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Case Report

Early Management of Shock Condition in STEMI Inferior Patient

Yudha Tria Nugraha[1*], Indra Prasetya[2], Heny Martini[2], Ardian Rizal[2]

[1] *Brawijaya Cardiovascular Research Center, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia.*
[2] *Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia.*

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ABSTRACT

Background: Acute myocardial infarction is one of the emerging cardiovascular events worldwide. Infarcts of the inferior wall were present between one-third and half of the patients with RV involvement. The worse outcome was strongly associated with cardiogenic shock, and 7% of cases were caused by RV failure.
Objective: This study aimed to describe the diagnosis and management of cardiogenic shock in STEMI
Case Presentation: A male in his 40s was brought to our hospital 12 hours after the onset of persistent epigastric pain. He had a history of hypertension and a family history of diabetes Mellitus. Diagnostic procedures included blood tests, ECG, X-rays, coronary angiogram, and echocardiography. He underwent PCI to implant DES in his proximal RCA, which had 100% occlusion. The patient was admitted to the intensive cardiovascular care unit for 30 hours and died due to various complications.
Conclusion: Current case was very complicated and seriously life-threatening. After acute myocardial infarction, Important problems kept coming up one after the other, and they all affected each other. Patients with cardiogenic shock must be found quickly and treated quickly and aggressively.

1. Introduction

Myocardial infarction results from coronary artery occlusion with decreased perfusion to that region of the myocardium. 40% of myocardial infarctions involve the inferior wall and have a better prognosis than other regions. One-third and half of the patients had inferior-wall infarct with RV involvement.1 Clinical triads of RV infarction, such as hypotension, clear lung fields, and elevated jugular venous pressure, are considered specific markers. The cause of cardiogenic shock was predominantly RV failure in 7% of case.2

2. Case Illustration

A 46-year-old male was referred to our hospital because epigastric pain persisted for 12 hours while sleeping with VAS 9/10 (November 8, 2022, at 01.00 am). The duration of pain was more than 20 minutes. The pain was accompanied by nausea, vomiting, and dyspnea. There was no stabbing-like or tearing-like sensation, and it was not affected by changes in position and respiration. The patient tried to rest, but the pain didn't relieve. The following day at 07.00 am, the complaint worsened with VAS 10/10. Then his family brought him to the previous hospital (6 hours onset), and ECG examination showed STEMI inferior and RV infarct, with junctional rhythm 40bpm. He was diagnosed with a heart attack and got loading aspirin 320 mg, clopidogrel 300 mg, and atorvastatin 80 mg.

He was diagnosed with hypertension two years ago, on no medications. He had no history of chest pain, dyspnea on effort (DOE), paroxysmal nocturnal dyspnea (PND), orthopnea, leg swelling, palpitation, and syncope. His family history includes his parents and brothers suffering from diabetes Mellitus.

The patient arrived with GCS 356, BP 113/56 on Dopamine 5mcg/kg/min, HR 75 bpm with junctional rhythm, RR 36 with increasing WOB and fatigue, so we decided to intubate the patient. Physical examination revealed blood pressure (BP) 113/51 mmHg on dopamine 5mcg/kg/m, HR 76 bpm, RR 36 pm, T 36,4C, saturation (SpO2) 100% on NRBM 15 liter per minute (ppm) then switched to ventilator mode PC AC, with urine output 0 cc/5 hours. The patient's weight was 90 kg, height 170 cm, and BMI 31,1 kg/m2, categorized as obese. There was no pale conjunctiva and icteric sclera. Jugular venous pressure (JVP) was R + 2cmH20. Precordial auscultation revealed regular S1 and S2 without S3, S4, murmur, or rub. There were clear lung fields and no peripheral edema. A heart enlargement was found from auscultation with palpable apex cordis at intercostalis (ICS) V and mid clavicula line (MCL). Cold acrals were not found in extremities. The electrocardiography (ECG) examination showed a Junctional rhythm with acute inferior myocardial infarction. (Figure 1). An X-ray examination in the AP position showed an infiltrated right inferior Pulmo and an increased cardiothoracic ratio (CTR) of 66% (Figure 2).

