Case Report: Initial Management of ST-Elevation Myocardial Infarction Patients with Hypotension in the Emergency Unit of Merauke Regional General Hospital

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ST-elevation myocardial infarction (STEMI) is a cardiovascular emergency caused by acute coronary artery occlusion that requires immediate reperfusion therapy to limit infarct size and reduce mortality. In healthcare facilities with limited access to primary percutaneous coronary intervention (PCI), fibrinolytic therapy remains an important reperfusion strategy. A 71-year-old man presented to the emergency department with typical chest pain that had begun one hour before admission. The pain radiated to the left arm, left neck, and back and was accompanied by nausea, vomiting, and cold sweats. Electrocardiography (ECG) revealed ST-segment elevation in leads V1–V5, consistent with anteroseptal STEMI with an onset of less than 12 hours. Initial management included oxygen supplementation, dual antiplatelet therapy, anticoagulation, high-intensity statin therapy, intravenous nitrates, and fibrinolytic therapy with 1.5 million units of streptokinase. Ten minutes after fibrinolysis was initiated, the patient developed severe hypotension, with a blood pressure of 60/43 mmHg. Intravenous nitrate therapy was immediately discontinued, and dobutamine infusion was initiated. Following this intervention, the patient's blood pressure increased to 116/69 mmHg, accompanied by improved hemodynamic stability. Early diagnosis and prompt reperfusion therapy are essential in the management of anteroseptal STEMI. Fibrinolytic therapy remains an effective reperfusion option in healthcare facilities with limited access to primary PCI. Close monitoring for hemodynamic complications, particularly hypotension during treatment, is crucial to prevent progression to cardiogenic shock and improve patient outcomes.

Keywords: Anteroseptal STEMI, Fibrinolysis, Streptokinase, Dobutamine, Hypotension

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1. Introduction

Cardiovascular disease (CVD) remains one of the leading causes of morbidity and mortality worldwide and continues to represent a major global health burden. According to the World Health Organization (WHO), an estimated 19.8 million people died from cardiovascular diseases in 2022, accounting for approximately 32% of all deaths worldwide. Acute coronary syndrome (ACS) is one of the most important clinical manifestations of cardiovascular disease and includes a spectrum of conditions ranging from ST-segment elevation myocardial infarction (STEMI) to non-ST-segment elevation myocardial infarction (NSTEMI) and unstable angina. In 2019, approximately 5.8 million new cases of ischemic heart disease were estimated to occur across the 57 member countries of the European Society of Cardiology, highlighting the substantial burden of coronary artery disease globally.

The burden of ACS is also reflected in its high mortality and the need for timely reperfusion therapy. Data from the multicenter One ACS Registry reported that 48.8% of ACS cases presented with ST-segment elevation (ACS-EST), while 51.2% presented with non-ST-segment elevation (ACS-NEST). Among

patients with ACS-EST, approximately 65.2% underwent reperfusion therapy. Overall mortality among patients with ACS was 8.9%, with a significantly higher mortality rate observed in patients with ACS-EST compared with those with ACS-NEST (11.7% vs. 6.2%). These findings demonstrate that STEMI remains a particularly serious form of ACS and requires rapid recognition and immediate treatment to reduce the risk of myocardial damage and death (PERKI, 2024).

In Indonesia, cardiovascular disease similarly represents a major public health problem. According to data from the Indonesian Ministry of Health in 2023, approximately 651,481 deaths per year were attributed to cardiovascular disease. These deaths included 331,349 deaths from stroke and 245,343 deaths from coronary heart disease. The high mortality associated with coronary heart disease emphasizes the importance of early diagnosis, appropriate emergency management, and timely reperfusion in patients presenting with acute myocardial infarction. In addition, differences in healthcare infrastructure and access to advanced cardiac intervention between regions may influence the selection and timing of reperfusion strategies in clinical practice (Ministry of Health of the Republic of Indonesia, 2023).

