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Case Report

Acute ST-Elevation Myocardial Infarction (STEMI) in Young Male with Nephrotic Syndrome: A Case Report

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ABSTRACT

Background: In young males, an acute myocardial infarction is an uncommon event. Thromboembolism caused by nephrotic syndrome (NS) is one of the pathophysiologies of their infarctions.
Objective: This study aimed to elaborate the case of acute St-Elevation Myocardial Infarction (Stemi) in young male with nephrotic syndrome
Case Presentation: A-24-y.o male patient, came with prolonged typical chest pain 72 hours before admission. The electrocardiogram (ECG) from the chest leads indicated ST-Elevation. Cardiac troponin was significantly elevated. For the last two weeks, he had been experiencing nephrotic syndrome symptoms including anasarca edema. It was supported by laboratory data which showed proteinuria, hyperlipidemia, and hypoalbuminemia. Coronary angiography showed complete acute occlusion of the left proximal anterior descending artery segment. Coronary angiography indicated complete acute occlusion of the left proximal anterior descending artery segment. The hypercoagulable condition in this patient was seen to be influenced by the increased fibrinogen levels, implying a correlation between coronary thrombosis and nephrotic syndrome.
Conclusion: Nephrotic syndrome should be considered as a contributing factor in any patient presenting with acute STEMI, particularly in young males.

1. Introduction

Approximately only 4% of acute myocardial infarction in patients before the age of 40 years old occur in young males.[1] In older patients, the main underlying disease is typically atherosclerotic plaque rupture. But in young patients, infarcts may have a variety of pathophysiology.[2] Nephrotic syndrome (NS) caused Thromboembolism is one of the pathophysiologies of their infarctions. Since NS is a relatively uncommon cause of acute coronary syndrome (ACS), the prevalence, pathogenesis, and treatment of these patients are still unclear.

This case aimed to describe the occurrence of acute myocardial infarction in a young adult, which was most likely caused by arterial thrombosis caused by the hypercoagulable state in nephrotic syndrome. In young people with chest pain, acute myocardial infarction should be considered, even though it is an uncommon occurrence. A thorough clinical history may help to determine the cause and guide treatment, but diagnostic coronary angiography is also required. Recurrent cardiac events should be minimized by careful risk factor management and treatment of the underlying cause.[3,4,5]

2. Case Presentation

A-24-years-old male patient, presented at our hospital ER with progressive typical chest pain since 72 hours before admission along with cold sweating, vomiting, and shortness of breath. He also complained of anasarca edema for the last two weeks. As his chest pain was not relieved by rest, he was brought to the nearest private hospital. There, the ECG showed anterior extensive STEMI (figure 1) and he got some medications, including loading Aspilet 320 mg, Clopidogrel 300 mg, ISDN 3x5 mg, Furosemide 40 mg intravenously, and Fondaparinux 1x2,5mg subcutaneously. On the second day of care at the first hospital, the pain worsened and they decided to refer him to our hospital for further management. There was a delay in referring him to a hospital capable of performing PCI due to financial issues. He had been an active smoker for 10 years, smoking 1 pack per day. He was also an alcoholic. There was no history of ischemic heart disease, Diabetes Mellitus, hypertension, or sudden cardiac death in his family.

At the time of admission, physical examination revealed that his blood pressure was 145/101 mmHg, heart rate 118 bpm, respiratory rate 22 per minute, oxygen saturation 95% on room air. He had

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palpebra, scrotal, and bilateral ankle oedema and ascites. Laboratory finding showed elevated cardiac troponin I (55.8 ug/L), CKMB (213 U/L), elevated fibrinogen (645,8 mg/dL); normal renal function with serum creatinine 1.26 mg/dL, ureum 38.9 mg/dL; severe Hypoalbuminemia (1.48 g/dL); low-density lipoprotein cholesterol, 431 mg/dL; and triglyceride, 299 mg/dL. Twenty-four hours urine analysis showed albuminuria (62.2 mg/dL). Urinalysis showed protein 3+; blood 3+. Complete blood count was normal; SGOT 221 U/L; SGPT 82 U/L; NT-Pro BNP 6122 pg/mL. ANA/anti-dsDNA were negative. The electrocardiogram showed an anterior extensive MI (Figure 2). The coronary