*Corresponding author at: Brawijaya Cardiovascular Research Center, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia
E-mail address: Triayudha21@gmail.com (T.Y. Nugraha).

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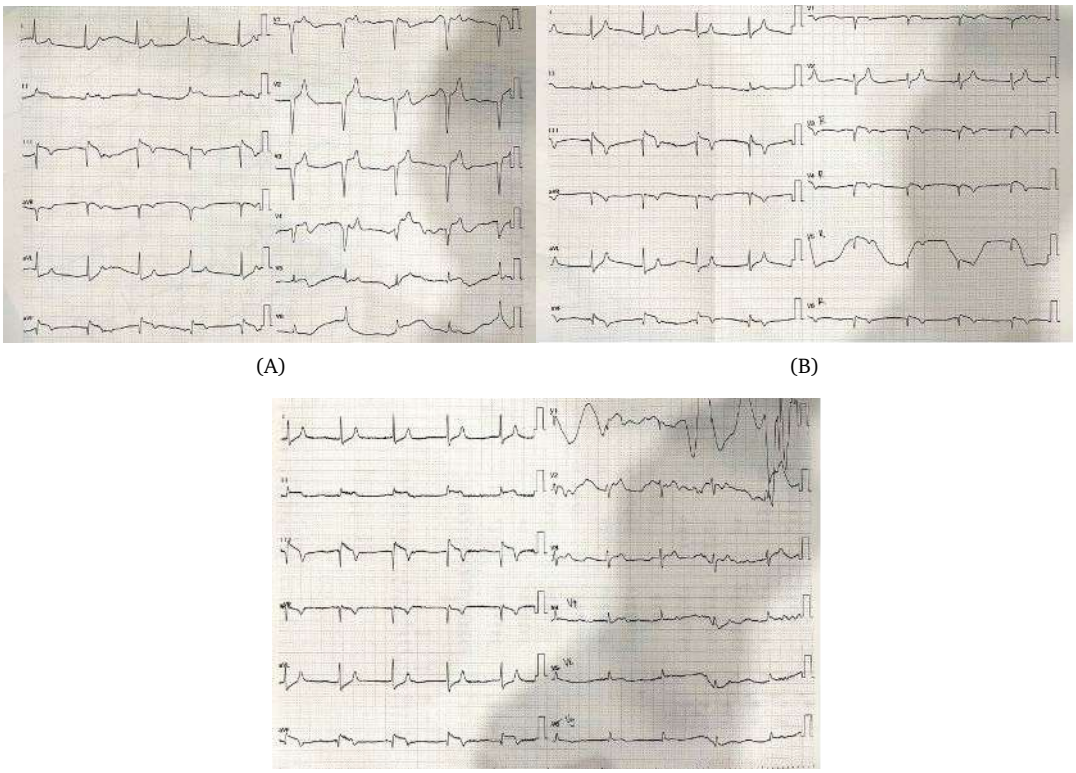

(A) (B)

(C)

Figure 1. ECG (a: left; b: right; c: posterior) showed Junctional Rhythm, HR 40 bpm, Normoaxis, QRS 80 ms, QT 400 ms, QTc 327 ms, ST elevation at II (2 mm), III (3 mm), aVF (2 mm), T inversion III, aVF, ST depression I (2 mm), aVL (3 mm).

Laboratory examination showed increased leucocyte (27,130/μL), decreased platelets (140,000/uL), increased serum (72,1) increased creatinine (5,58 mg/dl), and decreased e- GFR (14,275 ml/min/ 1.73 m2). Liver function tests showed a massive increase in SGOT (16301 U/L) and SGPT (6138 U/L). The Sodium level decreased (132 mmol/L), and potassium increased (6,08 mmol/L). Partial prothrombin time 22,3 and activated partial thromboplastin time 80,8 and INR 2,14. This patient had high random blood sugar (501 g/dl).

