



Delayed Diagnosis of Myocarditis After Blunt Abdominal Trauma: A Case Report

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ABSTRACT

Introduction: Myocarditis is an inflammation of the myocardium that can lead to impaired heart function and sudden death. Most cases are caused by viral infections and are very rarely reported as a post-traumatic complication. **Case:** A 54-year-old male developed abdominal pain and vomiting after blunt abdominal trauma. The patient was initially diagnosed with colonic rupture and underwent exploratory laparotomy. Despite initial improvement, the patient experienced hemodynamic deterioration. Electrocardiography, cardiac biomarkers, and echocardiography evaluation revealed myocarditis with decreased ejection fraction. Despite supportive and pharmacological therapy, the patient fell into cardiogenic shock and passed away. **Discussion:** This case demonstrates that the diagnosis of myocarditis can be significantly delayed in patients with atypical presentations and no obvious cardiac symptoms. Abdominal trauma accompanied by systemic infection, such as colonic rupture, may potentially cause secondary myocardial inflammation. Clinicians need to consider the possibility of myocarditis in patients with postoperative hemodynamic instability, even without a history of cardiac disease. **Conclusion:** This report highlights the urgency of improving diagnostic awareness of myocarditis and access to support facilities.

Keyword: Abdominal trauma, cardiogenic shock, case report, impaired heart function, myocarditis.

ABSTRAK

Pendahuluan: Miokarditis merupakan inflamasi miokardium yang dapat menyebabkan gangguan fungsi jantung hingga kematian mendadak. Sebagian besar kasus miokarditis disebabkan oleh infeksi virus, dan sangat jarang dilaporkan sebagai komplikasi pasca-trauma. **Kasus:** Laki-laki usia 54 tahun mengalami nyeri perut dan muntah setelah trauma tumpul abdomen. Pasien didiagnosis awal ruptur kolon dan menjalani laparotomi eksplorasi. Meskipun kondisi awal membaik, pasien mengalami perburukan hemodinamik. Hasil elektrokardiografi, biomarker jantung, serta ekokardiografi menunjukkan gambaran miokarditis dengan fraksi ejeksi menurun. Terapi suportif dan farmakologis telah diberikan, namun pasien jatuh pada kondisi syok kardiogenik dan meninggal dunia. **Pembahasan:** Kasus ini menunjukkan bahwa diagnosis miokarditis dapat sangat tertunda pada pasien dengan presentasi yang atipikal tanpa gejala kardiak yang jelas. Trauma abdomen yang disertai infeksi sistemik, seperti ruptur kolon, berpotensi menimbulkan inflamasi miokardium sekunder. Klinisi seyogyanya mempertimbangkan miokarditis pada situasi instabilitas hemodinamik pasca-operasi, meskipun tidak ada riwayat penyakit jantung. **Simpulan:** Kasus ini menyoroti urgensi peningkatan kesadaran diagnosis miokarditis dan akses terhadap fasilitas penunjang. **Astrid Carolinn Valleriana Kaiba, Putu Aditya Darmawan Kuntadi, Nabil Athoillah. Diagnosis Miokarditis Tertunda Setelah Trauma Tumpul Abdomen: Laporan Kasus.**

Kata Kunci: Trauma abdomen, syok kardiogenik, laporan kasus, gangguan fungsi jantung, miokarditis.

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INTRODUCTION

Myocarditis is an inflammatory disease of the heart muscle with a high mortality rate due to its serious short and long-term effects.¹ Global studies show that the burden of this disease reaches 22 cases per 100,000

patients.² The primary and most common etiologies are idiopathic and infection.³ The incidence of myocarditis due to trauma is very rare.⁴ Cases of myocarditis with uncommon etiologies present a challenge to clinicians, as the symptoms are sometimes uncommon and

present late. We describe a case of delayed diagnosis of myocarditis after laparotomy due to suspected intra-abdominal organ rupture. The association between blunt abdominal trauma and the finding of myocarditis is also discussed.

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CASE

A 54-year-old man presented to the emergency department of a regional hospital with complaints of abdominal pain and vomiting after falling on the sidewalk while avoiding a traffic accident 7 hours before admission. The patient claimed that he fell on his stomach, with his abdomen hitting the sidewalk. On arrival at the hospital, he was fully conscious but complained of a headache with no history of fainting. The patient denied having risk factors for gastric diseases, such as GERD or gastritis, nor cardiovascular diseases, such as smoking, hypertension, heart disease, diabetes mellitus, dyslipidemia, or previous similar complaints.

