

Case Report

Challenges in Managing Myocardial Stunning Following Cardiac Arrest in a Very High-Risk NSTEMI-ACS Patient: 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: Myocardial stunning is a reversible myocardial dysfunction that can occur due to various conditions, including focal ischemia—such as acute coronary syndrome (ACS)—and global ischemia, such as cardiac arrest. The proposed mechanisms include oxygen free radical damage during early reperfusion and altered calcium inflow leading to transient myofilament desensitization. **Case Presentation:** A 61-year-old woman with a history of uncontrolled hypertension and obesity presented with sudden loss of consciousness lasting one hour, preceded by chest pain and dyspnea. She experienced cardiac arrest and achieved return of spontaneous circulation (ROSC) after 10 minutes of cardiopulmonary resuscitation. Initial echocardiography showed a left ventricular ejection fraction (LVEF) of 32.9% and global hypokinesia. She was diagnosed with very high-risk non-ST-elevation ACS (NSTEMI-ACS), with suspected left main coronary artery obstruction or three-vessel disease. Her clinical course was complicated by acute pulmonary edema, respiratory failure, and cardiogenic shock. Management included intubation, dual antiplatelet therapy, anticoagulation, statin therapy, and inotropic support with dopamine and dobutamine. Her hemodynamic status improved within 18 hours, and she

was extubated. On the third day post-arrest, follow-up echocardiography revealed a restored LVEF of 69.5% with normal wall motion. The patient was discharged after 9 days and continued to do well on outpatient follow-up for at least 6 months. **Conclusions:** Myocardial stunning should be considered in post-cardiac arrest and ACS patients with transient ventricular dysfunction. Early supportive care and close monitoring are essential, particularly in settings lacking advanced cardiac interventions.

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1. INTRODUCTION

Myocardial stunning is a phenomenon that was first described as “prolonged, post-ischemic ventricular dysfunction that occurs after brief periods of nonlethal ischemia” by Braunwald and Kloner in 1982.¹ In the later days, it is also known that stunning occurs not only after transient coronary artery occlusion but also during extended periods of reduced myocardial blood flow and reperfusion after non-transmural myocardial infarction.² Two hypotheses underlying the mechanism of myocardial stunning are oxygen radical damage in a few minutes after reperfusion and desensitization of myofilaments by altered calcium inflow.³

Acute coronary syndrome (ACS), a clinical condition that consists of a wide range of manifestations, including unstable angina, non-ST elevation myocardial infarction (NSTEMI), and ST elevation myocardial infarction (STEMI), remains the leading cause of death worldwide.^{4,5} ACS rapidly causes cardiac contractile dysfunction in the region supplied by the impaired coronary artery, and with timely reperfusion, myocardial necrosis can be prevented, leading to myocardial stunning condition.⁶ In contrast to focal ischemia, such as in ACS, cardiac arrest causes global ischemia condition, and when the return of spontaneous circulation (ROSC) is achieved, post-resuscitation myocardial dysfunction also can occur, which has different myocardial stunning characteristics.⁷

Here, we report a case of myocardial stunning in post-cardiac arrest, very high-risk NSTEMI-ACS patient at Nunukan Regency General Hospital, a hospital on a remote island of Indonesia without a cath lab or cardiac surgery facility.

2. CASE PRESENTATION

A 61-year-old woman presented to our hospital with loss of consciousness in the past 1 hour. She previously complained of breathlessness and chest pain. The patient had a history of uncontrolled hypertension and obesity. Initial physical examination revealed GCS E3M2V5, blood pressure (BP) 300/180 mmHg, heart rate (HR) 139 beats per minute (BPM), respiratory rate (RR) 48 times per minute, SpO₂ of 39% with room air, elevated jugular venous pressure, regular heart sounds, and crackles in the whole quadrant of both lungs. After 5 minutes, the patient experienced cardiac arrest and required 10 minutes of cardiopulmonary resuscitation until ROSC was achieved. She was subsequently intubated. Post-cardiac arrest vital signs were BP of 80/palpation mmHg, HR of 120 bpm, and SpO₂ of 94% with ventilator support. Her electrocardiogram (ECG) revealed sinus rhythm, 128-135 bpm, a normal axis, left ventricle hypertrophy,

and ST depression in leads I, II, III, avL, avF, and V2-V6, with ST elevation in avR and V1 (**Figure 1A**). Echocardiography revealed left ventricle ejection fraction (LVEF) of 36.4% (Teich), 32.9% (Biplane), global hypokinetic, and estimated right atrial pressure (eRAP) of 15 mmHg (**Figure 2A**). The initial laboratory values are shown in **Table 1**.