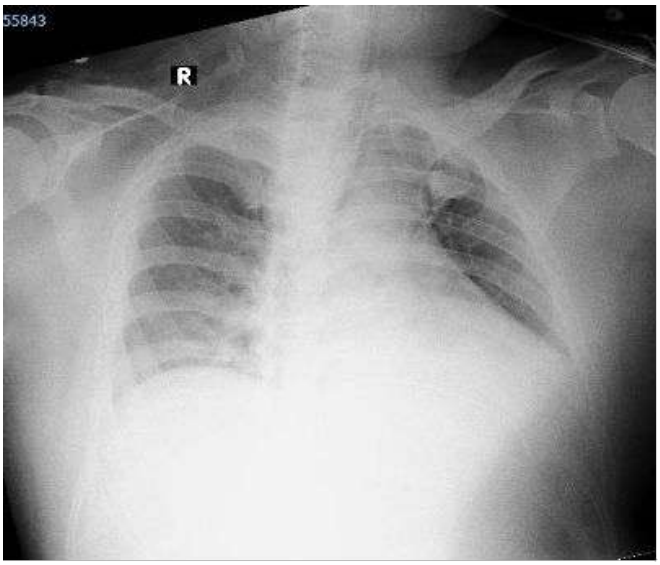

Figure 2. Chest X-Ray in AP position showed infiltrated right inferior lung and an increased cardiothoracic ratio (CTR) of 66%

Troponin was 22.76 mcg/L, and CK-MB was 795 U/L. Blood gas analysis showed uncompensated metabolic acidosis.

CRUSADE score was counted to predict major bleeding. The patient got 70 points and underwent a coronary angiogram, which revealed 50% stenosis at distal LAD, 40% stenosis at distal LCx, and total occlusion 100% at proximal RCA with thrombus appearance post 1 DES implantation was done on proximal RCA performed in (figure 3). He was transferred to the cardiac intensive care unit for ongoing management. Initial therapy was given such as O2 ventilator mode PC-AC, total fluid 3000 cc/24 h, IVFD NS 0.9% 1cc/kgbw/h ~ 90cc/h, oral Intake 1000 cc /24 h, equal fluid balance, cardiac diet nutrition 1800 kcal/day, bolus Heparin 5000 IU continued with drip Heparin 1000 IU/h, drip Dopamin 5 mcg/kg/m, drip Dobutamin 5 mcg/kg/m, drip Insulin 0,1 IU/kg/h with target RBG 140-180 mg/dl, and correction of hyperkalemia with Insulin, Ca Gluconate insulin and IV cefoperazone 2x1 gr. Oral drugs such as ASA 80 mg once daily, ticagrelor 90 mg twice daily, and atorvastatin 40 mg once daily were given.

Echocardiography revealed systolic RV function, MR mild, TR mild, calcification in the aortic valve, AR mild, and PR. Other laboratory finding HbA1C 10,7 mmol/L, triglyceride 410 mg/dl, and HDL 18mg/dl. The patient was assessed with STEMI inferior and RV infarct Killip class of IV as the primary diagnosis. Secondary diagnoses were respiratory failure, heart failure, cardiogenic shock, hyperglycemia in the critically ill, acute kidney injury, transaminitis, and hyperkalemia. Disease severity was stratified by TIMI risk score (7 points) and GRACE score (234 points). Overall, his hospital stay was complicated. The patient's condition was getting worse. He suffered refractory shock and survived cardiac arrest several times. He died after 30 hours of monitoring in the cardiac intensive care unit.

