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Case Report

A Suspected COVID-19 Associated Myocarditis Case Mimicking Life-threatening STEMI: A Shark Fin Appearance

Ratna Mariana Tamba[1,4*], Andika Sitepu[2,3,4]
[1]*Faculty of Medicine, Universitas Sumatera Utara, Medan, Indonesia.*
[2]*Department of Cardiology, Faculty of Medicine, Universitas Sumatera Utara, Medan, Indonesia.*
[3]*H. Adam Malik General Hospital, Medan, Indonesia.*
[4]*Grandmed Hospital, Lubuk Pakam, Indonesia.*

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**Corresponding author:*
ratnatamba82@email.com

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ABSTRACT

Some cases of myocarditis have been reported associated to Covid-19. The presence of proinflammatory response from immune cells is suggested to occur by binding to membrane protein ACE-2 leading to myocardial damage. Our hospital got a referral from a non-PCI-capable hospital with a 51-years-old woman chest pain and progressive heavy breath with history of fever two days ago. She was diagnosed with anterior STEMI and cardiogenic shock on vasopressors. Her electrocardiogram showed shark fin appearance suggesting life-threatening STEMI. Her rapid test for Covid-19 is non-reactive for both IgM and IgG. Her NCCT-thorax showed ground glass opacity in both lungs. Her laboratories finding showed elevated inflammatory markers and elevated cardiac biomarker. We took her naso-oro-pharingeal swab in the same day and process her with emergency PCI. Surprisingly, her angiography showed normal coronary artery without any significant stenosis. From her swab, there was no SARS-CoV-2 detected. In myocarditis, patient can mimic the same symptoms as STEMI such as chest pain and heavy breath with elevated cardiac biomarker, but the electrocardiogram usually shows widespread concave ST-elevation with PR-segment depression. Shark fin appearance is usually seen in life-threatening STEMI. In our patient, the NCCT-thorax showed GGO suggested Covid-19 involvement. But, the first swab was negative for SARS-CoV-2. Unfortunately, the patient was discharged without doctor's consent, so we cannot process the second swab and echocardiography to evaluate the function of myocardium. We still cannot confirm whether myocarditis-associated Covid-19 is true or how the involvement of the myocardium was could create a shark fin mimicking life-threatening STEMI.

Introduction

COVID-19 (corona virus disease 2019) was firstly declared by World Health Organization (WHO) in December 2019 in Wuhan, China [1]. It is caused by SARS-CoV2 virus with the flu-like symptoms [1-4]. Initially, it has been known that SARS-CoV2 involves the respiratory system, mostly characterized by cough, dyspnea, leading to acute respiratory distress syndrome, with mortality claimed below 1 %. Closely relative to Middle East respiratory syndrome coronavirus (MERS-CoV) and

SARS-CoV that possess cardiotropism, SARS-CoV2 is presumed to infect myocardiocytes [2,4]. Lately, it has been known that Covid-19 is involved with heart diseases, leading to myocarditis, heart failure, cardiogenic shock and arrhythmias [2,4]. Covid-19 associated myocarditis has now been reported in many countries with multiple presentations. Diagnosis of Covid-19 associated myocarditis is made both by finding signs and symptoms consistent with myocarditis and by finding the single stranded RNA of SARS-CoV2 from nasopharyngeal or oropharyngeal swab from the infected person [2-6]. Other than clinical presentations, electrocardiogram can help us to recognize Covid-19 associated myocarditis. In some cases reported before, it has been found that electrocardiogram in these cases ranging from normal, non-significant to concave ST-elevation and ST-depression, non-specific T-wave inversion, PR depression, low voltage ECG, atrial fibrillation, to life threatening conditions such as ventricular tachy-arrhythmias [3,4,5,7,8]. Meanwhile, it is never mentioned before that Covid-19 associated myocarditis case could present as life threatening STEMI, with a shark fin appearance/sign on electrocardiogram. Finding a shark fin sign on electrocardiogram suggest a proximal stenosis of the left anterior descending coronary artery and large anterior ischemia leading to a life threatening myocardial infarction [22]. Here we report a case of a woman who developed clinically suspected myocarditis associated with Covid-19 presenting a shark fin appearance in electrocardiogram mimicking a life threatening STEMI.

