Acute Shock Liver in Inferior ST-Segment Elevation Myocardial infarct with Total Atrioventricular block: A Case Report

Mochamad Rizky Hendiperdana[1], Sumardjo[2]*

[1] Department of Cardiology Regional Public Hospital Pandan Arang of Boyolali, Indonesia.
[2] Gastroenterohepatology division, Department of Internal Medicine Regional Public Hospital Pandan Arang of Boyolali, Indonesia.

Corresponding Author:

Mochamad Rizky Hendiperdana, MD. Department of Cardiology, Regional Public Hospital Pandan Arang of Boyolali, Indonesia. Email: mhendiperdana@gmail.com.

ABSTRACT

Liver dysfunction frequently accompanies heart diseases, especially in hemodynamically unstable acute heart failure or cardiogenic shock. This condition is marked by significant elevation of liver transaminases and brings high morbidity and mortality for > 50 % of cases. Despite the high mortality rate, early recognition with prompt management results in the recovery of liver function. A 53-year-old man presented with late-onset non-reperfused inferior STEMI. The patient presented with persistent chest pain and shortness of breath. The electrocardiogram showed atrioventricular (AV) block grade III and ST-segment elevation evolution in the inferior lead. The patient was diagnosed with a late-onset inferior STEMI with cardiogenic shock and total AV block complication, acute shock liver, lactic acidosis, and acute renal failure. We administered inotropic and chronotropic support drugs as well as post-MI anti-remodelling therapy to treat heart failure (HF) and left ventricular (LV) systolic dysfunction, such as angiotensin-converting enzyme inhibitor and aldosterone antagonist, after systemic perfusion improved. Anti-ischemic therapy, such as antithrombotics, was also administered. Renal and liver function test evaluation after a week of patient discharge showed normalization of these parameters. There is no definite treatment strategy for shock liver. The management strategy is directed at the treatment of underlying causes. Hemodynamic insult is the mainstay therapeutic target. Recovery of liver transaminases was demonstrated after the underlying insult had been eliminated.

Keywords: *shock liver, hypoxic hepatitis, cardiogenic shock, myocardial infarction.*

INTRODUCTION

Liver dysfunction frequently accompanies heart diseases such as pericarditis, acute myocardial infarction, and heart failure (HF); this is referred to as the cardiohepatic interaction.[1,2]

Acute ischemic hepatitis or hypoxic hepatitis (HH) is described as a consequence of hemodynamically unstable acute heart failure or cardiogenic shock (78% of cases) and is marked by significant liver transaminase elevation.[3–5] Hepatic hypoperfusion becomes the primary mechanism of this phenomenon, and has high morbidity and mortality.[3]

Liver transaminase elevation in HH may have a reversible or transient course after the underlying causes have been eliminated. These liver enzymes usually peak early in the course of hemodynamic insult, often within 24 hours, but decrease to nearly half of their peak values within 24 to 72 hours.[3]

We report a case of HH in a patient with post–myocardial infarction cardiogenic shock whose liver function recovered after a few days of hemodynamic stabilization.

CASE ILLUSTRATION

A 53-year-old male patient was referred from another hospital after 3 days of ICU

admission with inferior (ST-elevation myocardial infarction) STEMI without reperfusion therapy for worsening dyspnea and hypotension. In our hospital emergency department, the patient presented with persistent chest pain and shortness of breath. The patient had a history of type II diabetes mellitus (DM) and smoking one pack of cigarettes per day for twenty years.

Vital signs at admission were blood pressure (BP), 80/60 mmHg; heart rate, 47 beats per minute; SpO_2 93%; and respiratory rate 24. An electrocardiogram (ECG) showed a grade III AV block with QRS duration of 80 ms and ST-segment elevation evolution in the inferior lead (**Figure 1**). Initial blood test results are presented in **Table 1**. Blood gas analysis results showed pH 7.29, PO_2 63 mmHg, PCO_2 20 mmHg, HCO_3 9 mmol/L, BE −15, and lactate 2.8 mmol/L.