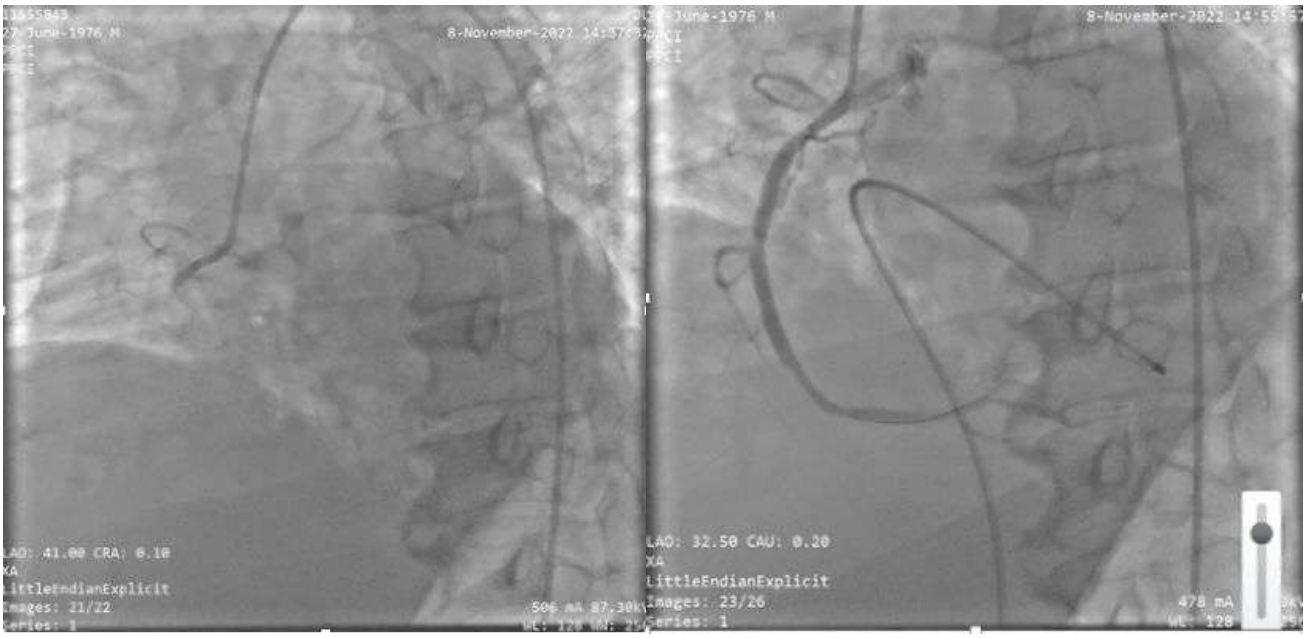

Figure 3. Total occlusion 100% at proximal RCA with thrombus appearance post 1 DES implantation was done on proximal RCA.

3. Discussion

According to studies that looked backward, only 1% to 5% of patients with AMI arrived at the hospital in cardiogenic shock. However, another 5% to 7% of patients developed shock in the hospital. Our extensive, prospective, multicenter study reveals that just nine percent of patients were already experiencing shock when they arrived at the hospital. In contrast, the vast majority of patients developed shock after presentation. Despite this, 46.6% of our patients who suffered from CS did so during the first six hours after the commencement of their infarction. Patients with an inferior MI on clinical grounds or whose right coronary artery was the culprit were substantially more likely to develop shock earlier during their illness. It is possible to lower the mortality rate of these individuals by providing intensive medical treatment and implementing reperfusion methods (11–13). Even though patients who had been clinically diagnosed with an "isolated" RV MI were not included in the research, it is possible that RV involvement played a crucial role in many patients who were assumed to suffer primarily from LV involvement. There is a possibility that an excessive vagal tone plays a part in this scenario by reducing the compensatory tachycardia, inotropy, and vasoconstriction that are usually associated with myocardial dysfunction.[3]

Recognizing the disease early on in its progression and having a solid grasp of what caused it are two of the most crucial components of the initial treatment of a patient suffering from cardiogenic shock. A rapid assessment of the patient's medical history, physical examination, and chest radiograph must be performed to identify the symptoms of heart failure, pulmonary edema (which may occasionally be accompanied by clear lung fields on review, in this case, we did not found the sign of acute lung edema), and tissue hypoperfusion. These symptoms include low blood pressure, rapid heart rate, agitation, confusion, oliguria, cyanosis, and cool and clammy skin. It is essential to have a solid understanding of the electrocardiographic symptoms of acute myocardial ischemia, myocardial infarction, new left bundle branch block, and arrhythmias.[2]

