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Case Report

Twists and Turns in The Fibrinolytic Therapy of Infero-Posterior ST Elevation Myocardial Infarction and Right Ventricular Infarction Patient with Cardiogenic Shock: A Case Report

Jonathan Edbert Afandy [1], Taslim [2]

[1] *Nunukan Regency General Hospital, North Kalimantan, Indonesia*
[2] *Nunukan Regency General Hospital, North Kalimantan, Indonesia*

Corresponding Author:
Name: Jonathan Edbert Afandy
Email: jonathanedbert@yahoo.co.id

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ABSTRACT

Introduction: *Fibrinolytic therapy is preferred for ST-segment elevation myocardial infarction (STEMI) when the timeframe for percutaneous coronary intervention (PCI) cannot be achieved. Although effective, fibrinolytics are also associated with several adverse effects in addition to the complications of STEMI itself.* ***Case:*** *A 45-year-old active smoker man presented with chest pain, dyspnea, and diaphoresis for the past hour. Electrocardiography revealed infero-posterior STEMI with right ventricular infarction. Echocardiography demonstrated akinetic inferior, infero-septal, and posterior walls with an estimated right atrial pressure of 15 mmHg.* ***Management:*** *Therapy consisted of aspirin, clopidogrel, furosemide, and streptokinase infusion. Within five minutes of initiating streptokinase, the patient developed sudden hypotension that required norepinephrine, dobutamine, and dopamine. At approximately halfway of the streptokinase infusion, he developed accelerated idioventricular rhythm which progressed to pulseless ventricular tachycardia lasting for one minute. Before defibrillation was performed, his rhythm reverted to sinus, after which a bolus of amiodarone was administered.*

Given his instability, streptokinase was discontinued after approximately 70 percent of the total dose had been delivered. ***Outcome:*** *The patient was transferred to the intensive care unit with stable hemodynamic, resolved chest pain, and more than 50 percent ST-segment resolution on ECG. Heparin, atorvastatin, maintenance amiodarone, and furosemide were added to his regimen. He continued to improve clinically and was discharged without complication.* ***Conclusion:*** *This case shows that fibrinolysis remains essential when PCI is unavailable, but streptokinase can cause hemodynamic and arrhythmic complications, highlighting the need for close monitoring and rapid intervention in resource-limited settings.*

1. INTRODUCTION

Once a working diagnosis of ST-elevation myocardial infarction (STEMI) is established, healthcare providers should prioritize the patient for immediate reperfusion therapy. While primary PCI is the preferred strategy, it may not always be feasible within the necessary timeframe, especially in remote areas, making fibrinolytic remain the first choice of therapy.[1] Unfortunately, alongside the complications that STEMI patients already face, fibrinolytics themselves can lead to several adverse effects. Fibrinolytic agents, especially streptokinase (SK), which is the most commonly used in our country, are associated with allergic reactions, hypotension, bleeding, and arrhythmias.[2] Here we present a challenging fibrinolytic therapy of infero-posterior STEMI and right ventricular (RV) infarction patient at Nunukan Regency General Hospital, North Kalimantan.

2. CASE PRESENTATION

A 45-year-old man presented to our emergency department with a chief complaint of chest pain for the past hour, accompanied by dyspnea and diaphoresis. Smoking was identified as his only risk factor, and he denied any other history of illness. Initial vital signs were blood pressure 110/60 mmHg, heart rate 77 bpm, respiratory rate 26, and SpO_2 99% on room air. His physical examination was within normal limits.

Electrocardiography (ECG) revealed sinus rhythm at 75 bpm, a normal axis, normal P-wave morphology, no pathological Q waves, ST elevation in leads II, III, and aVF, and ST depression in leads I, aVL, and V2–V5. Additional posterior and right-sided ECGs showed ST elevation in leads V2R–V4R and slight ST elevation in leads V7–V9 without pathological Q waves **(Figure 1)**. Based on these findings, the patient was diagnosed with an infero-posterior STEMI and RV infarction. Bedside echocardiography revealed akinetic inferior, infero-septal, and posterior walls with an estimated right atrial pressure (eRAP) of 15 mmHg **(Figure 2)**. His laboratory values were within normal limits, including creatinine of 0.9 mg/dL, sodium 138 mmol/L, and potassium 4.1 mmol/L. Cardiac enzyme testing was unavailable at our hospital. He was given a loading dose of 360 mg aspirin, 300 mg clopidogrel, and 40 mg IV furosemide. Fibrinolytic therapy with SK was prepared, and the door-to-needle time was achieved in under 30 minutes.

