiographic criterias.

DISCUSSION :

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has presented a health emergency of unprecedented magnitude. There is mounting evidence regarding the cardiovascular system affections in the setting of COVID-19: acute cardiac injury, high risk of thrombosis including stroke, pulmonary embolism, and acute coronary syndrome.(4)

Similarly to other viral infections, when COVID-19 elicits an inflammatory response, it is reasonable that this may lead to pericarditis and pericardial effusion (5). Nonetheless, very few attention has been paid to pericardial effusion (PE) (4). On the other hand, hypercoagulability complicating COVID-19 has been well described.

To our knowledge, this is the first reported case of an association of pericarditis and intracardiac microthrombi.

Some emerging case reports suggest that critically all patients with COVID-19 develop complications from hypercoagulability (6), including both pulmonary emboli (7) and microscopic thrombi (8). A coagulopathy has been reported in up to 50% of patients with severe COVID-19 manifestations (9). But, Only very few cases of PE, in patients having COVID-19, have been reported (4).

The dysfunction of endothelial cells induced by infection and hypoxia found in severe COVID-19 can stimulate thrombosis not only by increasing blood viscosity but also through a hypoxia-inducible transcription factor-dependent signaling pathway (10).

At the present time, among the benchmarks of COVID-19 therapy are antiaggregant/ anticoagulants as well as antivirals/ immunomodulating agents (11).

The International Society on Thrombosis and Haemostasis recommends that all hospitalized COVID-19 patients should receive a prophylactic dose of LMWH unless they have contraindications (active bleeding and a platelet count $<25 \times 109/L$).(12) Many centers recommend intermediate dose or even therapeutic intensity anticoagulation guided by the levels of D-dimer and fibrinogen, but whether this will improve outcomes is still unknown(13).

Limited data suggest a high incidence of deep vein thrombosis and pulmonary embolism in up to 40% of patients, despite the use of a standard dose of low-molecular-weight heparin (LMWH) in most cases (9). Adapted to the individual risk of thrombosis and the d-dimer value, higher doses can be considered, especially since bleeding events in COVID-19 are rare. Besides the anticoagulant effect of LMWH, non-anticoagulant properties such as the reduction in interleukin 6 release have been shown to improve the complex picture of coagulopathy in patients with COVID-19 (9).

So, an individualized patient-based (“tailored”) approach is recommended, aimed at balancing the risk/benefit ratio of the various antithrombotic strategies, taking into account his/her underlying hypercoagulable state (11).

Conversely, when pericarditis adds up to the clinical picture, the management is more challenging. Albeit Most patients with acute pericarditis recover in 2–4 weeks with supportive measures comprising, non-steroidal anti-inflammatory drugs (NSAIDs), colchicine and with treating the causative disease, applying this to a patient with COVID-19 requires balancing this conventional approach with an emerging understanding of pharmacotherapy in COVID-19 (14).

Currently, our understanding of the spectrum of clinical illness of COVID-19 is limited. We certainly will be facing up with challenging conditions in terms of management, as in our case, with a unique clinical presentation, the association of acute pericarditis and intracardiac microthrombi.

CONCLUSION:

The propensity for thrombosis has been well documented in the setting of covid 19, nonetheless, the combination of free floating intracardiac microthrombi and pericardial effusion offers a new insight into the variety of complications of Covid 19, and raises the question about the therapeutic dilemma we practitioners are facing, in taking care of this subset of patients.

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