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PACEMAKER LEAD-ASSOCIATED PULMONARY EMBOLISM IN PATIENT WITH COVID- 19 INFECTION

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Abstract

Coronavirus disease is a novel severe acute respiratory syndrome, caused by RNA coronavirus (SARS-CoV-2) originating from Wuhan, China, and with the development of global pandemic state. It was declared a pandemic in February of 2020 by the World Health Organization's Director-General, dubbing it COVID-19. Mounting cases and deaths pose a major challenge for health care systems all over the globe There have been several reported cases, confirming that COVID-19 is associated with abnormal coagulation parameters and hypercoagulable state with increased risk of both venous and arterial thromboembolism. Some of the other conditions that are associated with higher risks for developing thromboembolic disease include diabetes mellitus, obesity, metabolic disease, use of oral glucocorticoids, and cardiac implantable electronic devices. This case describes clinical deterioration in a patient, who developed pulmonary embolism with pacemaker lead-associated thrombus complicated by acute right-sided heart failure 2.5 months after COVID-19 infection. With presentation of this clinical case we emphasize that in management of post-covid patients, it is highly essential to evaluate other possible hypercoagulable states and to provide personalized thromboprophylaxis strategies. Our case highlights the importance of long-term anticoagulation therapy in comorbid high-risk patients with device lead in cardiac chambers.

Keywords: *COVID-19, coronavirus disease, coagulation abnormalities, implanted cardiac pacemaker, pulmonary embolism.*

Case presentation

A 71 years old female was admitted to the emergency department of University Hospital 1 with complaints of shortness of breath and two episodes of loss of consciousness.

The patient had Past Medical History of arterial hypertension, chronic heart failure, diabetes mellitus, chronic kidney disease, and an implanted dual chamber pacemaker five years prior due sick sinus syndrome.

She was tested positive from nasopharyngeal swab by polymerase chain reaction (PCR) and hospitalized for Covid-19 two months preceding admission, and passed treatment for polysegmental bilateral Covid-19 pneumonia with corticosteroids and anticoagulant administration. After discharge, she was advised to continue one month of anticoagulant therapy with rivaroxaban 15 mg, but she ended her treatment prematurely without consulting with physicians.

Investigation

On admission, the patient was hemodynamically stable. She was afebrile and her arterial oxygen saturation was 97% on two liters of supplemental oxygen. Physical examination was unremarkable, besides lung auscultation demonstrating coarse crackles throughout lower lung fields bilaterally.

ECG revealed normal sinus rhythm with left axis deviation, premature atrial complexes and

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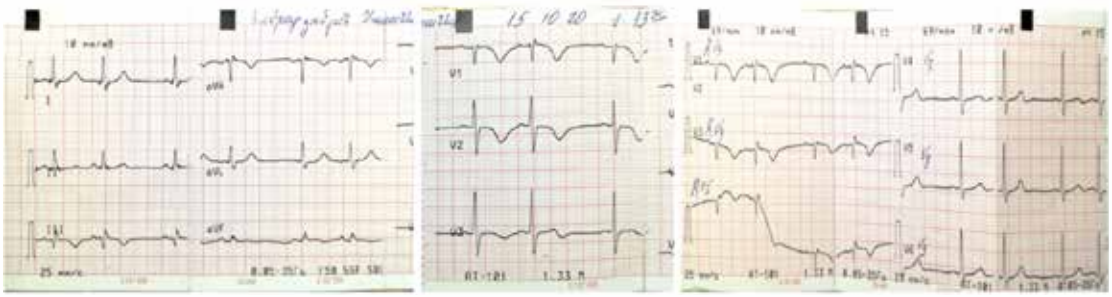

***Figure** 1 .ECG performed on admission. Normal sinus rhythm with left axis deviation, premature atrial complexes and negative T waves in leads III and aVF.*

negative T waves in leads III and aVF. (Fig. 1). Troponin T-was 0.061 on admission and increased to 0.094 *ng/ml* after six hours.