Physical examination showed Glasgow Coma Scale (GCS) E4V5M6, blood pressure 112/70 mmHg, heart rate 95 beats/min, respiratory rate 20 beats/min, body temperature 37.1°C, and SpO₂ 96%. No abnormalities in respiratory or cardiovascular examinations were observed. Abdominal examination revealed an imprint and tenderness. No

edema on limb examination and no bruising or evidence of injury to the chest and other regions except for the abdomen. Initial 12-lead ECG examination in the emergency room showed sinus rhythm, 88 beats/min, normal axis, and nonspecific abnormal ST and T changes in leads I, II, and V1–V4 (**Figure 1A**).

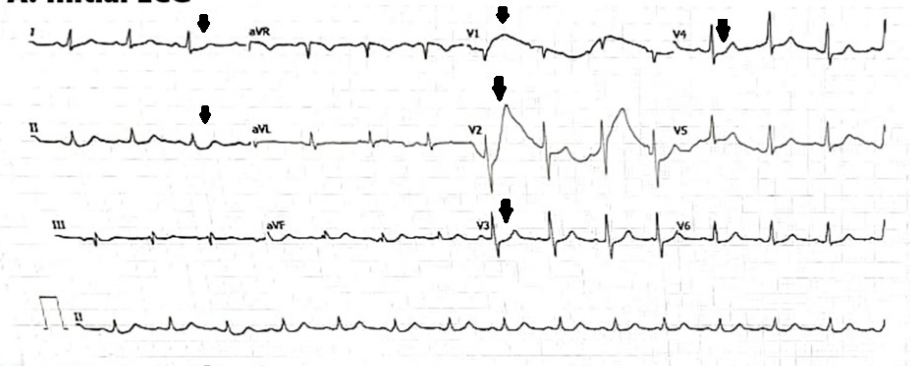
Chest radiographs and laboratory investigations revealed no significant findings. Head CT-scan examination revealed an intrasellar mass without fracture and other significant findings. Abdominal x-ray revealed increased bowel gas distribution, step ladder sign, and coiled spring sign suggestive of paralytic ileus and pneumoperitoneum (**Figure 2**).

The preliminary diagnosis was blunt abdominal trauma with suspected internal organ rupture, and a laparotomy was decided. An intravenous line was placed, and conservative management was given in preparation for surgery. A few hours later, the patient's condition deteriorated, and blood

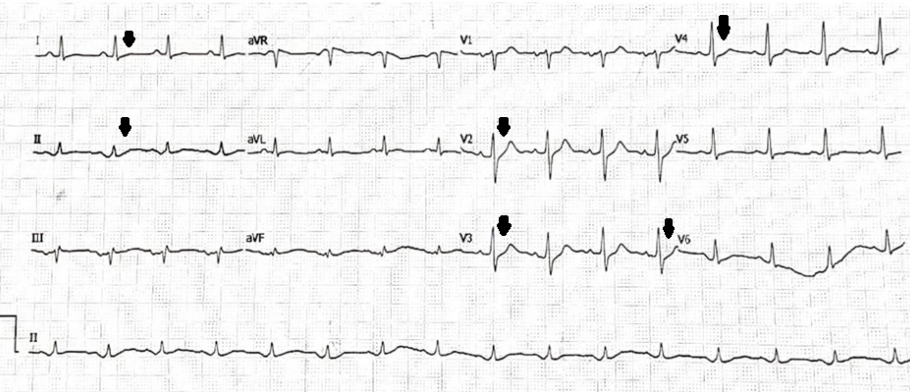
pressure dropped to 66/41 mmHg with a heart rate of 108 beats/min. Hemodynamic stabilization and laparotomy were performed immediately. Exploratory laparotomy revealed a ruptured colon at the meso-transversum and ileal necrosis. Resection of non-viable tissue and stoma insertion were performed. The patient then underwent intensive care in the intensive care unit (ICU) with supportive management, including fluid infusion of 1,000 mL Ringer Acetate and 1,500 mL Ringer Lactate per 24 hours, ketamine infusion 7 mg/hour, in addition to dopamine 5 mcg/kg/min and norepinephrine 200 ng/kg/min infusions for inotropic support. The patient also received mechanical ventilation and was diagnosed with post-laparotomy *et causa* colon rupture, septic shock, with differential diagnosis of cardiogenic shock.