The patient's chest x-ray (**Figure 2**) demonstrated mild cardiomegaly with increased pulmonary vascular markings. Echocardiography findings showed concentric LV hypertrophy with reduced LV systolic function (Left ventricular ejection fraction of 31% by Simpson; averaged LV global longitudinal strain (GLS) −6.0%), an akinetic inferior segment with other segments hypokinetic, good RV contractility (RV S' 11 cm/s), grade II diastolic dysfunction (MV E/A > 1), E/e' 18.61, and normal valves (**Figure 3**).

The patient was diagnosed with a late-onset inferior STEMI with cardiogenic shock and total AV block (TAVB) complications, acute HH, lactic acidosis, and acute renal failure (ARF).

The patient was admitted to the intensive cardiovascular care unit (ICVCU) for 3 days.

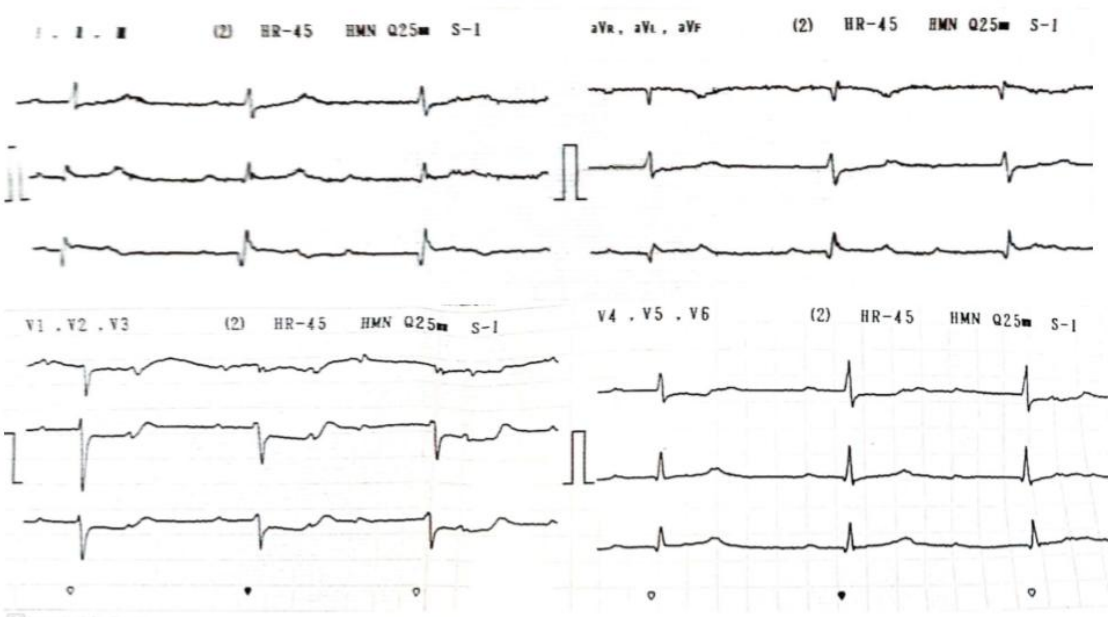

Figure 1. Patient Electrocardiogram (ECG) at initial presentation.

Table 1. Initial Presentation Blood Test Examination Result

Blood test	Result	Reference range
Haemoglobin (g/dL)	12.7	13.8-17.2
Haematocrit (%)	38	41-50
Leukocyte (cell/microliter)	12,030	4,500-11,000
Troponin I level (ng/mL)	>10.0	< 0.02
Thrombocyte (cell/microliter)	215.000	150,000-450,000
Serum creatinine (mg/dL)	4.7	0.3-0.9
Blood urea nitrogen (BUN) (mg/dL)	167	6-24
SGOT (U/L)	1143	5-40
SGPT (U/L)	1085	7-56