Case Presentation

A 51-years-old woman was referred from a non-PCI-capable hospital with chest pain and progressive heavy breath with history of fever two days ago. She was diagnosed with acute anterior STEMI and cardiogenic shock. After getting blood thinner and dual anti platelet therapy (DAPT), she was referred to undergo PCI. In our hospital, the patient is lethargic with blood pressure 80/60 mmHg on vasopressors. She spontaneously breathes, 32 times per minutes and tachycardia with heart rate was 127 beats per minute. Heart sounds are normal and rates were heard on both lungs, suggestive for acute heart failure accompanied with pneumonia. Her electrocardiogram showed shark fin appearance suggestive for life-threatening STEMI (Figure1).

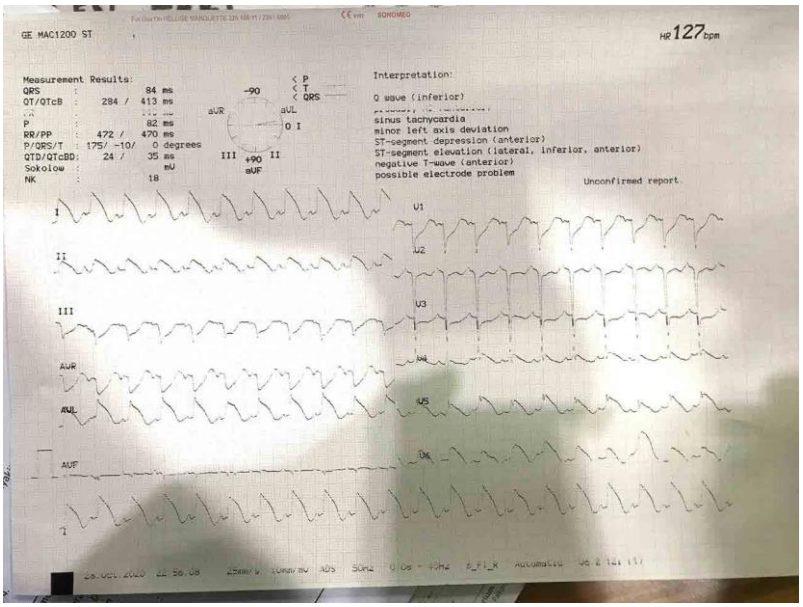

Figure 1. Shark-fin appearance in initial electrocardiogram

We did some screening tests for Covid-19, including rapid test and non-contrast computer tomography (NCCT) scan of thorax. Her rapid test for Covid-19 is non-reactive for both IgM and IgG. Her NCCT-thorax showed ground glass opacity in both lungs (figure 2).