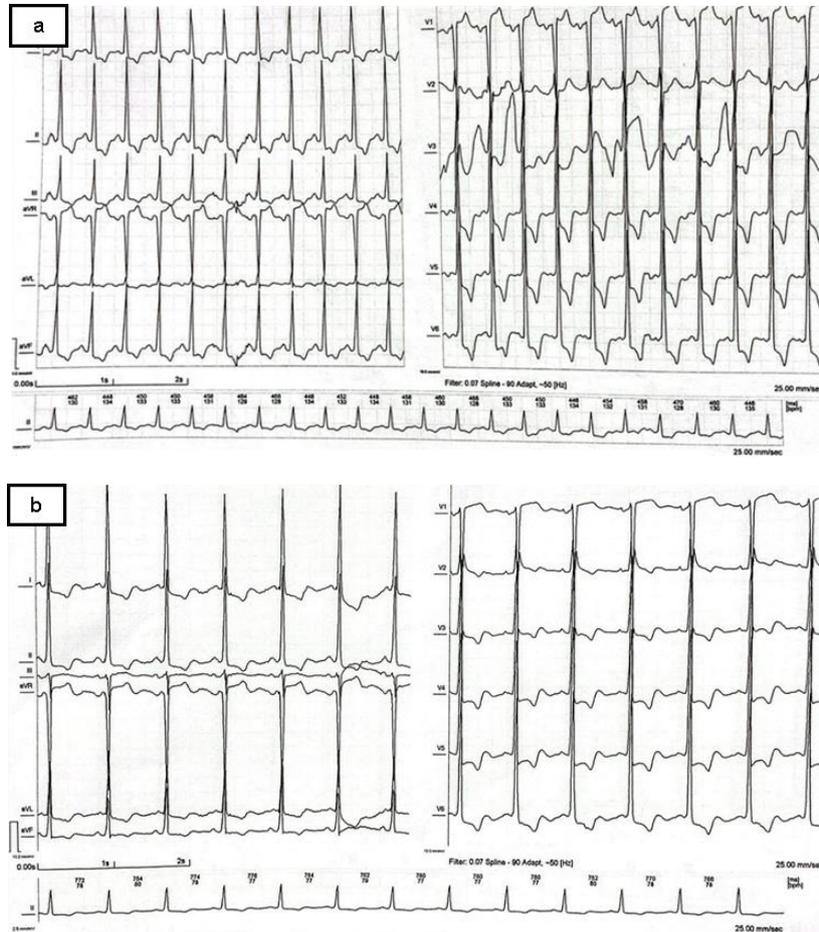


Fig. 1. Electrocardiogram of the patient revealed widespread ST depression with ST elevation in lead aVR and V1. a) on admission. (b) on discharge.

The patient was diagnosed with very high-risk non-ST-elevation ACS, suspected of having left main coronary artery (LMCA) obstruction with differential diagnosis of three-vessel disease (3VD), acute lung edema with respiratory failure, cardiogenic shock, and post-cardiac arrest. Dobutamine and dopamine were used for hemodynamic support. She also received 160 mg of aspirin followed by 80 mg daily, 300 mg of clopidogrel followed by 75 mg daily, 40 mg of atorvastatin, and anticoagulant therapy with unfractionated heparin. After 18 hours, her cardiogenic shock resolved, and she was extubated. Furosemide, isosorbide dinitrate, and hypertension medication using hydrochlorothiazide, amlodipine, and irbesartan were added to her therapy. Echocardiography on her 3rd day post-cardiac arrest revealed LVEF of 69.5%, resolved wall motion abnormalities into global normokinetic, concentric hypertrophy, grade 1 diastolic dysfunction, and an eRAP of 3 mmHg (**Figure 2B**). She was discharged after 9 days and still visited the outpatient clinic after 6 months. Unfortunately, she refused to be referred to another hospital with cathlab and cardiac surgery facilities.