Initial blood workup showed leukocytosis of (12.55 *K/μL*), elevated C-reactive protein (14.134 *mg/l*). SARS-COV-2 RNA test was negative.

Bedside transthoracic echocardiography revealed right ventricular and atrial dilatation with reduced function associated with severe tricuspid and pulmonary regurgitation. Pulmonary hypertension was present with P max estimated 45+15 *mmHg*. Concentric left ventricular hypertrophy with ejection fraction of 40-45 % was found.

In the right atrium, a 2.3 *cm* hyper and isoechoic mass was found on pacemaker lead, which raised suspicion of thrombus. (Fig. 2).

Transesophageal echocardiography was performed in Cardiac Care Unit, which confirmed the pacemaker lead-associated thrombus in the right atrium. (Fig. 3).

Chest Computed Tomographic Angiography (CTA) showed large filling defects in bilateral central and proximal segmental pulmonary arteries and a linear saddle pulmonary embolus (Fig. 4) confirming diagnosis of bilateral pulmonary embolism.

Management and follow up

The patient was admitted to the cardiac care unit and her hemodynamic state intensively was monitored. Intravenous infusions of furosemide and therapeutic anticoagulation with heparin were administered. The patient required continuous nasal cannula oxygenation during her stay. Her condition gradually improved over the next week. Heparin was switched to 30 *mg* rivaroxaban daily for the next three weeks, after which it was reduced to 20 *mg* with outpatient monitoring.

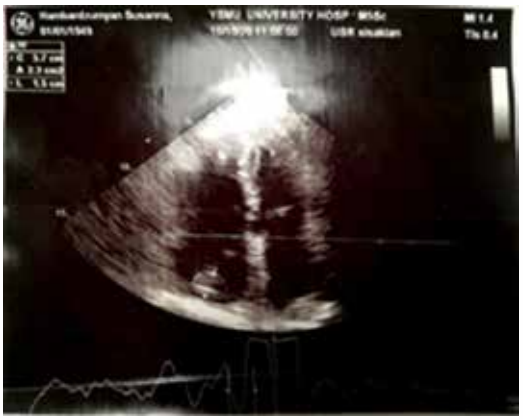

***Figure** 2. Transthoracic echocardiography showing a 2.3 cm hyperechogenic mass in the right atrium, on pacemaker lead.*

On her first follow-up visit after discharge, the patient remained asymptomatic, with no further episodes of breathlessness or weakness. On follow-up visit 1 month later, transesophageal echocardiography was repeated (Fig. 5) on anticoagulant treatment. No evidence of any thrombotic masses was found.

Discussion

Since the start of the COVID-19 pandemic, discussions within the medical community have been opened to focus on preventive anticoagulation to reduce the risk of thromboembolic events in such patients. Reports and studies have shown a higher risk of thromboembolism in hospitalized COVID-19 patients; though some guidelines/recommendations do exist for hospitalized patients,

To overcome it is possible, due to the uniting the knowledge and will of all doctors in the world

there is limited data and no recommendations related to thromboprophylaxis for high risk patients post-discharge.

COVID-19 induced hypercoagulation is a common clinical complication particularly in the presence of severe respiratory disease and inflammation, which can act as a triggering risk factor for VTE [*Zhou F et al., 2020*]. Deteriorating respiratory status in post-COVID-19 patients should alarm physicians to look for signs of PE.