This report discusses a clinical ST-elevation myocardial infarction (STEMI) with RV infarction on presentation linked with prolonged shock. A review of the current literature indicates that approximately one-third and half of the patients with inferior-wall infarct have some RV involvement. Electrocardiography frequently shows ST-segment elevation >1 mm in lead V4R, representing an ischaemic injury of the posterobasal septum. The mortality rate was higher than that in the presence of RV. The worse outcome is mainly linked to refractory cardiogenic shock. RV ischemic dysfunction is caused by the loss of the important compensatory contribution of enhanced right atrial contraction in the typical scenario of a hemodynamically substantial RV infarction. RCA blockage affects right atrial and RV branch perfusion, resulting in RV ischaemic dysfunction. Patients with proximal RCA culprit lesions had poorer baseline features, lower rates of spontaneous recanalization, and higher clot burdens than patients who did not have these lesions.[4]

In this case, the patient did not use Intra aortic balloon pumping (IABP) because all the IABP tools had already been used. In the current standards, cardiogenic shock caused by an acute myocardial infarction is a class 1 indication for IABP. But there aren't many randomized clinical trials, so most data come from retrospective or prospective registries. So, IABP is only used in 20% to 40% of cases of cardiogenic shock right now. The idea behind this study is that adding IABP to early revascularization with either percutaneous coronary intervention or coronary artery bypass grafting will help patients in cardiogenic shock have a better clinical outcome. In cardiogenic shock, the most common way to help the left ventricle is with an Intra aortic balloon pump, which is also highly suggested by guidelines. The large-scale IABPSHOCK II study aims to test the idea that patients with cardiogenic shock caused by acute myocardial infarction will have a better clinical outcome if they get IABP therapy instead of just the best medical care. This is based on the idea that early revascularisation will improve blood flow to the heart.[5]

Hypertension and obesity contributed as risk factors in this patient. Class I obesity has significantly higher risk-adjusted hospital mortality rates than class III obese patients. The proportion of patients with STEMI based on BMI is dominated by overweight 38.7%.6 The frequency of antecedent hypertension varies from 31 to 59% among patients diagnosed with acute myocardial infarction (AMI). To a large extent, the connection between hypertension and myocardial infarction can be understood by focusing on two primary factors: (1) the presence