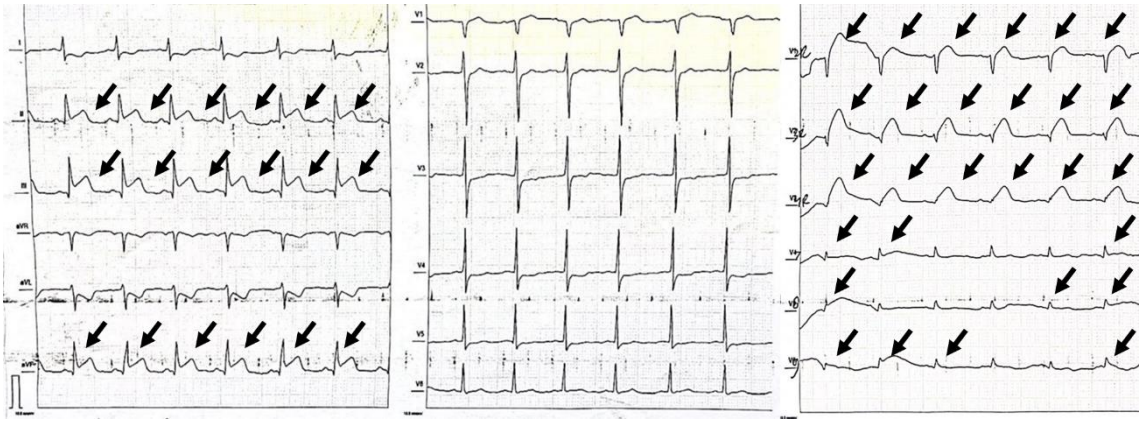

Fig. 1. Initial Electrocardiogram Showing Infero-Posterior STEMI and Right Ventricular Involvement
The initial ECG demonstrates ST elevation in leads II, III, and aVF, with ST depression in leads I, aVL, and V2–V5. Additional right-sided leads (V2R–V4R) and posterior leads (V7–V9) show ST elevation, consistent with infero-posterior STEMI and right ventricular infarction.
Arrow = ST elevation

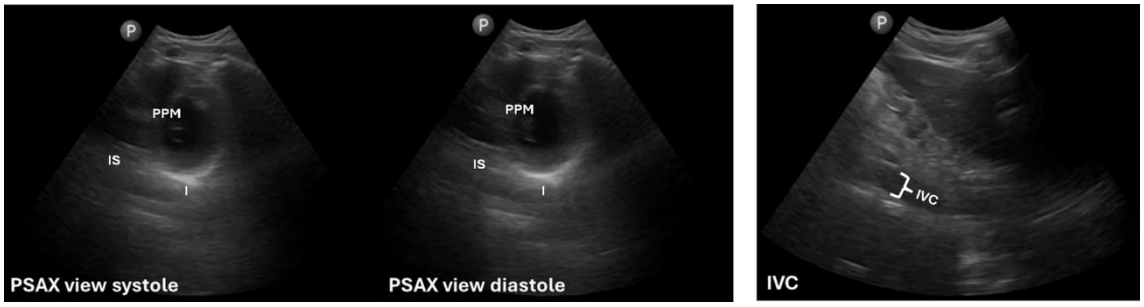

Fig. 2. Bedside Echocardiography Demonstrating Regional Wall-Motion Abnormalities
Echocardiography reveals akinesia of the inferior, infero-septal, and posterior walls with an estimated right atrial pressure (eRAP) of 15 mmHg, indicating right-sided pressure overload and confirming extensive infero-posterior involvement.
I = inferior wall, IS = infero-septal, PPM = posterior papillary muscle, IVC = inferior vena cava

Around five minutes after the SK infusion started, his blood pressure suddenly dropped to 80 mmHg / palpation. The SK infusion was temporarily stopped. A fluid challenge was given with 100 mL crystalloid, but his blood pressure did not improve. Considering his elevated eRAP, we did not continue the fluid challenge and instead provided supportive therapy. A combination of norepinephrine, dobutamine, and dopamine was titrated. His hypotension lasted for about fifteen minutes and when his systolic BP was greater than 90 mmHg, the SK infusion was resumed.