Acute ST-segment elevation myocardial infarction occurs primarily as a consequence of acute and usually complete occlusion of an epicardial coronary artery, resulting in prolonged myocardial ischemia and progressive myocardial necrosis. Because myocardial injury progresses with time, early restoration of coronary blood flow is essential to limit infarct size, preserve ventricular function, and improve clinical outcomes. Anterior STEMI, particularly anteroseptal STEMI, is commonly associated with occlusion of the proximal left anterior descending (LAD) coronary artery. Because the LAD supplies a substantial portion of the left ventricular myocardium and interventricular septum, proximal occlusion may result in a larger area of myocardial necrosis, severe left ventricular systolic dysfunction, and a greater risk of life-threatening complications compared with infarctions involving other coronary territories (Rao et al., 2025).

Hypotension is an important and potentially life-threatening complication in patients with STEMI. A reduction in blood pressure may reflect extensive myocardial damage, impaired left ventricular contractility, reduced cardiac output, significant arrhythmias, mechanical complications, or the early development of cardiogenic shock. In patients with anterior STEMI, extensive involvement of the anterior wall and interventricular septum can substantially impair the heart's pumping capacity, thereby increasing the likelihood of hemodynamic instability. The presence of hypotension in STEMI is clinically significant because it has been associated with increased in-hospital and long-term mortality. Furthermore, hypotension may complicate the administration of several standard pharmacological therapies, including nitrates, beta-blockers, and certain analgesic agents, which may further limit therapeutic options in patients with unstable hemodynamics (Sinha et al., 2025).

Primary percutaneous coronary intervention (PCI) is currently the preferred reperfusion strategy for STEMI when it can be performed within the recommended time frame. However, timely access to PCI remains challenging in many healthcare facilities, particularly in hospitals located in remote or resource-limited areas where catheterization laboratories are unavailable or patient transfer may result in substantial treatment delays. In such circumstances, fibrinolytic therapy remains an important alternative reperfusion strategy. The 2025 ACC/AHA/ACEP/NAEMSP/SCAI guidelines recommend fibrinolytic therapy for eligible patients with STEMI presenting within 12 hours of symptom onset when the anticipated first medical contact (FMC)-to-device time exceeds 120 minutes and there are no absolute contraindications to fibrinolysis. When administered promptly, fibrinolytic therapy can restore patency of the infarct-related artery, reduce the extent of myocardial necrosis, and decrease mortality.

Although fibrinolytic therapy can provide an essential reperfusion option in settings where primary PCI cannot be delivered promptly, its administration requires careful monitoring because patients may develop

clinically significant hemodynamic complications during treatment. Hypotension occurring shortly after fibrinolytic therapy may complicate the clinical management of STEMI and requires prompt assessment to determine its underlying cause and prevent progression to cardiogenic shock. Appropriate management requires continuous monitoring of blood pressure, cardiac rhythm, oxygenation, and clinical signs of tissue hypoperfusion, together with timely adjustment of medications that may worsen hypotension and the use of hemodynamic support when indicated. Therefore, a case of anteroseptal STEMI complicated by severe hypotension during fibrinolytic therapy is clinically important to illustrate the challenges of STEMI management in a healthcare setting with limited access to primary PCI, as well as the importance of rapid recognition and appropriate management of hemodynamic instability to improve patient outcomes.

2. Literature Review And Problem Statement

ST-Elevation Myocardial Infarction (STEMI)

ST-elevation myocardial infarction (STEMI) is a form of acute coronary syndrome (ACS) that is commonly caused by rupture or erosion of an atherosclerotic plaque, followed by thrombus formation and acute occlusion of an epicardial coronary artery. This occlusion results in interruption of myocardial blood flow, leading to ischemia that may progress to necrosis if not treated promptly. Clinically, STEMI is characterized by symptoms of myocardial ischemia, particularly typical chest pain, accompanied by ST-segment elevation on electrocardiography (ECG) corresponding to the location of the infarction. This condition represents a cardiovascular emergency because myocardial injury progresses with increasing duration of coronary occlusion. Therefore, the primary principles of STEMI management are rapid diagnosis, appropriate antithrombotic therapy, and timely reperfusion to restore coronary blood flow, limit infarct size, preserve left ventricular function, and reduce the risk of mortality and complications. The 2025 ACC/AHA/ACEP/NAEMSP/SCAI guidelines emphasize reperfusion strategies as a fundamental component of STEMI management, including pharmacological therapy, primary percutaneous coronary intervention (PCI), fibrinolysis, and management of complications such as heart failure and cardiogenic shock.

