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Treatment of Inferoposterior ST-Elevation Myocardial Infarction in Rural Area

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ABSTRACT

ST-elevation myocardial infarction (STEMI) is a life-threatening medical emergency characterized by complete occlusion of a coronary artery, leading to myocardial ischemia and subsequent tissue damage. A 51-year-old male with chest pain since two hours before admission, not relieved by rest. Electrocardiogram (ECG) showed ST elevation in leads II, III, avF, and ST elevation in lead V7-V9. Streptokinase 1,500,000 units was administered within 1 hour. ST waves in leads II, III and avF were decreased. The administration of fibrinolytic resulted in successful reperfusion of the occluded coronary artery, as evidenced by the resolution of ST-segment elevation on subsequent ECGs. Although primary PCI is considered the gold standard treatment for STEMI, fibrinolytic therapy remains a viable and potentially life-saving alternative, particularly in settings where immediate invasive interventions are not readily accessible. Fibrinolytic therapy should be started immediately for the best benefits.

Keywords: Fibrinolytic, Percutaneous Coronary Intervention, STEMI

INTRODUCTION

ST-Elevation Myocardial Infarction (STEMI) is a life-threatening medical emergency characterized by complete occlusion of a coronary artery, leading to myocardial ischemia and subsequent tissue damage. In Indonesia, Coronary Heart Disease (CHD) death came in second place among all causes of death in 2019 [1,2]. World Health Organization (WHO) recorded around 17.9 million patients died from cardiovascular diseases such as coronary heart disease, acute coronary syndrome (ACS), angina pectoris, rheumatoid arthritis and stroke. Most deaths occur in middle- and low-income countries. In addition to increased mortality, an ACS episode is linked to a high rate of hospital stays; 30% of patients who are discharged are readmitted within six months. An estimated $177 billion in major economic losses are expected due to this load. This case is a 51 year-old male with inferoposterior STEMI successfully treated with fibrinolytic, highlighting the diagnostic and subsequent management [3–5].

CASE PRESENTATION

A 51-year-old male patient arrived at the emergency department, with chest pain that persisted for two hours without relief from rest. The patient also had diaphoresis, no nausea, vomiting, nor tightness. The patient had a smoking habit since 15 years ago. The patient had no prior history of stroke or surgery. The patient never checked his blood pressure and blood sugar. None of the patient's relatives had similar complaints.

The patient was conscious on arrival with Glasgow Coma Scale (GCS) E4V5M6. Vital signs were: blood pressure 162/95 mmHg, heart rate 91/minute, respiratory rate 23/minute, temperature 36.3oC, and oxygen saturation 97%. Physical examinations found no abnormality, symmetrical chest, normal S1S2 heart sounds, no murmur, no palpable thrill, abdomen within normal limits, no palpable pulsation of the abdominal aorta, and warm extremities. The electrocardiograph (ECG) in the emergency room revealed ST elevation in leads II, III, and avF, and ST depression in leads V2-V6, consistent with an acute myocardial infarction in the inferior area **(Fig. 1).** The ECG posterior leads V7, V8, and V9 showed ST elevation consistent with acute myocardial infarction in the posterior area **(Fig. 2).** The complete blood test shows: Hb 16.5 g/dL, WBC 7,760/uL, platelet 263,000/uL, clinical chemistry and electrolyte were within normal limits, blood sugar 142 mg/dL, creatinine 0.71 mg/dL, CKMB 33.00 U/L, Troponin I 5920 ng/L. The chest X-ray did not show any abnormalities. No echocardiography was obtained. The patient was diagnosed with inferoposterior STEMI. Streptokinase 1,500,000 units in 1 hour was administered. The patient was also given double antiplatelet loading with aspirin 160 mg and clopidogrel 300 mg, bisoprolol 2.5 mg, atorvastatin 20 mg, enoxaparin 30 mg/0.3 mL iv, diazepam 5 mg, isosorbide dinitrate drip 2 mg per hour, ramipril 2.5 mg, and lansoprazole 30 mg iv. After fibrinolytic therapy, the patient's ECG shows a more than 50% reduction in ST elevation in leads II, III, and AVF **(Fig. 3).** The chest pain has decreased; no nausea, vomiting, nor sweating. On the second day, the patient no longer has chest pain, nausea, vomiting, and sweating. Vital signs were within normal limits. The patient did not have symptom on the third day of hospitalization. Treatment continued, and the patient was discharged on the fourth day of treatment and continue follow-up at the outpatient clinic.