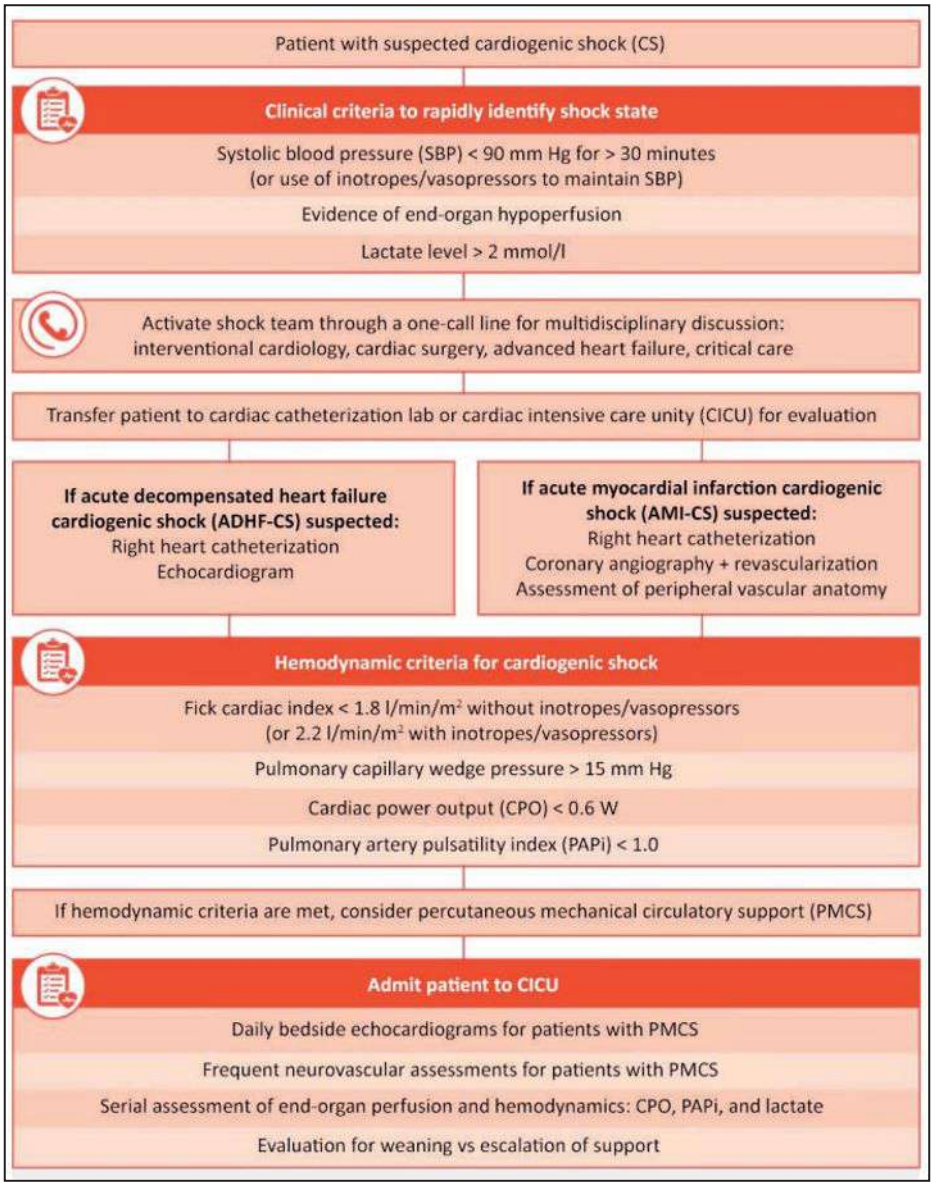

Figure 4. Cardiogenic Shock Algorithm developed by the INOVA Heart and Vascular Institute

of risk factors that are common to both diseases, such as genetic risk profiles, insulin resistance, sympathetic hyperactivity, and vasoactive chemicals (such as angiotensin II). (2) Hypertension is linked to accelerated atherosclerosis, which plays a role in the evolution of myocardial infarction and is related to this condition. Moreover, STEMI hypertensive patients had a higher risk of developing acute renal failure or contrast-induced nephropathy. Patients who suffered from hypertension and STEMI had a higher incidence of cardiogenic shock, pulmonary edema, ventricular tachycardia and fibrillation, and third-degree atrioventricular block than those who did not.[7]

Shock in patients with myocardial infarction involves many organs. Metabolic acidosis is mainly present in cardiogenic shock after myocardial infarction linked to poor prognosis. Ischemia and hypoxia occur across all body tissues and organs due to cardiogenic shock from a widespread myocardial infarction. After the patient's reperfusion, acidosis and severe renal insufficiency were seen briefly. In addition, there was a state of hyperkalemia. Therefore, hemodialysis may represent a viable therapeutic alternative for the patient. Mechanical ventilation was crucial to treating acute respiratory failure due to cardiogenic shock.[8]

A drop in cardiac output and arterial hypoxemia leads to tissue hypoxia, metabolic acidosis, and a decrease in plasma bicarbonate due to increased lactic acid in acute myocardial infarction (AMI). AMI stands for acute myocardial infarction. An increase in carbon dioxide not only brings about an increase in acidosis but also brings about a reduction in arterial oxygen tension, which is a dangerous combination. The level of peripheral blood pH decreases as the mortality rate increases..6 Elevated catecholamine and cortisol levels stimulate the sympathetic nervous system and the hypothalamus-pituitary axis. This condition stimulates gluconeogenesis, glycogenolysis, and lipolysis processes, resulting in stress hyperglycemia. The prevalence of hyperglycemia varies from 3-71%. Mortality rises for every 18mg/dl increase in glucose level.[9]

Patients with higher maximum potassium levels were older. They had a more significant load of comorbidities, including a higher burden of heart failure, lung illness, acute kidney injury, and renal disease. Patients with higher maximum potassium levels also had higher potassium levels overall. They also had lower hemoglobin levels, a lower median estimated glomerular filtration rate, and higher peak

amounts of glucose and troponin. In hospitals, mortality exceeded 15% in patients with potassium levels> 5,5 mEq/L.10 Systemic hypoperfusion in myocardial infarct activates neurohormonal response, including RAAS and the adrenergic autonomic system. Hypoperfusion of the kidneys and vasoconstriction of the glomeruli leading to reduced glomerular filtration, promoting potassium retention and hyperkalemia.[9]