Anteroseptal STEMI

Anteroseptal STEMI is a manifestation of STEMI involving the anterior wall and interventricular septum, which is generally associated with occlusion of the left anterior descending (LAD) coronary artery. The area supplied by the LAD includes a substantial portion of the anterior myocardium and interventricular septum; therefore, proximal occlusion of this vessel can result in a large infarct area and significant impairment of left ventricular function. On electrocardiography, involvement of the anteroseptal region is typically indicated by ST-segment elevation in the precordial leads, particularly V1–V4, which may extend to V5 or V6 when the affected anterior area is more extensive. The extensive myocardial ischemia associated with anterior STEMI places patients at a higher risk of reduced left ventricular systolic function, pulmonary edema, ventricular arrhythmias, conduction disturbances, and hemodynamic instability. Therefore, anteroseptal STEMI requires prompt identification and rapid reperfusion, as delays in restoring coronary blood flow may lead to further myocardial necrosis and increase the risk of serious cardiovascular complications. When primary PCI can be performed promptly, it is the preferred reperfusion strategy; however, in healthcare facilities without access to PCI or when transfer is expected to result in significant delays, fibrinolytic therapy may serve as an important alternative for eligible patients.

Hipotensi pada Pasien STEMI

Hypotension in patients with STEMI is an important clinical condition that may indicate hemodynamic impairment resulting from reduced cardiac output and left ventricular pump function. A decrease in blood pressure may occur due to extensive myocardial damage, left ventricular dysfunction, arrhythmias,

mechanical complications of myocardial infarction, hypovolemia, medication effects, or progression to cardiogenic shock. In anterior STEMI involving a large area of the myocardium, impaired left ventricular contractility may lead to reduced stroke volume and cardiac output, resulting in decreased systemic arterial pressure. Hypotension also presents a challenge in pharmacological management because several medications commonly used in STEMI, such as nitrates and beta-blockers, may further lower blood pressure in patients with existing hemodynamic instability. Therefore, the development of hypotension during the acute phase of STEMI requires immediate evaluation, including assessment of blood pressure, heart rate and rhythm, signs of peripheral hypoperfusion, volume status, ventricular function, and the possibility of mechanical complications. Hypotension accompanied by signs of hypoperfusion should be considered a high-risk condition because it may progress to cardiogenic shock, a complication of STEMI associated with high mortality. Current guidelines recommend prompt management of patients with ACS and hemodynamic instability through strategies aimed at restoring adequate tissue perfusion and performing timely revascularization of the culprit vessel.

Tata Laksana STEMI dengan Hipotensi

The management of STEMI complicated by hypotension requires a rapid, targeted approach tailored to the patient's hemodynamic condition. Initial management includes close monitoring of vital signs and cardiac rhythm, identification of the underlying cause of hypotension, administration of antiplatelet and anticoagulant therapy when indicated, and selection of the most appropriate and feasible reperfusion strategy. Primary PCI is the preferred reperfusion strategy when it can be performed within the recommended time frame, whereas fibrinolytic therapy is an alternative for patients presenting within 12 hours of symptom onset when primary PCI cannot be performed in a timely manner and there are no absolute contraindications to fibrinolysis. The 2025 guidelines recommend that, in non-PCI-capable facilities, patients with STEMI should be transferred for primary PCI when the first medical contact-to-device time can be achieved within 120 minutes. If this target cannot be achieved and the patient presents within 12 hours of symptom onset, fibrinolytic therapy is recommended to facilitate timely reperfusion.

In patients who develop hypotension during therapy, medications that may further reduce blood pressure, such as nitrates, should be carefully evaluated and discontinued when clinically inappropriate. Hemodynamic support should be provided according to the severity of impaired perfusion and the underlying cause of hypotension. If hypotension progresses to cardiogenic shock, the patient requires intensive management and urgent revascularization. Current guidelines recommend revascularization of the culprit vessel in patients with ACS complicated by cardiogenic shock or hemodynamic instability to improve the likelihood of survival.