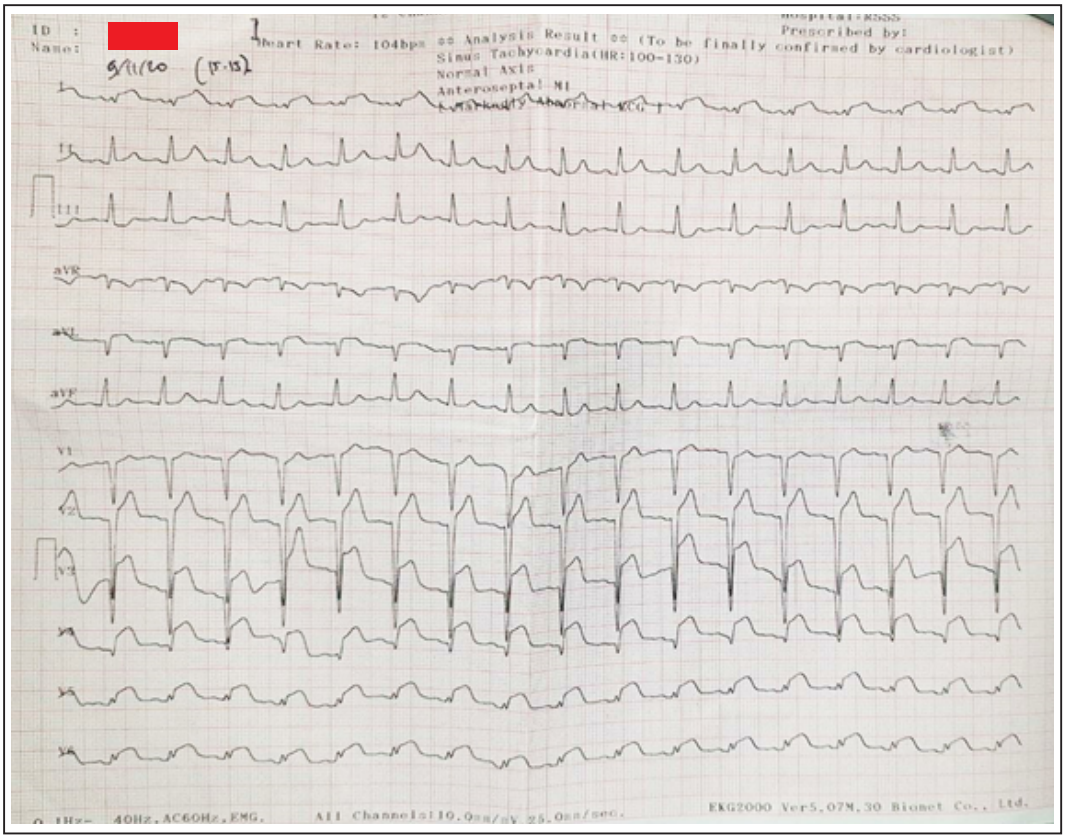

Figure 1. ECG at the local hospital showed ST-elevation in chest leads

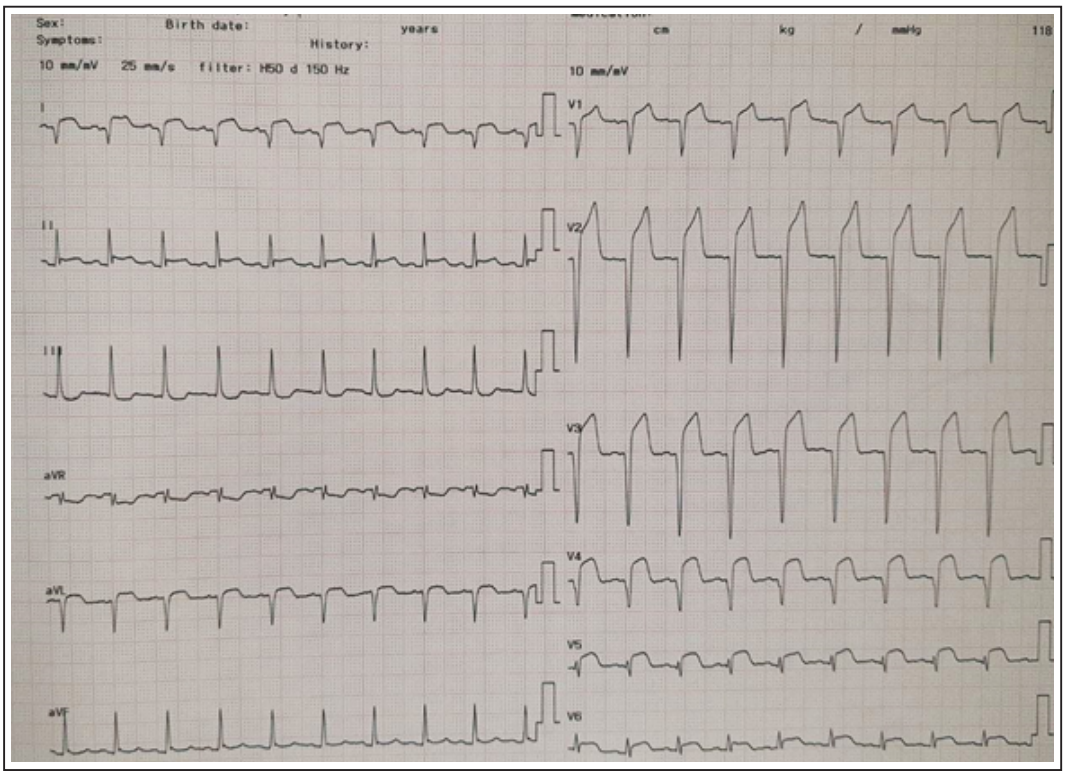

Figure 2. Electrocardiogram on admission, shows Sinus Rhythm with HR 125 bpm, frontal axis normal, horizontal axis clockwise rotation, normal p wave, PR duration 120ms, QRS duration 90ms, ST-elevation I, AVL, V1 - V6, ST depression III, QS I AVL, V1-V4

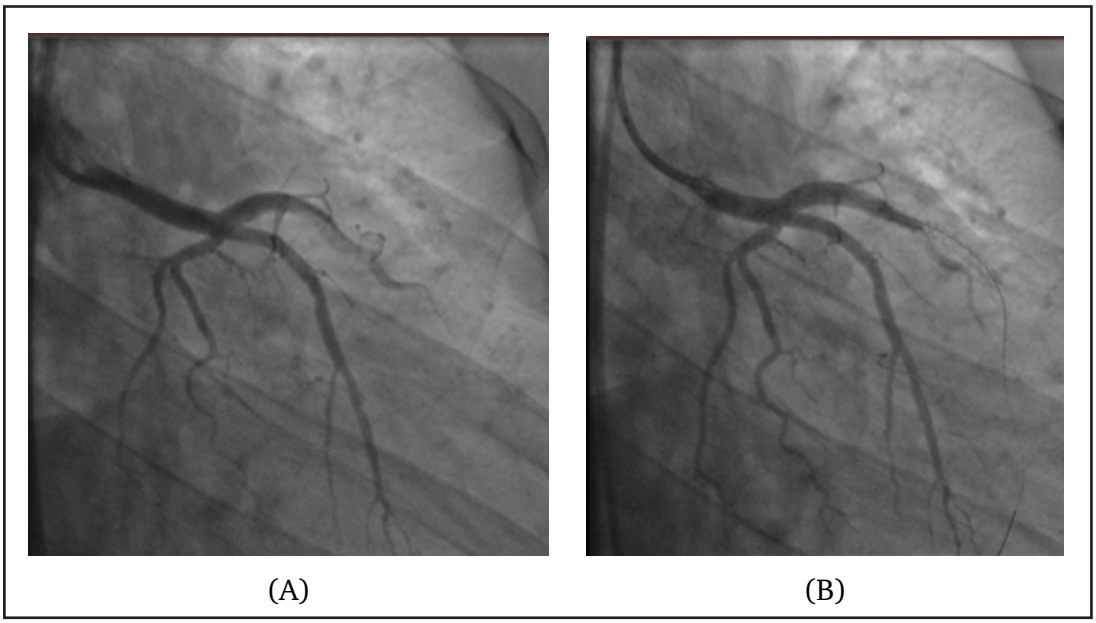

Figure 3. (A) Coronary angiography showed complete thrombotic occlusion of the proximal left anterior descending branch (LAD) (B) Coronary flow following aspiration of thrombus and insertion of a stent in LAD.

angiography revealed complete thrombotic occlusion of the mid-left anterior descending artery (LAD), while the left circumflex arteries and right coronary artery were normal (Figure. 3), The occluded artery was recanalized by a guidewire crossing of the thrombus, followed by thrombosuction. which resulted in a large amount of white thrombus and implantation of a drug-eluting stent at mid – distal LAD. Echocardiography showed that LV ejection fraction was reduced to 42%. There was akinesia in the anteroseptal, apicoseptal, and apicoanterior area, as well as hypokinesia in the mid anterior area.

Six days after admission, he was discharged on daily aspirin (80 mg), ticagrelor (90 mg bid), atorvastatin (20 mg), Ramipril (10 mg), Bisoprolol (2,5 mg), and methylprednisolone (3x16 mg). He followed up with the nephrologist for immunosuppressive therapy. He remained stable and asymptomatic at the three-month follow-up. The LDL-C and triglyceride were well below 120.74 mg/dL and 141.76 mg/dL, and the albuminuria was 2.5 g/24 hours.