Approximately thirty minutes through the SK infusion, his monitor showed an accelerated idioventricular rhythm (AIVR) **(Figure 3)**. After around two minutes minutes, his rhythm unexpectedly changed to ventricular tachycardia (VT) with a rate of 150 bpm **(Figure 4)**. He experienced a cardiac arrest for approximately one minute; however, before defibrillation was performed, his rhythm reverted to sinus. A bolus of 150 mg amiodarone was given, followed by a 1 mg/min infusion.

Fig. 3. Accelerated Idioventricular Rhythm During Streptokinase Infusion
ECG monitor strip obtained during fibrinolytic therapy shows the onset of accelerated idioventricular rhythm, occurring approximately halfway through the streptokinase infusion and consistent with early reperfusion arrhythmia.

Fig. 4. Ventricular Tachycardia Following AIVR During Streptokinase Administration
The rhythm transitioned from accelerated idioventricular rhythm to sustained ventricular tachycardia at a rate of 150 bpm, leading to brief cardiac arrest prior to spontaneous return of sinus rhythm.

We decided to stop the SK infusion after 70% had been administered. His chest pain resolved, and a follow-up ECG showed resolution of the ST elevation **(Figure 5).** Notably, Q waves also formed in leads V7–V9 later on, confirming the significance of the earlier ST elevation. He was then transferred to the intensive care unit with stable vital signs. Therapy included heparin, dual antiplatelet therapy (DAPT), atorvastatin, maintenance amiodarone, and furosemide. We recommended the patient be referred to a PCI center, but he declined. He remained stable throughout his hospitalization and was discharged without complications after five days.

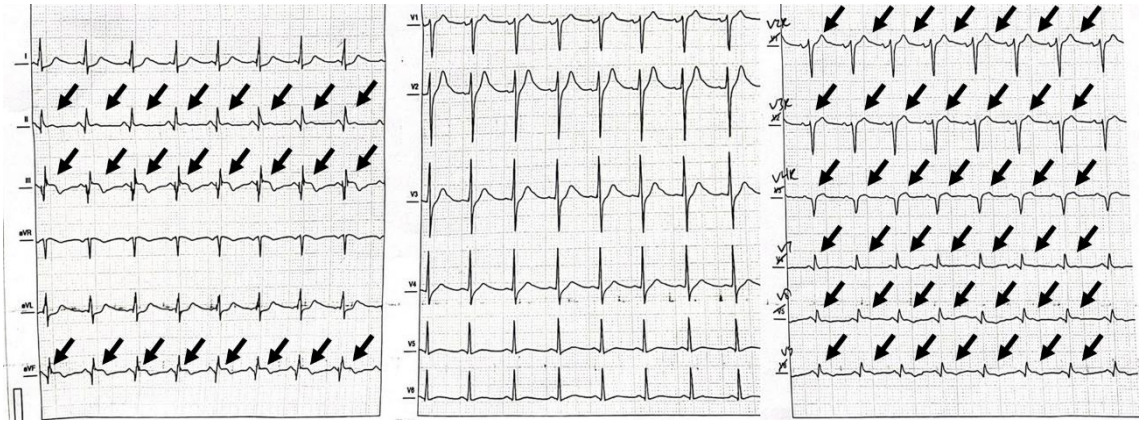

Fig. 5. Post-Fibrinolytic Electrocardiogram Demonstrating ST-Segment Resolution
Follow-up ECG shows more than 50 percent resolution of ST-segment elevation in the inferior and posterior leads. Q waves later appeared in V7–V9, supporting the significance of the prior posterior ST elevation.
Arrow = ST elevation resolution

3. DISCUSSION

While primary PCI remains the gold standard for treating STEMI, large-scale trials like GISSI, ISIS-2, and GUSTO have demonstrated a significant mortality benefit of fibrinolytic therapy compared to placebo. [3] Notably, when administered within the first two hours, fibrinolytics can reduce mortality by as much as 42%, compared to a 20% reduction when treatment is delayed.[4]