3. Method

This study used a descriptive case report design to describe the initial management and clinical course of a patient with ST-elevation myocardial infarction (STEMI) complicated by severe hypotension during fibrinolytic therapy. The case was conducted in the Emergency Department of Merauke Regional General Hospital. The patient was selected based on the presence of clinical and electrocardiographic findings consistent with anteroseptal STEMI and the development of significant hemodynamic instability during initial reperfusion management. The case report focused on the patient's initial presentation, diagnostic findings, emergency management, response to therapy, and changes in hemodynamic status during treatment.

Data were obtained from the patient's medical records and direct clinical observations during emergency management. The collected information included demographic characteristics, presenting symptoms,

duration and characteristics of chest pain, physical examination findings, vital signs, electrocardiographic findings, initial pharmacological therapy, fibrinolytic therapy, and subsequent hemodynamic changes. Particular attention was given to the occurrence of hypotension following administration of streptokinase, including the recorded blood pressure, therapeutic interventions, discontinuation of medications that could worsen hypotension, administration of hemodynamic support, and the patient's clinical response. The patient's clinical course was described chronologically to provide a clear overview of the decision-making process and management of STEMI complicated by acute hypotension.

Patient confidentiality was maintained throughout the preparation of this case report. Personal identifiers and other information that could reveal the patient's identity were excluded from the report. The case was presented for educational and scientific purposes, with emphasis on the clinical management of STEMI and the recognition and treatment of acute hemodynamic instability during fibrinolytic therapy. The preparation of the case report followed applicable institutional requirements and ethical principles concerning patient privacy, confidentiality, and the responsible use of clinical information.

4. Results And Discussion

Result

A man, initials SL, aged 71 years came to the emergency room with complaintsComplaints of chest pain began 1 hour after visiting the hospital. The chest pain was felt suddenly, as if a heavy object had fallen on it. The pain radiated to the left arm and neck, as well as to the back. The patient had previously complained of fever and an occasional cough with mucus. He had nausea, vomiting, heartburn, and cold sweats. He was weak, restless, and had difficulty communicating. The patient had experienced similar complaints about 20 years ago. He denied other complaints.

Past Medical History : Hypertension (+), Cholesterolemia (+), Heart Disease (+) in 1999. Vital signs examination was performed and found Blood pressure: 139/97, Pulse: 98x/minute, SPO2: 94% all within normal limits. Followed by a complete physical examination from head to toe, the results of the examination were within normal limits. Next, the patient underwent an ECG examination and found a typical picture of ST Elevation in leads V1, V2, V3, V4, and V5 accompanied by Acute Anterior Myocardial Infraction.

Figure 1. Initial ECG results

Based on anamnesis, physical examination and supporting examinations, the patient was diagnosed with Anteroseptalonset STEMI <12 hours. Next, initial management was carried out with O2 3 LPM, IVFD Nacl 0.9% 20 tpm, fibrinolytic with Steptokinase 1.5 million units in D5% 50 cc finished in 90 minutes /sp with premedication of dexamethasone 1 ampoule / IV and diphenhydramine 1 amp / IV, farsorbide 2 mg / hour /

SP (1 amp + 40 cc NS - 10 cc / hour / SP) -> up titration, omeprazole 40 mg / 12 hours / IV, Ondanceteron 4 mg / 12 hours / IV, loading aspirin 160 mg continued 1x80 mg, loading briclot 180 mg, continued 1x90 mg, enoxrin 0.6 cc / 24 hours / SC, bisporlolol 1x2.5 mg, rosuvastatin 1x40 mg, alprazolam 0.5 mg 0-0-1/2 tab, lactulose syrup 3x2 e.g. 10 minutes after starting fibrinolytics, the patient's blood pressure dropped to 60/43 mmHg, so farsorbide administration was stopped. Then, an initial dose of dobutamine was given at 5 mcg/kg/minute/SP with up titration every 30 minutes, then the dose was increased by 2.5 mcg/kg/minute and ondanceterone 8 mg/iv. After dobutamine administration, the patient's blood pressure began to normalize to 116/69 mmHg. After completing fibrinolytics, an ECG examination was performed and the following results were obtained.