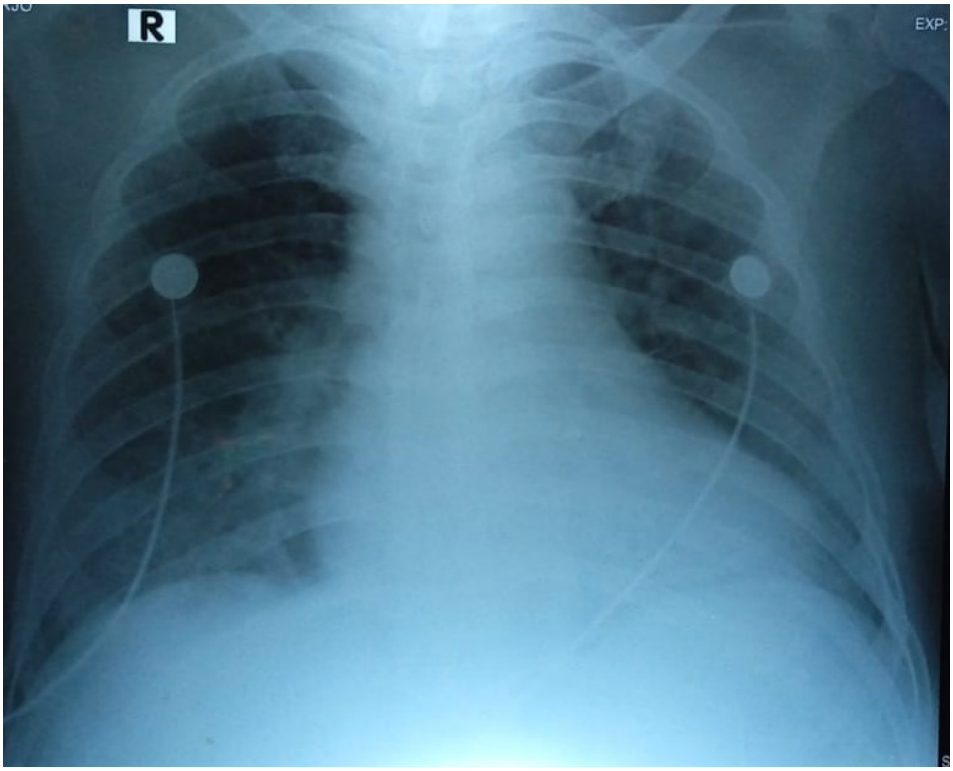

Figure 2. Patient chest x-ray

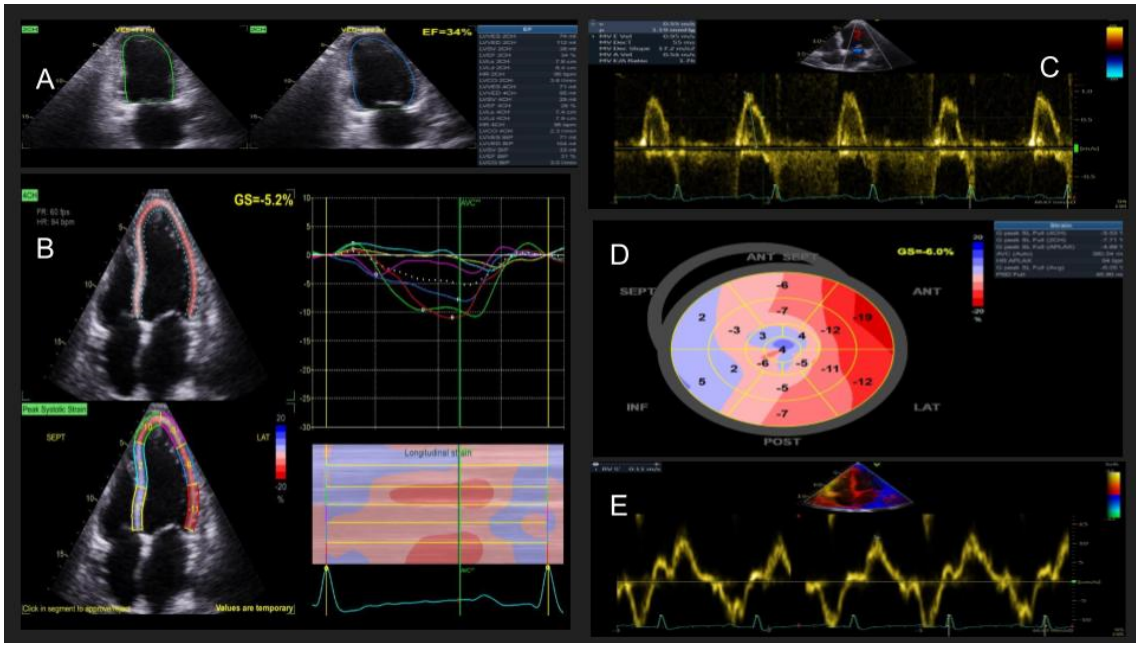

Figure 3. Patient baseline echocardiography examination: A. LVEF assessment by Simpson 4 chamber method; B. 4 chamber LV longitudinal strain tracking; C. Mitral inflow doppler; D. Average LV GLS bull eye; E: RV TDI (LVEF: Left ventricle ejection fraction; LV: Left ventricle; GLS : Global longitudinal strain; RV : Right ventricle; TDI: Tissue doppler imaging)

In hemodynamically unstable and electrical complications of STEMI, it is mandatory to revascularize the patient with a percutaneous coronary intervention (PCI) procedure. The patient was admitted to a non-PCI center with a non-transportable condition. Unfortunately, the patient refused to be referred for further invasive procedures.

On the first day, the ECG showed a TAVB rhythm with a narrow QRS complex and a

QRS rate of 47 beats per minute. BP was 80/48 mmHg and SpO_2 was 98%. Diuresis was 2.1 cc/kgBW/hour. We administered dopamine 7 microgram/kg/min continuous infusion with dobutamine 5 micron/kg/min, ASA 100 mg o.d, clopidogrel 75 mg o.d, atorvastatin 20 mg o.d, anticoagulant fondaparinux 2.5 mg SC o.d, loop diuretic furosemide 40 mg IV b.i.d, and i.v neo-minophagen C 40 mg b.i.d.

On the second day of ICVCU admission, the patient's systemic perfusion improved with a reduction in serum lactate to 2.4 mmol/L (**Table 2**) and diuresis to 1.42 cc/kgBW/hour. ECG still showed a TAVB rhythm with a QRS rate of 