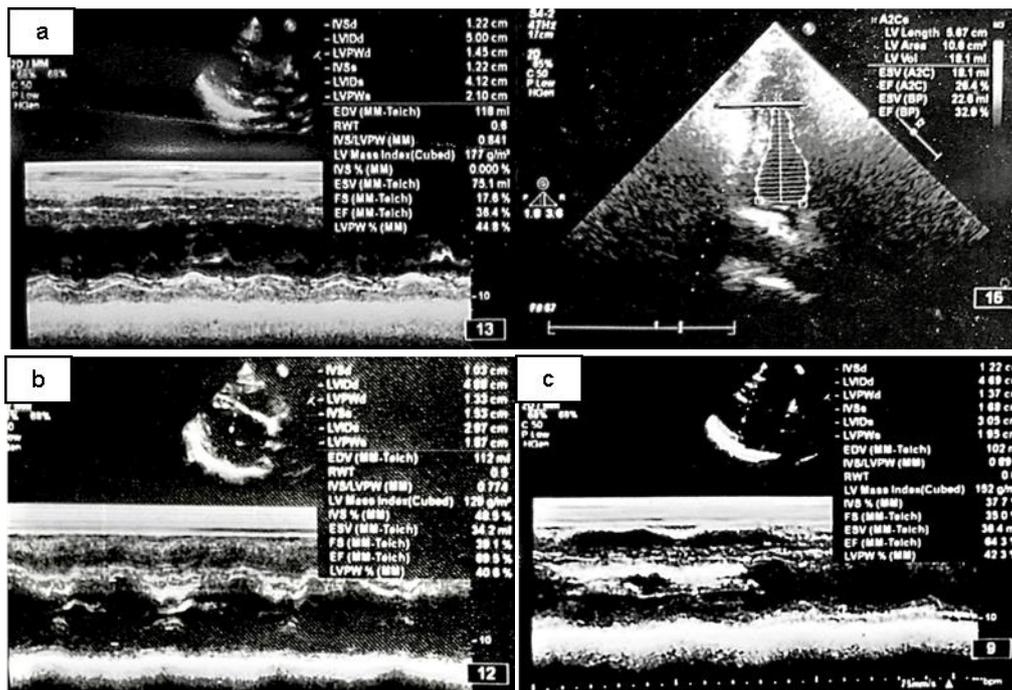


Fig. 2. Echocardiography of the patient. a) post-cardiac arrest revealed left ventricle ejection fraction (LVEF) of 38.4% (Teich), 32.9% (Biplane). (b) 3rd day post-cardiac arrest revealed LVEF of 68.9%. (c) 6 months post-cardiac arrest revealed LVEF of 64.3%

Table 1. Initial laboratory values on admission

Laboratory Test	Value	Normal Range
White blood count	21.58	4.3 – 10.3 x 10 ³ /μL
Hemoglobin	15.8	11.4 – 15.1 g/dL
Hematocrit	45.5	40 – 47%
Platelet	289	150 – 450 x 10 ³ /μL
Random blood sugar	145	<200 mg/dL
Creatinine	0.23	<1.2 mg/dL
Alanine Aminotransferase	88	0 – 41 U/L
Sodium	142	136 – 145 mmol/L
Potassium	3.53	3.5 – 5 mmol/L
Clorida	113	95 – 106 mmol/L
pH	7.31	7.35 – 7.45
pCO ₂	49.8	35 – 45 mmHg
pO ₂	126	80 – 100 mmHg
HCO ₃	25.6	21 – 28 mmol/L
Base Excess	-1	-3 s/d +3
Lactate	1.67	<2 mmol/L

3. DISCUSSION

Delayed recovery after ischemia was originally documented in animal research in the 1970s with dogs subjected to significant coronary artery occlusions lasting up to 15 minutes, followed by reperfusion. During ischemia, the myocardium perfused by the

occluded artery becomes dyskinetic, and following removal of the occlusion, the contractile function initially declines and then improves over a few days.^{2,3} In the histologic study, viable myocardial cells were observed at 72 hours following reperfusion.³ Stunning eventually is not a cardioprotective mechanism to downregulate cardiac contractile function, but it is an injury manifestation, demonstrated by the reversal of myocardial stunning with inotropic stimulation or calcium in dogs without affecting functional recovery or infarct size.²