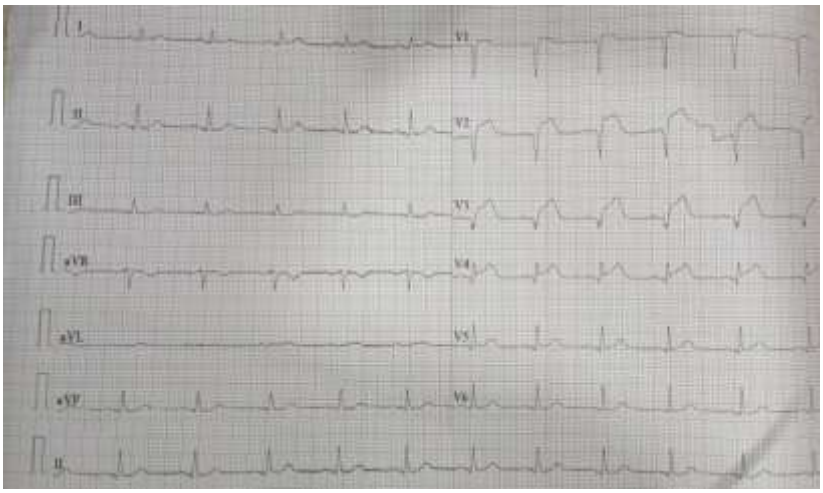

Figure 2. ECG After Fibrinolytics.

Discussion

STEMI is the most severe form of acute coronary syndrome, resulting from total occlusion of a coronary artery, leading to progressive myocardial ischemia and necrosis. The success of STEMI treatment is determined by the speed of diagnosis and reperfusion. Delayed reperfusion can increase infarction size, reduce left ventricular function, and increase mortality. In the case above, the patient presented with chest pain that had lasted for 1 hour, characteristically radiating to the left arm, left neck, and back. ECG findings revealed ST elevation in V1-V5, further supporting the diagnosis of anteroseptal STEMI.

Pathophysiologically, anteroseptal STEMI is generally caused by occlusion of the left anterior descending artery (LAD), particularly in the proximal segment. The LAD supplies most of the anterior wall of the left ventricle, the interventricular septum, and the apex of the heart. Infarctions in this area are often associated with a larger area of necrosis than other infarction sites. Research conducted by Johansson et al. showed that in LAD infarction, the decreased ability of the heart muscle to contract occurs not only in the infarcted area, but also in the entire left ventricular segment including the remote myocardium, whereas in RCA and LCx infarctions the function of the remote myocardium is relatively preserved.These findings explain why anterior STEMI is often associated with more severe left ventricular dysfunction and a higher incidence of heart failure (Johansson, 2021). Furthermore, a study by Poodlesnikar et al. reported that remote myocardial dysfunction is associated with adverse left ventricular remodeling after anterior STEMI and may contribute to long-term cardiac function decline (Poodlesnikar, 2020).

A crucial aspect in this case is the choice of fibrinolytic therapy as the reperfusion strategy. Primary percutaneous coronary intervention (PCI) is the preferred reperfusion therapy for STEMI due to its higher reperfusion success rate and lower risk of intracranial hemorrhage compared to fibrinolysis. However, in

hospitals with limited cardiac catheterization facilities, including remote areas in Indonesia, the target FMC-to-device time of ≤120 minutes is often difficult to achieve. Under these conditions, the ACC/AHA 2025 and ESC 2023 guidelines recommend fibrinolytic administration to STEMI patients with symptom onset less than 12 hours and without absolute contraindications. The effectiveness of fibrinolysis is strongly influenced by the timing of administration, with the greatest benefit achieved when therapy is administered within the first 2–3 hours after symptom onset. In this case, streptokinase was administered approximately one hour after the onset of chest pain, thus falling within the optimal reperfusion window. This decision was highly appropriate given the limited access to primary PCI. Various studies have shown that early administration of fibrinolytics can improve coronary artery patency, reduce infarct size, maintain left ventricular function, and reduce short-term and long-term mortality (Bikdeli, 2025).