SK, the most widely used first-generation thrombolytic agent, is derived from β-hemolytic streptococci. It works by altering the fibrinolysis cascade: binding to plasminogen and triggering a conformational shift that activates the streptokinase–plasminogen complex. This activated complex then targets and dissolves fibrin clots through a specific lysine binding site.[5] Rahman et al.[2] found that hypotension occurred in 15.9% of patients undergoing fibrinolytic therapy with streptokinase, while bleeding was reported in 4.9%, allergic reactions in 4.2%, and cardiac arrest in 1.2%. Another study by Thakkar et al.[3] in an elderly population found that arrhythmias were observed in 20.6% of patients, acute kidney injury in 6.6%, ventricular septal rupture in 5.7%, ischemic stroke in 4.7%, free-wall rupture in 2.8%, and intracranial hemorrhage in 0.9%.

In patients with cardiogenic shock, fibrinolysis demonstrated similar all-cause in-hospital mortality rates of 28.8% compared to 28.5% for those undergoing primary PCI. Additionally, pre-hospital fibrinolysis was linked to lower rates of cardiogenic shock in the CAPTIM trial, STREAM trial, and WEST study. This indicates that the delays faced by the primary PCI group, in contrast to those receiving pre-hospital fibrinolysis, may contribute to the increased rates of cardiogenic shock. Nonetheless, a pharmaco-invasive strategy is still recommended and led to better outcomes in these situations.[6]

Reperfusion arrhythmias arise from the complex cellular and humoral changes that occur when coronary flow is abruptly restored. AIVR is the most frequent early manifestation, often appearing repetitively within the first six hours after thrombolysis, yet it is typically benign and not associated with sustained VT or ventricular fibrillation.[7] The abrupt return of blood flow generates marked inhomogeneity in action potential duration around the previously ischemic border zone, creating a vulnerable substrate that facilitates re-entry. In addition to these re-entrant mechanisms, non-reentrant processes such as abnormal automaticity and triggered activity related to intracellular calcium accumulation during ischemia also contribute to persistent tachyarrhythmias.[8] Beyond these electrophysiologic disturbances, reperfusion itself may aggravate myocardial injury through oxidative stress, endothelial and microvascular dysfunction, and intracellular calcium overload.[9] These processes promote delayed afterdepolarisation, destabilize mitochondrial function, and increase dispersion of refractoriness across the reperfused territory, thereby supporting both triggered and re-entrant VT. Structural injury following reperfusion, including myocyte hypercontracture, contraction band necrosis, and accelerated neutrophil infiltration, can further disrupt conduction pathways and increase susceptibility to ventricular tachycardia.

In conclusion, this case highlights the complexities and risks associated with fibrinolytic therapy, particularly with the use of SK in patients presenting with infero-posterior STEMI and RV infarction. While fibrinolytic therapy remains a critical alternative in settings where primary PCI is not feasible, its potential complications, such as hypotension, arrhythmias, and transient cardiac arrest, require close monitoring and prompt management. This case underscores the importance of tailoring supportive

therapies to address hemodynamic instability and arrhythmias, guided by an understanding of the mechanisms of reperfusion injury and arrhythmogenesis. As the evidence suggests, timely reperfusion significantly improve outcomes, aligning with the observations in this case where early intervention resulted in clinical stabilization and resolution of ischemia. This case serves as a reminder of the pivotal role fibrinolytic therapy continues to play in managing acute coronary syndromes, especially in resource-limited settings, while emphasizing the necessity for careful patient selection, early recognition of adverse events, and adherence to a pharmaco-invasive strategy to optimize outcomes.

4. CONCLUSION

This case illustrates how fibrinolytic therapy, while essential in environments where PCI is not rapidly accessible, may precipitate significant hemodynamic and arrhythmic complications in infero-posterior STEMI with right ventricular involvement. The clinical course emphasizes the importance of understanding reperfusion physiology, maintaining close surveillance, and responding rapidly to instability. These learning points are critical to optimizing safety and outcomes when using streptokinase in resource-limited contexts.

ETHICAL APPROVAL

Not Applicable.

CONSENT FOR PUBLICATION

The patient provided informed consent for the publication of the case report and associated pictures.

ACKNOWLEDGMENTS

We express our gratitude to the patient for consenting towards publication of their case.

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Conflict of Interest Statement:

The author declares that the case report was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.