58 beats per minute**,** BP 126/76 mmHg, and SpO_2 98%. Dopamine was down-titrated to 3 microgram/kg/min. We initiated low-dose anti-heart failure therapy with captopril 6.25 mg t.i.d per oral. and spironolactone 25 mg o.d per oral.

After the third day of ICVCU admission, hemodynamic and systemic perfusion were improved (**Table 2**). We continued the antiplatelet, anticoagulant, and anti-heart failure therapy. Dopamine and dobutamine therapy were withdrawn. Evaluation of serum creatinine and urea levels showed improvement to 2.1 mg/dL and 146 mg/dL, respectively. The patient was transferred to the medical ward. Concerning clinical and hemodynamic stabilization after chronotropic support, the patient did not undergo performed temporary transvenous pacemaker procedure.

At the medical ward, the ECG rhythm was converted to sinus rhythm. (**Figure 4**) Serum transaminases SGOT and SGPT evaluation showed significant recovery to 146 / 420 U/L, respectively. Patient was discharged at 6 days of admission with medication ASA 100 mg o.d, clopidogrel 75 mg o.d, atorvastatin 20 mg o.d, ramipril 5 mg o.d, spironolactone 25 o.d, oral loop diuretic furosemide 40 mg o.d. Evaluation of serum creatinine and ureum level before patient discharge showed further improvement to 1.1 mg/dL and 95 mg/dL, respectively.

Furthermore, renal and liver function test evaluation after a week of patient discharge showed normalization of these parameters. Serum creatinine 0.9 mg/dL and SGOT/SGPT 81/ 112 U/L.