The patient's condition improved with supportive therapy; the next day, his blood pressure reached 106/68 mmHg and pulse rate of 100 beats/min. However, serial complete blood counts showed an increase in leukocyte count from 3,000 to 10,860 cells/uL, with a leftward shift in the differential count. The cardiologist administered spironolactone 25 mg and digoxin 0.25 mg, and suggested troponin screening, repeat

A. Initial ECG

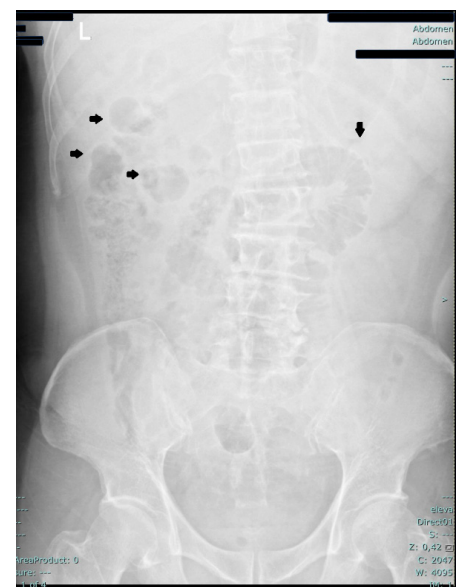


B. ECG Re-evaluation



*Photo documentation by the Emergency and Cardiovascular Department of Dr. Haryoto General Hospital.

Figure 1. (A) Initial ECG examination in the emergency room. (B). Repeat ECG examination shows nonspecific abnormal ST and T changes, especially in leads I, II, and V1–V4 (black arrow).



*Photo documentation by the Radiology Department of Dr. Haryoto General Hospital

Figure 2. Abdominal x-ray shows increased bowel gas distribution, step ladder sign, and coiled spring sign, indicating paralytic ileus (black arrow).

CASE REPORT



ECG, and echocardiography to determine the cause of shock. The troponin test was positive, with the ECG still showing nonspecific ST and T abnormalities (**Figure 1B**). The most important finding was echocardiography, which found Stroke Volume (SV) 40 mL with mild left ventricle dilatation, ejection fraction (EF) 35%, tricuspid annular plane systolic excursion (TAPSE) 1.6 cm, global hypokinetic, eccentric left ventricular hypertrophy suggestive of myocarditis (**Figure 3**). The final diagnosis was post-laparotomy *et causa* colonic rupture with cardiogenic shock, heart failure with reduced ejection fraction (HFrEF), and myocarditis. Additional ramipril 2.5 mg and tapered off norepinephrine to 25 ng/kg/min were administered. After 12 hours, the patient's condition continued to deteriorate; he passed away an hour later.