Imbalance electrolyte was found in this patient, such as hyperkalemia. According to the previous study, The liver is a particularly significant organ because it acts as a reserve for the blood volume that the body produces. Because it receives approximately 25% of the total cardiac output, it is sensitive to changes in the hemodynamic environment. This pathophysiology is linked to patients' elevated liver enzymes (SGOT and SGPT). While serum alanine transaminase SGPT is mainly found in the liver, serum aspartate transaminase, also known as SGOT, is present in different organs in addition to the liver. It is usual practice to evaluate liver cell damage by calculating the SGOT to SGPT levels ratio. Patients diagnosed with STEMI had a mean De Ritis ratio (SGOT/SGPTT) that was significantly greater.[11]

Because of a combination of factors, including a rapid decrease in CO and tissue perfusion and passive venous congestion, acute cardiac failure brought on by a STEMI may lead to acute cardiogenic liver injury (ACLI). This condition is abbreviated as ACLI. Only around one-fifth to one-third of the total liver blood flow is supplied by the blood that flows via the hepatic arteries, despite this blood having high pressure and being oxygenated very effectively. The low-pressure, less oxygenated blood from the portal veins supplies roughly two-thirds of the body's blood supply. A drop in portal blood flow of between 25 and 60 percent may be compensated for by the autoregulation of the hepatic artery that is mediated by the hepatic arterial buffer response. In contrast, the portal vein depends on the hepatic-portal venous pressure gradient and the mesenteric circulation if there is a reduction in the amount of blood flowing through the liver. Because the liver is susceptible to variations in perfusion, the dual vasculature of the liver serves to protect it against hypoperfusion.[12]

Treating shock in patients who have experienced RV infarction should begin with volume replacement, and Recent studies have illuminated the negative consequences of excessive volume loading. We give fluid 1000cc/1h, and then boluses are stopped because there is no improvement in systolic blood pressure. Early revascularisation is also employed in treating RV infarction, a condition in which total revascularisation of the injured arteries, including the significant RV branch, is essential for restoring RV function. Early revascularisation is also used in the treatment of RV infarction. Electrical stabilization, which includes an appropriate heart rate and the maintenance of atrioventricular synchronization, is another important aspect in sustaining cardiac output in patients who have suffered from RV infarction. Electrical stabilization, which includes the care of an appropriate heart rate and preserving atrioventricular synchronization, is another important aspect of conserving cardiac output in the case of RV infarction. In addition, numerous extracorporeal support devices have been employed to assist RV failure caused by RV infarction. These devices have contributed to the improvement of RV shock and have been used to sustain patients. 1 Patient who suffered from acute myocardial infarction and cardiogenic shock as a result of primarily RV failure had a more favorable short-term prognosis than patients who were affected by LV involvement.[13]

In a nutshell, the situation was highly convoluted and posed a significant risk to human life. Following an AMI, a series of severe difficulties manifested themselves one at a time and interacted with one another. It is essential to diagnose people developing cardiogenic shock as soon as possible and begin intensive treatment as quickly as possible. The relatively modest gains in CS outcomes over the past few decades call for a more in-depth investigation into the syndrome from a scientific perspective. Some of the components that prior studies have helped identify as being necessary to specify and validate the ideal treatment for CS are early evaluation and appropriate initiation of AMCS devices, integration of hemodynamic and metabolic data for diagnosis and risk stratification, and an organized algorithmic approach to decision-making (figure 4). The absence of a standardized CS treatment protocol may be a barrier to the general application of a single classification system. Still, there is little doubt that the variety of classification methods being developed is a step toward implementing CS classification more widely.[14]

4. Conclusion

Most people who acquire CS do so a reasonable amount of time after an MI. Approximately fifty percent of those who ultimately developed shock while in the hospital developed it within six hours, and seventy-five percent did so within twenty-four hours. Patients who are diagnosed with early shock have a somewhat greater mortality rate. In many cases, a therapeutic window is available, which makes it possible to intervene either before the onset of CS or early on in its progression. Cardiogenic shock was a mortality factor in patients with STEMI inferior and RV infarct. This case report showed that various extracardiac organ involvement and persistent shock brought worse outcomes in the patient. Therefore, early identification and proper management in patients with shock following RV infarction are essential.