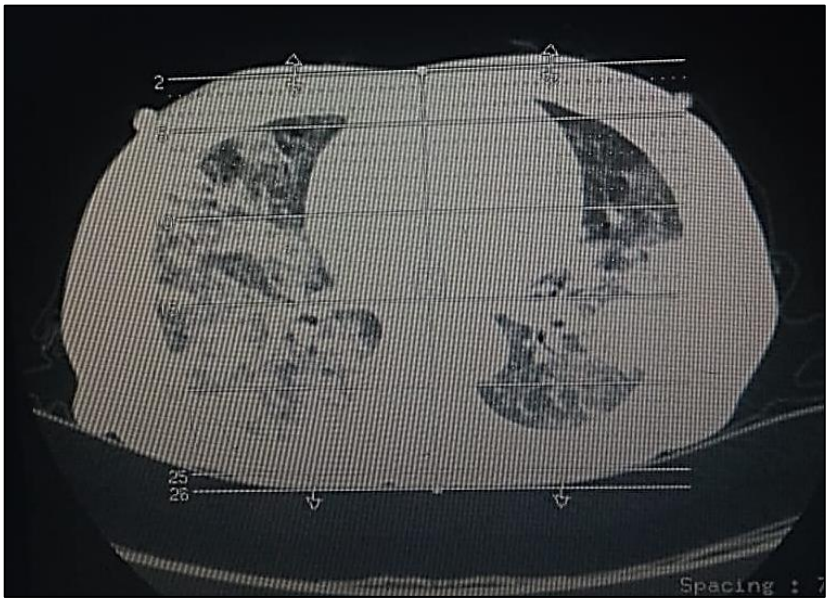

Figure 2. Ground glass opacity in both lungs

Her laboratories finding convenient with inflammatory process with highly elevated inflammatory markers; NLR 28.6 (N<3.13); PCT 0.53% (N 0.100-0.500%); monocyte 1,23x10^3 (N 0.3-0.8x10^3) and lymphocyte 0.6x10^3 (1.2-3.2 x10^3) and elevated cardiac biomarker; CKMB 9.6 ng/ml (n<4.3 ng/ml); Troponin-I 1.7 ng/ml (n<0.02 ng/ml); BNP 867 pg/ml (n<100 pg/ml), suggesting myocardial injury with acute heart failure. We suggested this case as an extensive anterolateral STEMI based on clinical presentations, physical examinations, electrocardiogram, and laboratory findings. We process her to catheterization laboratory for emergency percutaneous coronary

intervention (PCI). Surprisingly, her angiography showed normal coronary artery in right coronary artery (RCA), left anterior descending artery (LAD), and left circumflex artery (LCX) without any significant stenosis nor blood clot (figure 3 and figure 4).

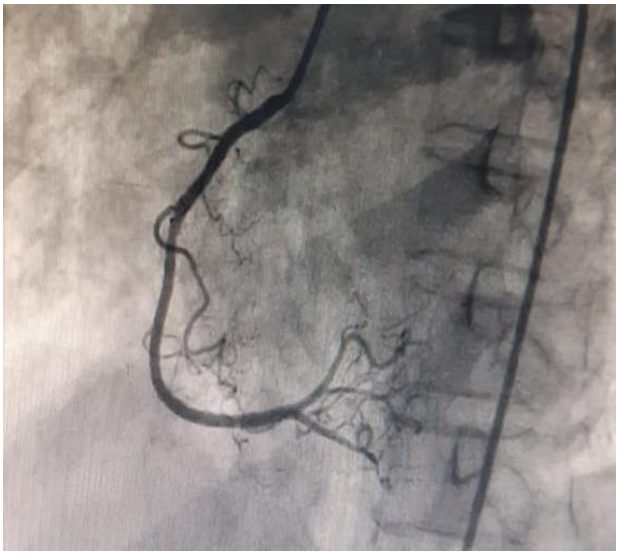

Figure 3.

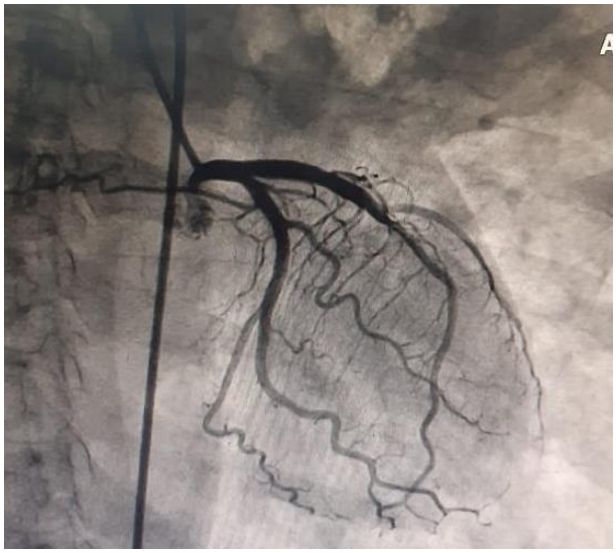

Figure 4. Both angiographic did not show any stenosis on RCA, LAD, and LCX.

Because the angiographic was normal, we suggested that the patient has myocarditis with covid-19 as the etiology. We took her naso-oro-pharyngeal swab in the same day and planned for the second swab in the next day. Unfortunately, the patient was discharged without doctor's consent, so we cannot process the second swab and echocardiography and other tests to evaluate the function of myocardium. The next day, we got the result from her swab, and it was negative for SARS-CoV-2.