DISCUSSION

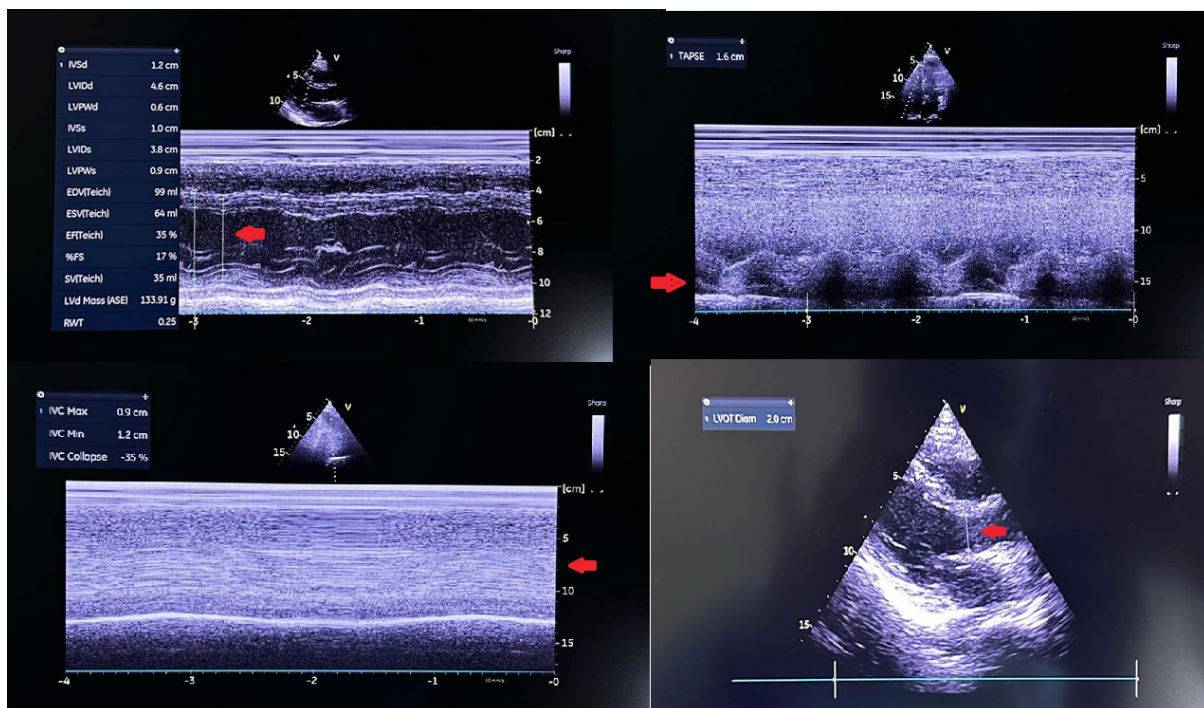
Myocarditis is an inflammation of the myocardium. The most common etiology is direct infection; causes range from excessive exposure to heat or chemicals, radiation, hypersensitivity to drugs, metabolic problems, to Kawasaki disease.⁵ The prevalence of myocarditis reaches 10.2 to 105.6 per 100,000 population worldwide,

with an estimated annual incidence of 1.8 million cases.⁶ It may be underestimated because various reports indicate potential underdiagnosis.⁶

Clinical symptoms of acute myocarditis vary widely, ranging from asymptomatic to mild fever, to cardiogenic shock and sudden cardiac death. Patients may experience fever, chest pain, malaise, palpitations, dyspnea, and syncope. Chest pain may mimic pericarditis, especially in cases of myocarditis with pericarditis, also known as myopericarditis.³ In severe conditions, patients may develop fulminant acute myocarditis with symptoms of decompensated heart failure, hemodynamic disturbances, and ventricular arrhythmias; some patients have decreased left ventricular ejection fraction.³ Physical examination in severe conditions may find pulmonary rales, S3 gallop, and peripheral edema. In milder cases, the physical examination may detect simple viral syndrome, such as fever, headache, body aches, and fatigue.⁷ Pericardial friction and pansystolic murmur may be found in myocarditis involving pericardial and mitral regurgitation.³

Many supporting examinations are

recommended to support the diagnosis of myocarditis.² Laboratory investigations may include findings of leukocytosis or elevated eosinophilic blood count, abnormal liver and kidney function, with increased C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) in more than 80% patients, specifically in autoimmune myocarditis.² Elevated troponin and serum brain natriuretic peptide (BNP) are the most important laboratory biomarkers. Imaging and biopsy are urgent in the setting of a marked increase in these two cardiac biomarkers.² Electrocardiography is the most important early examination, with common findings of ST elevation or abnormal T waves.⁸ The other important routine examination is transthoracic echocardiography (TTE), essential to detect decreased left ventricular ejection fraction, diastolic dysfunction, segmental wall motion abnormalities, increased heart wall thickness, abnormal echogenicity of the myocardium, or even pericardial effusion.² Cardiovascular magnetic resonance (CMR) imaging is also recommended in suspected acute myocarditis, especially in a fulminant condition.^{9,10} The examination is a standardized, non-invasive examination and should be performed within 2 to 3 weeks after



*Photo documentation by Cardiovascular Department of Dr. Haryoto General Hospital

Figure 3. The patient's echocardiogram showed mild left ventricular dilatation, reduced ejection fraction (35%), TAPSE of 1.6 cm, global hypokinetics, and eccentric left ventricular hypertrophy, indicating myocarditis (red arrow).



the onset of symptoms.¹¹ The gold standard examination is endomyocardial biopsy.⁸