Table 2. Follow-up of blood gas analysis examinations and the correlation with liver function test during hospital admission.

Blood gas analysis parameter	December 6th, 2023	December 7th, 2023	December 8th, 2023	December 12th, 2023
pH	7.29	7.47	7.41	7.53
PO2 (mmHg)	63	79	77	53
PCO2 (mmHg)	20	23	32	34
HCO3 (mmol/L)	9.9	16.7	20.4	28.6
BE (mmol/L)	-15.1	-5.9	-3.7	5.6
Lactate (mmol/L)	2.8	2.4	2.1	1.2
SGOT/SGPT (U/L)	1143/1085			146/120

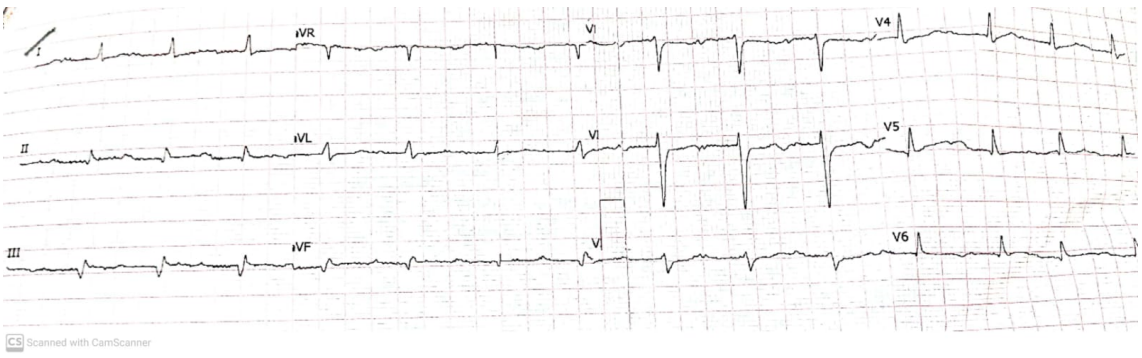

Figure 4. Electrocardiogram After 5-Days Admission Showed Sinus Rhythm.

DISCUSSION

Shock liver is marked by elevation of hepatic transaminases, either transient or persistent, because of hepatocellular injury and ischemia, which is also defined as '*ischemic hepatitis*' or HH. This clinical condition is related to a hepatic hypoperfusion state.[4,6] The response to hepatocytes hypoperfusion due to reduced hepatic arterial flow and/or passive venous congestion. In ICU patients, shock liver has an incidence rate of 50%. Acute heart failure, cardiogenic shock, and sepsis/severe shock were the most common etiologies.[2,3] Severe acute rises of transaminase levels (>1000 iu/l) are documented in HH.[4,6] Henrion et al proposed serum aminotransferase levels at 20 times the upper limit of normal,[7] but other authors have used a lower cutoff, ranging from 2.5 to 10 times the upper limit of normal.[3] Our recent case showed > 20 times the upper limit of normal serum transaminase, which is consistent with the diagnostic criteria proposed.

Several different aetiologies can disrupt liver physiology. Factors such as hypoperfusion, hypoxemia, acute hepatitis, and others can cause hepatic injury and shock liver. Sepsis, massive blood transfusion, nutritional support, and drug toxicity may also contribute.[3,4,6] This condition is associated with high in-hospital mortality (> 50 %).[2,3,4]

Current diagnosis of HH comprises three clinical criteria: an underlying clinical diagnosis that results in reduced oxygen delivery or uptake by hepatocytes, a significant and often transient elevation in liver transaminase level, and exclusion of liver injury causes like drug or virus-induced hepatitis. It could be assumed to diagnose HH without evidence of liver biopsy when all three criteria are fulfilled.[3] As reflected in our case setting, cardiogenic shock complicating acute MI results in hepatic hypoperfusion with significant serum transaminase elevation without any identified hepatitis marker.