An interesting clinical development in this case is the development of severe hypotension approximately 10 minutes after initiation of fibrinolytic therapy, with blood pressure dropping to 60/43 mmHg. Hypotension in STEMI patients should always be considered a serious clinical sign until proven otherwise, as it may reflect extensive left ventricular dysfunction, arrhythmias, mechanical complications, or the early development of cardiogenic shock. In anterior STEMI, decreased left ventricular contractility due to the large infarct area is the most common cause of hypotension and cardiogenic shock (Abdelnabi M, 2024).

In addition to the extensive infarction area, hypotension in this patient was likely exacerbated by intravenous nitrate (farsorbide) administration. Nitrates act through venous and arterial vasodilation, thereby reducing preload and afterload. In patients with significant left ventricular dysfunction, this vasodilatory effect can lead to a more severe decrease in blood pressure. Therefore, discontinuing farsorbide immediately after the onset of hypotension is appropriate and in accordance with the principles of STEMI management. Recent guidelines state that nitrates should be used cautiously and avoided in patients with hypotension or hemodynamic instability (Rao SV, 2025).

A rapid response to hypotension is achieved by administering dobutamine. Dobutamine is a β1-adrenergic agonist that increases myocardial contractility and cardiac output, making it frequently used as first-line therapy in STEMI patients with low cardiac output syndrome or cardiogenic shock, where blood pressure remains relatively stable. In this case, dobutamine administration successfully increased blood pressure from 60/43 mmHg to 116/69 mmHg, indicating improved systemic perfusion and a favorable hemodynamic response. This condition supports the hypothesis that the hypotension was primarily caused by acute cardiac pump dysfunction and the vasodilatory effects of nitrates (Abdelnabi M, 2024).

The success of reperfusion after fibrinolysis is generally assessed based on improvement in clinical symptoms, resolution of ST-segment elevation ≥50% within 60–90 minutes, and the emergence of reperfusion arrhythmias. In this case report, a repeat ECG was performed after fibrinolytic therapy was completed. Further evaluation of the degree of ST-segment resolution is crucial because it can be an indicator of reperfusion success and determine the need for further pharmacoinvasive strategies. According to ESC 2023 and ACC/AHA 2025, patients receiving fibrinolysis are still recommended to undergo coronary angiography and PCI within 2–24 hours after successful fibrinolysis, or immediate rescue PCI if there are signs of reperfusion failure (Byrne RA, 2024).

Based on the above case, this case illustrates the importance of early recognition of anteroseptal STEMI in the emergency department, prompt implementation of reperfusion therapy, and awareness of hemodynamic complications that can occur during initial management. The decision to perform early fibrinolysis in a facility without access to primary PCI is in accordance with current guideline recommendations and provides the best opportunity for maintaining myocardial viability. Prompt treatment

of hypotension with nitrate discontinuation and dobutamine administration also plays a crucial role in stabilizing the patient's condition.

5. Conclusion

Anteroseptal ST-elevation myocardial infarction (STEMI) is a cardiovascular emergency that requires rapid diagnosis and timely reperfusion therapy to minimize myocardial damage and reduce the risk of mortality and complications. In healthcare facilities with limited access to primary percutaneous coronary intervention (PCI), fibrinolytic therapy remains an important reperfusion strategy for eligible patients presenting within 12 hours of symptom onset. However, patients with anterior STEMI are at increased risk of extensive myocardial damage and hemodynamic complications, including severe hypotension and cardiogenic shock. Therefore, continuous monitoring of hemodynamic status during reperfusion therapy is essential for early detection of clinical deterioration.

In this case, severe hypotension developed shortly after the initiation of fibrinolytic therapy, requiring immediate discontinuation of intravenous nitrate therapy and administration of dobutamine to support hemodynamic stability. The patient's blood pressure subsequently improved, demonstrating the importance of prompt recognition and appropriate management of hypotension during STEMI treatment. This case highlights that optimal STEMI management requires not only rapid reperfusion but also careful assessment of treatment-related complications and individualized hemodynamic support. Early intervention may prevent progression to cardiogenic shock and contribute to better clinical outcomes, particularly in settings where access to primary PCI is limited..

6. References

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