5. Declarations

5.1. Ethics Approval and Consent to participate
Not applicable

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: YTN, IP. Design: YTN, IP. Control/supervision: IP, HM, AR. Data collection/processing: YTN, IP.Analysis/interpretation: YTN, IP. Literature review: YTN, IP, HM. Writing the article: YTN, IP.Critical review: IP, HM, AR. 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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References

1. Topalian S, Ginsberg F, Parrillo JE. Cardiogenic shock. Crit Care Med. 2008;36(1 SUPPL.). doi:10.1097/01.CCM.0000296268.57993.90

2. Berkowitz MJ, Picard MH, Harkness S, Sanborn TA, Hochman JS, Slater JN. Echocardiographic and Angiographic Correlations in Patients With Cardiogenic Shock Secondary to Acute Myocardial Infarction. Am J Cardiol. 2006;98(8):1004-1008. doi:10.1016/J.AMJCARD.2006.05.014

3. Josiassen J, Helgestad OKL, Møller JE, et al. Cardiogenic shock due to predominantly right ventricular failure complicating acute

myocardial infarction. Eur Heart J Acute Cardiovasc Care. 2021;10(1):33-39. doi:10.1093/ehjacc/zuaa010

4. Das SR, Alexander KP, Chen AY, et al. Impact of body weight and extreme obesity on the presentation, treatment, and in-hospital outcomes of 50,149 patients with ST-segment elevation myocardial infarction: Results from the NCDR (National Cardiovascular Data Registry). J Am Coll Cardiol. 2011;58(25):2642-2650. doi:10.1016/j.jacc.2011.09.030

5. Thiele H, Schuler G, Neumann FJ, et al. Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock: Design and rationale of the Intra-aortic Balloon Pump in Cardiogenic Shock II (IABP-SHOCK II) trial. Am Heart J. 2012;163(6):938-945. doi:10.1016/j.ahj.2012.03.012

6. Zeller M, Labalette-Bart M, Juliard JM, et al. Metformin and contrast-induced acute kidney injury in diabetic patients treated with primary percutaneous coronary intervention for ST-segment elevation myocardial infarction: A multicenter study. Int J Cardiol. 2016;220:137-142. doi:10.1016/j.ijcard.2016.06.076

7. Yu H, Wang Q, Li X, et al. Case Report ST-Segment Elevation in a Patient with Myocardial Infarction after Primary PCI Due to Cardiogenic Shock, Hyperkalemia, and Acidosis. Vol 11.; 2018. www.ijcem.com/

8. cardiogenick shock ACS.

9. Dixon DL, Abbate A. Potassium levels in acute myocardial infarction: Worth paying attention to. Eur Heart J Cardiovasc Pharmacother. 2015;1(4):252-253. doi:10.1093/ehjcvp/pvv029

10. Grodzinsky A, Goyal A, Gosch K, et al. Prevalence and Prognosis of Hyperkalemia in Patients with Acute Myocardial Infarction. Am J Med. 2016;129(8):858-865. doi:10.1016/J.AMJMED.2016.03.008

11. Djakpo DK, Wang ZQ, Shrestha M. The significance of transaminase ratio (AST/ALT) in acute myocardial infarction. Archives of Medical Science – Atherosclerotic Diseases. 2020;5(1):279-283. doi:10.5114/amsad.2020.103028

12. Bannon L, Merdler I, Bar N, et al. The cardio-hepatic relation in STEMI. J Pers Med. 2021;11(12). doi:10.3390/jpm11121241

13. Inohara T, Kohsaka S, Fukuda K, Menon V. The challenges in managing right ventricular infarction. Eur Heart J Acute Cardiovasc Care. 2013;2(3):226-234. doi:10.1177/2048872613490122

14. Kimura K, Kimura T, Ishihara M, et al. JCS 2018 guideline on diagnosis and treatment of acute coronary syndrome. Circulation Journal. 2019;83(5):1085-1196. doi:10.1253/circa.CJ-19-0133