Two prior studies stated that ARF reflects the hemodynamic disturbance that causes HH and could aid in diagnosis, because ARF is an outcome of hemodynamic insult, which depicts this set of patients, and its presence is uncommon in viral or drug-induced hepatitis.[7,8] Our case showed overt ARF, which normalized after hemodynamic stabilization.

However, there is no definite treatment strategy for shock liver. The management strategy is directed at the treatment of underlying causes.[3] This can be achieved by stabilization of systemic and hepatic perfusion, control of infection, mechanical ventilation control, and controlled administration of vasoactive drugs such as dobutamine for splanchnic blood flow preservation into the liver and/or dopamine.[3,4,6] Our recent case showed a tremendous transient elevation in hepatic transaminases during cardiogenic shock, which was proved by increased serum lactate level at initial presentation, whereas improvement in liver transaminase levels correlated with reduction of serum lactate level during admission follow-up (**Table 2**).

The prognosis of HH is generally poor, since this condition is a result of life-threatening medical conditions.[2,3] The importance of prompt diagnosis and early management of the underlying disease is clear. The in-hospital mortality was found to be significantly higher in patients with higher peak levels of aspartate aminotransferase (mean: 5.129 U/L), lactate dehydrogenase (mean: 5.047 U/L), INR (mean: 2.9), and serum lactate (mean: 10.4mmol/L). Prolonged durations of HH (defined as >24 hours of rising aspartate aminotransferase levels) were also found to be associated with higher mortality.[3]

Cardiogenic shock is one of the most common HH underlying conditions.[2,3] Especially in post-MI cardiogenic shock setting, which results in LV systolic dysfunction as reflected in reduced ejection fraction (EF) in our case. Post-MI LV systolic dysfunction could initiate circulatory failure in extensive myocardial damage.[9]

Non-reperfused STEMI results in a high risk of mechanical and electrical complications. Gopar-Nieto et al studied this patient population has a higher in-hospital mortality compared to reperfused STEMI (12.7 % versus 7.2 %). The in-hospital mortality main predictor among non-reperfused STEMI were renal insufficiency (HR 3.41), systolic blood pressure (SBP) < 100 mmHg (HR 2.26), and LVEF < 40% (HR 1.97).[10] Our patient showed all combinations of those

risk factors (Cr 4.7 mg/dL, SBP 85 mmHg, and LVEF 31 %), which put the patient at high risk of mortality.

Total AV block is the most common electrical complication in inferior STEMI, resulting in symptomatic bradycardia, which aggravates the pump failure.[11,12] Cardiogenic shock was about seven times more frequent in TAVB patients (33.0 vs. 4.5%).[13] The combination of reduced heart rate and LV systolic dysfunction became the pathophysiological basis of hepatic hypoperfusion in this case report.

Management goals in this clinical situation are focused on improving systemic perfusion by positive inotropic and chronotropic drugs, as well as post-MI anti-remodelling therapy to treat HF and LV systolic dysfunction.[9] Anti-ischemic therapy, such as antithrombotic, is also important to reduce in-hospital mortality. Lastly, to ensure reversibility of TAVB in this circumstance, since this electrical complication has good reversibility compared to TAVB in anterior STEMI.[11-13] However, there was a case report of anterior STEMI which showed rhythm reversibility after a few days of a temporary pacemaker.[14]

The rapid primary hepatocyte injury is marked by high levels of liver transaminases and recovers within a few days after hemodynamic instability has been treated.[4] As seen in our case, the patient's liver transaminase levels returned to near normal levels after systemic perfusion and heart rate had been corrected.

CONCLUSION

We described a case of HH secondary to cardiogenic shock in non-reperfused inferior STEMI. The hemodynamic insult that caused HH is the mainstay therapeutic target.[3] In this case setting is LV failure and TAVB secondary to inferior STEMI. Recovery of liver transaminases was demonstrated after the underlying insult had been eliminated.

CONFLICT OF INTEREST

The authors declare there is no conflict of interest in this study.

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