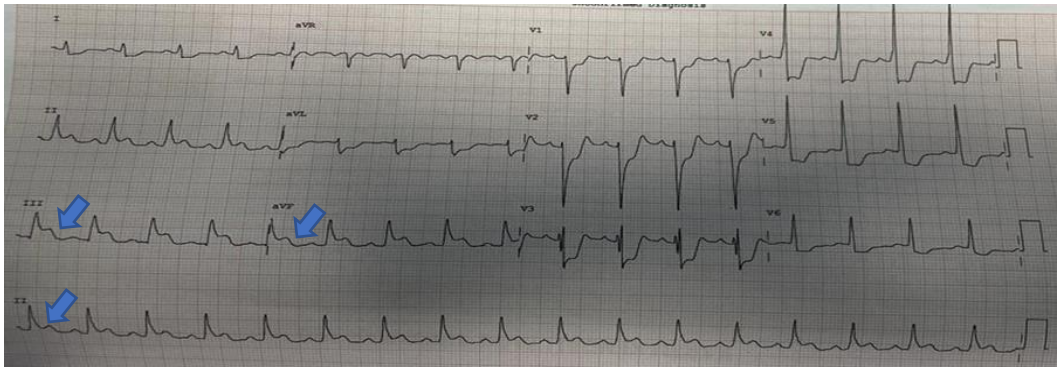

Figure 1. ECG in Emergency: STEMI Inferior (ST Elevation in lead II, III, avF)

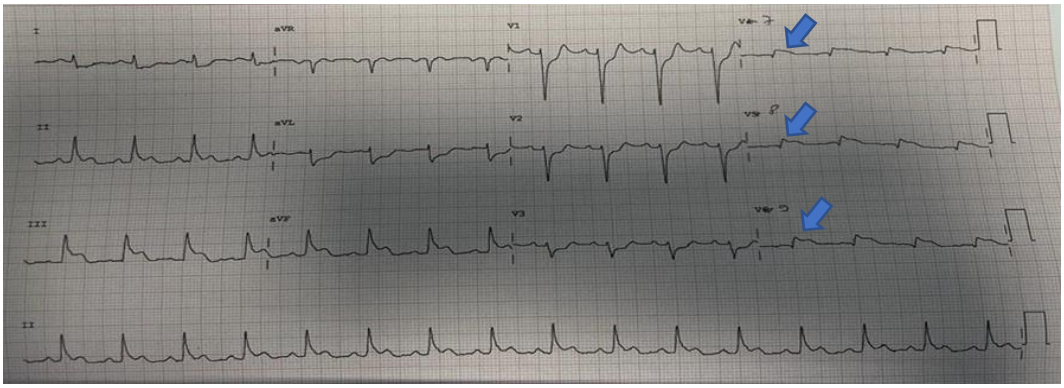

Figure 2. ECG on the posterior side: the ST Elevation in lead V7, V8, V9

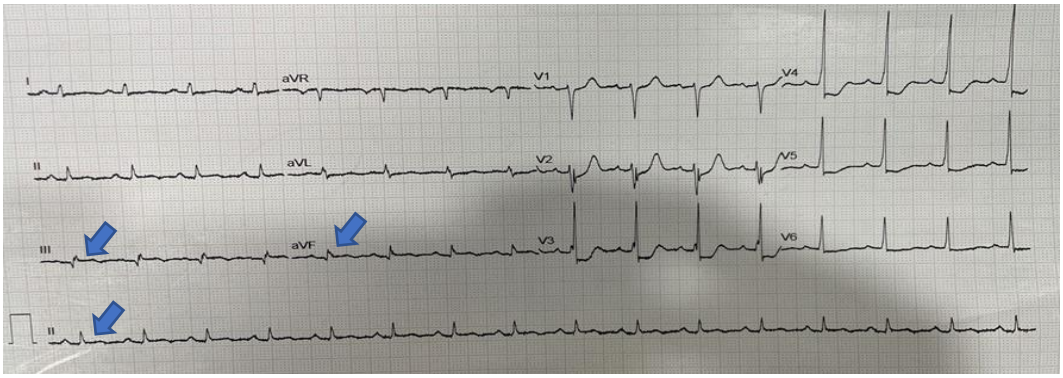

Figure 3. ECG after fibrinolysis: the ST Elevation decreased in lead II, III, avF

DISCUSSION

This case highlights a successful fibrinolytic therapy for inferoposterior ST-elevation myocardial infarction (STEMI). STEMI in the inferoposterior territory is relatively less common compared to anterior or lateral wall involvement. The timely administration of fibrinolytic agents remains a widely accepted treatment strategy for STEMI, particularly in resource-limited settings where immediate invasive interventions like percutaneous coronary intervention (PCI) may not be readily available [6,7]. The most crucial part of treatment is to achieve early, complete epicardial and microvascular reperfusion. Primary percutaneous coronary intervention (PCI) is currently the gold standard method of reperfusion because PCI reduces the risk of intracranial hemorrhage (ICH) and improves survival when compared to fibrinolytic therapy [8,9]. However, if immediate PCI administration is not possible, the mortality rate sharply increases, particularly when the delay exceeds 60 minutes from the first medical contact to PCI. The infarction of the inferior wall of the heart is typically caused by the occlusion of the right coronary artery or, less frequently, the left circumflex artery. Disruption of the posterior coronary circulation leads to a posterior myocardial infarction. About 10% population derives the posterior descending artery from the LCx artery, a condition known as the "left dominant" circulation. In the remaining 20%, the RCA and LCx both supply the posterior descending artery, a condition known as co-dominant circulation [10].