3. Discussion

Acute myocardial infarction (AMI), which is uncommon in young adults, is categorized into two forms. Clear coronaries are a condition caused by coronary artery embolism, thrombosis, spasms, or a mix of these factors. Hypercoagulable states such as antiphospholipid syndrome, nephrotic syndrome, and others may cause coronary thrombosis. Including In this patient, the result of coronary angiography was one vessel disease with thrombotic occlusion with high burden thrombus in LAD, while the other coronary arteries were normal. Another condition is abnormal coronary, which involves significantly accelerated atherosclerosis, aneurysm, Spontaneous dissection, and coronary artery anomalous origin.[6] The pathophysiology between these two groups is significantly overlapping.

The involvement of anasarca edema, proteinuria, hypoalbuminemia, and hyperlipidemia in this patient indicated nephrotic syndrome. In addition, based on the symptom of typical chest pain, electrocardiography, and laboratory findings on admission, the patient had an acute myocardial infarction. We suggest that his acute myocardial infarction probably might be caused by arterial thrombosis, which might have been due to the hypercoagulable state caused by the nephrotic syndrome. However, he had tobacco smoking as an additional risk factor. Myocardial infarction pathophysiology in nephrotic patients is still uncertain. According to Fahal et al, thrombosis is most common in the first few months after a diagnosis, but it can occur at any time during the course of NS.[7] Several risk factors for thrombotic complications in nephrotic patients have been identified. Plasma hypercoagulability has long been known to be responsible for the nephrotic syndrome's tendency for thrombotic episodes. The alteration of blood level and function of antithrombotic and prothrombotic factors could cause hypercoagulability.[7,8,9] Urinary loss decreases the levels of factors IX, XI, XII, and antithrombin III. Low antithrombin III levels were seem insufficient for these procoagulant factors to be inactivated which resulted in thrombosis.[3,10,11,12] Factor XII, protein C, and protein S deficiencies were all related to an increase in thromboembolic events in nephrotic patients. Similarly, hypoalbuminemia triggered increased synthesis in the liver which resulted in higher levels of fibrinogen, factors II, V, VII, VIII, and X. Higher fibrinogen levels have been shown to change plasma viscosity.[3,10,11,12] Hypoalbuminemia in NS causes intravascular dehydration. Platelet dysfunction was a common and significant abnormality in nephrotic syndrome.[13,14] It is considered that the high incidence of thromboembolic consequences is due to an increase in platelet aggregation. Nephrotic hyperlipidemia, which was found in our patient, might be another factor in MI.[15] Diuretics which intended to decrease the edema caused by NS might induce hemoconcentration and increased the risk of thrombosis.[13] Similarly, steroids used to treat NS, as in this patient, could cause alteration of the blood level of coagulation factors, aggravating hypercoagulability thus causing thrombosis.[7,8] Additional risk factors in this patient include being an active smoker and an alcoholic.

Acute MI management in this context, is comparable with MI management in general. Primary percutaneous coronary transluminal angioplasties were utilized with or without coronary artery stenting in some cases.[2,4,5] Thrombolysis has been reported to be successful in comparable cases.[3,4,5] Prophylactic anticoagulation is only recommended in idiopathic membranous nephropathy, according to the 2012 Kidney Disease: Improving Global Outcomes (KDIGO) guidelines; however, available evidence indicates that anticoagulation should be considered in all patients with NS.[16] Prophylactic anticoagulation role in patients with NS who do not have thrombotic complications is still debated. Considering that platelets are the primary cause of arterial thrombi in NS, the use of antiplatelet agents such as aspirin seems rational, and some practitioners prescribe long-term low-dose aspirin in patients with chronic NS. Other general measures, such as hypertension

management, hyperlipidemia treatment, smoking cessation, and proteinuria reduction with angiotensin-converting enzyme inhibitors and/or angiotensin receptor blockers, are also beneficial.2,7,8

4. Conclusion

In the absence of atherosclerotic disease, young patients may suffer acute coronary events. In any patient diagnosed with ischemic heart disease and renal diseases, nephrotic syndrome should be taken as a risk factor.

5. Declarations

5.1. Ethics Approval and Consent to participate
Patient has provided informed consent prior to involve in the study.

5.2. Consent for publication
Not applicable.

5.3. Availability of data and materials
Data used in our study were presented in the main text.

5.4. Competing interests
Not applicable.

5.5. Funding source
Not applicable.

5.6. Authors contributions
Idea/concept: RP. Design: RP. Control/supervision: CTT, AFR, IP Data collection/processing: RP. Extraction/Analysis/interpretation: RP, CTT, AFR, IP Literature review: RP, CTT, AFR, IP. Writing the article: Ratna Pancasari. Critical review: CTT, AFR, IP. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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