Management of myocarditis varies widely, from conservative to advanced. Current heart failure guidelines suggest angiotensin-converting enzyme (ACE) inhibitors, beta blockers, mineralocorticoid receptor antagonists (MRA), diuretics, and SGLT-2 inhibitors such as dapagliflozin to reduce rehospitalization due to worsening heart failure, eliminate congestion, improve patient survival, and reduce mortality.¹² It can also be applied to stable myocarditis patients with diastolic dysfunction. Patients may be given anti-arrhythmics when indicated. In fulminant myocarditis, such as cardiogenic shock or LV dysfunction with unstable hemodynamics, vasopressors, inotropes, or mechanical circulatory support should be given. Advanced management of myocarditis may include the administration of steroids, NSAIDs (ibuprofen, aspirin, and indomethacin), colchicine, and other immunosuppressant therapies such as azathioprine and methotrexate.¹³ However, the use of these immunosuppressant agents is still controversial because these drugs have the potential to trigger viral spread and direct cardiomyocyte damage.¹³ The use of NSAIDs is recommended in low-risk myocarditis without left ventricular dysfunction. In contrast, the use of NSAIDs in patients with high-risk myocarditis complicated by heart failure can be harmful.¹³

The key concern in this case is identifying potential etiologies of myocarditis that may significantly contribute to our patient's morbidity and mortality. Viral infections are the most common cause, followed by bacterial and protozoal agents.³ These may induce myocarditis through direct myocardial invasion or immune-mediated injury.¹⁴ Notably, pathogens such as *Coxsackievirus*, HIV, *Legionella*, and *Trypanosoma cruzi* have been implicated.³ Following infection, immune dysregulation may lead to persistent

inflammation, myocardial remodeling, and dysfunction, even after pathogen clearance.¹⁴ Although rare, trauma has also been reported as a potential cause of myocarditis. Most documented cases involve direct chest trauma.¹⁵ Reports of myocarditis following blunt abdominal trauma are exceedingly rare. However, some literature, such as Dunsire, et al., has noted cardiac complications like tamponade in such settings.¹⁶ Although there were no reports directly linking myocarditis to colonic perforation, gastrointestinal infections, especially by pathogens such as *Campylobacter jejuni* and *Salmonella enteritidis*, have been associated with myocarditis.^{17,18} It is plausible that severe colonic infections, particularly if leading to bacteremia, could contribute to myocardial inflammation through systemic spread and immune activation.² In this case, abdominal trauma could potentially cause damage to the intestinal integrity, leading to gastrointestinal tract infection, which triggers sepsis, and then myocarditis by the aforementioned mechanisms.^{19–21}

This patient presented with some interesting challenges. The patient came with a presentation and history not characteristic of myocarditis, such as abdominal pain, nausea, and vomiting after abdominal trauma. He was stable post-operatively and had no symptoms suggestive of myocarditis except for less specific leukocytosis and abnormal ST-T changes. The diagnosis of myocarditis is based on global hypokinetic findings, decreased ejection fraction, and mild left ventricular dilatation and hypertrophy on echocardiography accompanied by leukocytosis and an increase in the marker of cardiac damage. Endomyocardial biopsy and CMR were not performed due to limitations in the patient's condition and supporting facilities. In many developing countries, cost-effectiveness considerations are essential, which is quite limiting. Hence, some essential supporting examinations are unavailable. In

cases of myocarditis, early diagnosis is crucial as it determines the treatment plan. Clinicians should be able to diagnose myocarditis with limited physical examination and supportive findings. The uncommon presentation of this case also led to a delayed diagnosis that may have contributed to the patient's deterioration and led to fulminant myocarditis. This case report highlights the need for awareness that myocarditis can be caused by uncommon conditions and for healthcare providers to maintain 24-hour readiness to maximize the recovery process.

CONCLUSION

Myocarditis diagnosis and management remain challenging due to its uncommon presentation and nonspecific history. Acute myocarditis can present atypically and be easily masked by non-cardiac emergencies, such as blunt abdominal trauma and subsequent colonic rupture. Severe abdominal injuries accompanied by systemic infection may trigger secondary myocardial inflammation, leading to rapid clinical deterioration. Therefore, clinicians should perform basic cardiac evaluations, such as electrocardiography and bedside echocardiography, in postoperative patients with unexplained hemodynamic instability. Early diagnostic recognition is critical to differentiate secondary cardiogenic shock from septic shock, allowing for timely and targeted supportive therapy.

Informed Consent

The patient or a family member has provided written or verbal consent for the publication of the manuscript and all identifiable data.

Conflict of Interest

None

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None

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