Diagnosis of STEMI based on clinical criteria is in accordance with myocardial ischemia, with at least two contiguous leads showing ST elevation of ≥ 2.5 mm in men under 40 years old, ≥ 2 mm in men over 40 years old, or ≥ 1.5 mm in women with leads V2-V3 and/or ≥ 1 mm in other leads (without left ventricular hypertrophy/LVH or left bundle branch block/LBBB). NSTEMI is clinically based on myocardial ischemia with a normal EKG or abnormalities including ST segment depression, transient ST segment elevation, and T wave changes aside from dynamic elevation of cardiac markers.5 In this case, the patient presented with characteristic symptoms of acute myocardial infarction, including severe chest pain radiating to the left arm and diaphoresis. The ECG demonstrated ST-segment elevation and reciprocal changes in the inferior leads and posterior leads, confirming the diagnosis of inferoposterior STEMI.

Almost three-quarters of acute myocardial infarctions are still treated with fibrinolytic-based reperfusion in low- and middle-income countries due to the unavailability of PCI facilities [11]. Only 25 to 50 percent of STEMI patients transferred for PCI achieve the time from first medical contact to balloon inflation in less than 120 minutes. When rapid PCI delivery is not possible, fibrinolytic therapy is an appropriate option [12]. Absolute contraindications include hemorrhagic stroke at any onset, ischemic stroke within the last 6 months, central nervous system damage and neoplasms, severe head trauma within the last 3 weeks,

gastrointestinal hemorrhage within the last month, a history of bleeding, and aortic dissection; relative contraindications are a history of transient ischemic attack (TIA), the use of oral anticoagulants, pregnancy or postpartum within one week, the presence of punctures, non-compressible blood vessels, traumatic resuscitation, refractory hypertension, active peptic ulcer, and infective endocarditis [13]. Due to the absence of contraindications, the decision was made to administer fibrinolytics promptly. Immediate reperfusion therapy is indicated for all STEMI patients with symptoms onset <12 hours with fibrinolytic therapy. In this case, the patient has experienced angina symptoms for the past 3 hours, making PCI (Percutaneous Coronary Intervention) access unfeasible, as it requires a door-to-balloon time of >120 minutes [14]. Therefore, the preferred reperfusion therapy for this patient is fibrinolytics. Fibrinolytic therapy aims to restore coronary blood flow by dissolving thrombi responsible for coronary artery occlusion. Start fibrinolytic therapy as soon as feasible for optimal benefits, ideally within the first three to six hours and potentially up to twelve hours after the onset of symptoms [15]. If the onset was more than 12 hours, fibrinolytics are less effective because the plaque is already mature and difficult to lyse, and there is an increased risk of bleeding [16].

The most often used fibrinolytic agent is still nonfibrin-specific streptokinase [17]. Streptokinase binds to plasminogen, converting plasminogen into plasmin. Meanwhile, alteplase, urokinase, reteplase, and tenecteplase work by breaking down plasminogen to produce plasmin, which then breaks down fibrin-rich clots into fibrin degradation products. Alfimeprase directly breaks down fibrin, resulting in fibrin degradation products. Appropriate use of fibrinolytic therapy can reduce hospital mortality rates by 25–50%. The greatest benefits are observed when fibrinolytics are administered within 3 hours of symptom onset and in patients at the highest risk, such as the elderly. In observational studies, streptokinase may be more likely to activate platelets when used in conjunction with fibrin-specific agents as part of a pharmacoinvasive strategy. The goal of reperfusion therapy is the disappearance of chest pain, a reduction of ST elevation by more than 50% within 60-90 minutes, and the emergence of typical reperfusion arrhythmias [18].

Streptokinase was used in this case due to its availability and resulted in successful reperfusion, as evidenced by the resolution of ST-segment elevation on subsequent ECGs. The patient's symptoms also improved significantly, with relief of chest pain and resolution of diaphoresis. Close monitoring of the patient's vital signs, ECG changes, and laboratory parameters, such as cardiac enzymes, is crucial following fibrinolytic therapy to assess for adverse events or reocclusion. It is important to acknowledge that fibrinolytic therapy may carry potential risks, including bleeding complications, intracranial hemorrhage, or reocclusion of the coronary artery despite initial successful reperfusion. Careful patient selection based on established guidelines and risk-benefit assessment is essential [19].

CONCLUSION

Although primary PCI is considered the gold standard treatment for STEMI, fibrinolytic therapy remains a viable and potentially life-saving alternative, particularly in settings where immediate invasive interventions are not readily accessible. Further research and larger clinical trials are warranted to optimize the use of fibrinolytics in specific subsets of STEMI patients and refine treatment protocols.

Declaration by Authors

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