Multiple pathophysiological hypotheses are leading to stunned myocardium. Reactive oxygen species (ROS) can attack cellular components such as ion transport channels, sarcolemma, and mitochondrial supercomplexes, thereby inducing cellular calcium overload. In addition, an increase in ROS production leads to endothelial dysfunction that reduces blood flow. Furthermore, ischemia followed by reperfusion can cause an increase in the intracellular calcium concentration by rapidly reactivating Na⁺-Ca²⁺ channels after acidosis. Alterations in calcium homeostasis in ischemic myocardial tissue result in reduced excitation-contraction coupling due to sarcoplasmic reticulum dysfunction and structural changes to contractile proteins.⁶ Notably, cardiac stunning is known to involve different aspects, not only postischemic ventricular dysfunction (myocardial stunning), but also vascular injury (vascular stunning), postischemic metabolic dysfunction (metabolic stunning), neurotransmission alteration (neuronal stunning), and electrophysiological impairment (electrical stunning).⁸

Stunning also occurs in global ischemic conditions, such as cardiac arrest. Cha et al.⁷ reported that post-resuscitation myocardial dysfunction occurred in 33% of patients who were resuscitated from non-cardiac causes of cardiac arrest, with echocardiographic patterns showing global dysfunction in 62.2% of patients, regional wall motion abnormalities in 22.2%, and takotsubo patterns in 15.6%. Gradual improvement in global left ventricular systolic function and recovery to normal within 3 days were shown in all but one patient. Another cause of myocardial stunning is adrenergic hyperactivity or a sympathetic storm, such as stress cardiomyopathy or Takotsubo cardiomyopathy, which are associated with emotional stress and neurogenic stunning following neurogenic events such as stroke, subarachnoid hemorrhages, or seizures.³

Stunning can be diagnosed retrospectively by observation of gradual improvement in LV function after the ischemic period is relieved and prospectively by finding evidence of flow-function mismatch by imaging techniques such as ventricular dysfunction (from ventriculography, echocardiography, nuclear, or other imaging modalities) with normal perfusion (from scintigraphy, positron emission tomography, or contrast echocardiography) in the same territory after brief ischemia.³ Stunning is usually well tolerated, and no therapy should be needed if it is known to be the reason for mild contractile dysfunction; however, if cardiac function is severely altered, moreover, under high-risk conditions, stunning should be treated; thus, it can cause severe morbidity and mortality. Treatment with inotropes, levosimendan, and other classes of medicines, such as calcium channel blockers, ACE inhibitors, and adenosine, resulted in an excellent response in the majority of patients.⁶

We believe that our patient experienced myocardial stunning by the fact that she presented with acute pulmonary edema, which indicates ongoing myocardial dysfunction due to her ACS.⁹ Moreover, she experienced cardiac arrest that caused global ischemia,

which worsened her condition. The improvement of her echocardiography features within 3 days allowed her to be diagnosed with myocardial stunning retrospectively. We cannot conclude a single etiology of her myocardial stunning condition; rather, it is a combination of various mechanisms. Rapidly achieved ROSC, early dual antiplatelet therapy, and anticoagulant administration are clearly related to better outcomes and preserved more viable myocardial cells.^{10–12} Given that her ECG characteristics raised suspicion of LMCA obstruction or 3VD and her condition fulfilled the very high-risk criteria, an immediate invasive strategy should be preferred.^{4,13} However, due to our conditions on a remote island, it is impossible to follow the recommended guidelines. Fortunately, our patient responded well with conservative management only and achieved a satisfactory outcome that made her able to return to daily activities.

4. CONCLUSION

Myocardial stunning is a reversible myocardial dysfunction that may be caused by various mechanisms, including focal and global myocardial ischemia. Diagnosis can be made retrospectively or prospectively in conditions with available facilities. Treatment should address the underlying mechanism; however, in severely altered cardiac function conditions, close monitoring and supportive treatment should be given.

ETHICAL APPROVAL

Not Applicable.

CONSENT FOR PUBLICATION

Informed consent has been obtained from the patient for publication of the case report and accompanying images.

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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.

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