Discussion

Covid-19 associated myocarditis cases have been reported extensively nowadays. Studies have found that covid-19 associated myocarditis has up to 7% mortality [4]. Presumed similar to MERS-CoV and SARS-CoV that have ever been isolated from heart of infected animals, SARS-CoV 2 as the etiology of covid-19 also is suggested can possess cardiotropism [4]. The pathophysiology of myocarditis associated with Covid-19 is suggested to be related to cytokine storm syndrome, including T-cells and interleukin 6 (IL-6) by binding its protein to the membrane protein angiotensin converting enzyme 2 (ACE-2). [3,4,7-10]

Clinical presentations of Covid-19 associated myocarditis vary among cases. Because most of myocarditis cases are due to viral infection, prodromal symptoms such as fatigue, fever, myalgia, and malaise, consistent with our patient's complains [3,4]. Injured myocardium from myocarditis can mimics acute myocardial infarction, such as heavy pain, radiating to neck, chin, or left arm. It is usually acute and severe onset and progressive, exacerbated by exertional or emotional stressor. Patient also had other signs and symptoms such as chest pain and shortness of breath with cardiogenic shock. Similar myocarditis cases were observed in studies of Inciardi et al, Zeng et al, Doyen et al, Asif et al, Trogen et al accompanied by acute respiratory distress syndrome. [7-9,11,15]

The laboratory findings convenient with inflammatory process with highly elevated NLR 28.6, similar to studies of Oleszak et al, Nicol et al, Doyen et al [6,9,10]. PCT was elevated on 0.53%, similar to Oleszak's study on 1.95% [6, 9, 10]. As myocardium injured in myocarditis, cardiac enzymes are usually elevated from baseline, including troponin, N-terminal pro-B-type natriuretic peptide (NT-pro BNP) and BNP [7-21]. Cardiac biomarker was also elevated in our case with CKMB 9.6 ng/ml (n<4.3 ng/ml) similar to Fereirra's study. Troponin-I was elevated in 1.7 ng/ml (n<0.02 ng/ml) and BNP 867 pg/ml (n<100 pg/ml) similar to most of studies reporting covid-19 associated myocarditis, suggesting myocardial injury with acute heart failure. [7-9, 11-21]

Electrocardiogram in myocarditis usually shows concave ST elevation in all leads and PR depression in lead avR [3,10,14,16]. Different with the cases of STEMI, electrocardiograms show us signs of acute myocardial infarction, varies from conventional STEMI (elevation of ST segment at the J point), to life-threatening myocardial infarction such as J point transitioning in a convex ST-segment (T wave indistinguishable from ST-segment due to extreme ST deviation) or usually called shark fin electrocardiogram. [22]

Electrocardiogram on our case showed a shark fin sign, an electrocardiogram pattern associated with proximal stenosis of the left anterior descending coronary artery and large anterior ischemia. Shark fin appearance is usually seen in life-threatening STEMI [22]. Other abnormalities seen in myocarditis are new bundle branch block, QT prolongation, premature ventricular complexes, atrioventricular block, brady-arrhythmias, and pseudo infarct patterns [7, 13, 14, 16]. From echocardiogram, we can see increased wall thickness, dilatation of some or all chambers, pericardial effusion, and systolic dysfunction [3, 4, 5]. But, we did not get a chance to do an echocardiogram to this patient. Initially, we suggested this case as an extensive anterolateral STEMI leading to heart failure (cardiogenic shock) based on clinical presentations, physical examinations, electrocardiogram and laboratory findings. But the normal coronary angiogram rules out our first suggestion or diagnosis. In our patient,

the NCCT-thorax showed GGO suggested COVID-19 involvement [2,4,5]. But, the first swab was negative for SARS-CoV-2, confusing us if it is a true myocarditis due to Covid-19 or not.

Conclusion

Based on clinical presentation, the patient can be diagnosed as myocarditis with SARS-CoV-2 as the etiology and life-threatening STEMI. From the electrocardiogram, we prefer acute STEMI than myocarditis. Based on angiographic, we can exclude STEMI because there is neither stenosis nor blood clot in coronary artery. The swab we took from the patient also did not help us find the etiology of the myocardial injury. We still cannot confirm if it is true myocarditis-associated Covid-19 or not and how was the involvement of the myocardium creating a shark fin mimicking life-threatening STEMI. We need further studies or other cases similar to this case.

Acknowledgement

There are no conflicts of interest.

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