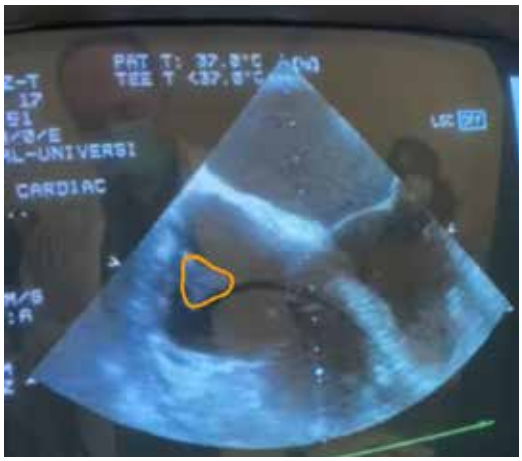

Figure 3. *Transesophageal echocardiography confirming the pacemaker lead-associated thrombus in the right atrium.*

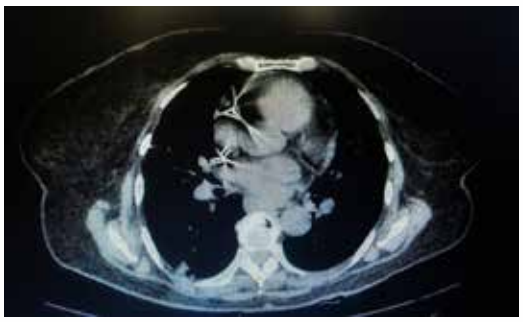

Figure 4. *Chest CTA showing large filling defects in bilateral central and proximal segmental pulmonary arteries and a linear saddle pulmonary embolus.*

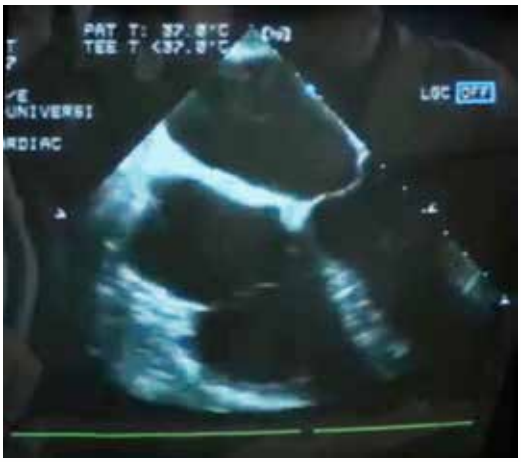

Figure 5. *Transesophageal echocardiography performed on follow-up visit. No evidence of any thrombotic mass was found.*

The suggested mechanisms for COVID-19–induced thrombosis include a disease-specific hypercoagulable state, cytokine-mediated diffuse microvascular damage and, in some instances, reactive thrombocytosis. Diabetes, obesity and immobility due to hospitalization can increase the risk of thrombosis and pulmonary embolism [*Levi M et al., 2020*]. Furthermore, Cardiac implantable electronic devices are known to increase the risk of thrombosis and pulmonary embolisms [*Pedersen S B et al., 2017; Saint S, Chopra V,2018]* . An endocardial lead acts as a foreign body in contact with the bloodstream and has a propensity to form thrombus. Intracardiac pacemaker lead-associated thrombosis may present with a variety of non-specific symptoms and can therefore be easily missed.

Occurrence of venous thromboembolism in COVID-19 patients is 20%-40% in the intensive care unit (ICU) and is 3%-8% in non-ICU, even when preventive anticoagulation is provided [*Klok FA et al., 2020*]. However, it is unknown if the risk for venous thromboembolism should be a long term concern. In this presented case, prevention of thromboembolic events during the initial hospitalization was effective, but patient stopped the anticoagulant treatment after discharge which, probably, was provoked by thromboembolic complications 2.5 months after Covid-19 infection.

Conclusion

Our case highlights the need for being aware of late hypercoagulable complications after Covid-19 infection, particularly in high risk patients with comorbidities such as diabetes mellitus, hypertension, and presence of device leads in cardiac chambers. This case raises the following questions:

Is COVID-19 an independent long-term risk factor for venous thromboembolism and how long does this risk last?

Is there a high-risk subgroup and how would we identify this group?

What is the appropriate medication, dosage and duration of treatment?

Further research studies and clinical trials are needed to address these questions and the efficacy of extended post-discharge thromboprophylaxis.

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