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Splenic Rupture Imitate Acute St-Segment Elevation Myocardial Infarction: A Case Report

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1. Abstract

ST-segment elevation myocardial infarction represents a critical and potentially life-threatening cardiac condition that necessitates immediate intervention [1]. Gastrointestinal perforations are non-cardiac conditions that can present with electrocardiogram changes mimicking ST-segment elevation [2]. We present the case of a 43-year-old male who developed acute peritonitis as a result of a traumatic splenic injury. This case was notable for dynamic electrocardiogram changes that initially mimicked anterior ST-segment elevation myocardial infarction. Subsequently, the electrocardiogram abnormalities normalized following splenectomy. The patient was discharged after a four-day hospital stay, completely free of lingering symptoms. The misinterpretation of the electrocardiogram changes led to the unnecessary administration of fibrinolytic therapy and a delay in receiving the appropriate treatment.

2. Case Report

A 43-year-old male with a history of cigarette smoking and drug abuse presented to our hospital for further evaluation with a provisional diagnosis of anterior ST-segment elevation myocardial infarction (STEMI). The patient had been previously admitted to another hospital due to a retrosternal chest discomfort that had started the night before. At the previous hospital, he had received fibrinolytic therapy in response to precordial ST-segment elevation on the electrocardiogram (ECG) and elevated cardiac troponin levels. The fibrinolytic therapy consisted of two doses of 10 Units Reteplase administered 30 minutes apart. Upon presentation to our facility (3 hours after intravenous thrombolysis), he reported a recent quarrel approximately 40 days prior to presentation and abdominal pain since then. The patient's chest pain had resolved, but his abdominal pain had worsened. He appeared pale, with a blood pressure of 100/70 mmHg and a pulse rate of 80 beats/min. Cardiac auscultation revealed no murmurs or friction rubs, and lung sounds were clear. However, there was considerable abdominal distention and tenderness in the epigastric, right upper, and lower quadrants. The initial ECG showed normal sinus rhythm with a normal axis and ST-segment elevation in leads V1-V6. Laboratory tests indicated anemia (hemoglobin: 13 gm/dl), a troponin-I level of 953 ng/l (positive at >19 ng/l), liver function test results nearly two times higher than normal, and a positive C-Reactive Protein. Other metabolic laboratory tests were within normal ranges. Echocardiography revealed normal left ventricle (LV) and right ventricle (RV) size and function (LVEF: 60%) with neither regional wall motion abnormality nor valvular lesions and no pericardial effusion.

Abdominal ultrasonography revealed hyperechoic areas around the right hepatic lobe (segments VII and VIII) with a moderate amount of free fluid in the abdominal cavity, while other organs appeared normal. Due to resource limitations, a contrast-free abdominopelvic CT scan was performed, showing heterogeneous density

in the right liver lobe and a moderate amount of free fluid in the abdominal space, suggestive of blood. To confirm the diagnosis of hemoperitoneum, sonography-guided aspiration of peritoneal fluid was performed, confirming the presence of blood. Following a surgical consultation, the patient was urgently taken to the operating room for laparotomy. Splenic rupture was confirmed as the cause of hemoperitoneum, and a splenectomy was performed. ECG changes returned to normal after surgery, as shown in (Figure 3). The patient was discharged four days post-surgery, free of any symptoms. The patient was refused to perform Coronary Artery Angiography or any Multi-slice Computed Tomography Coronary Angiography for evaluation of coronary anatomy.

3. Discussion

Acute Ischemic Heart Disease (IHD) is a condition characterized by myocardial muscle damage due to significant coronary artery stenosis. Among the various types of IHD, ST-segment elevation myocardial infarction (STEMI) is the most life-threatening and necessitates immediate recognition and treatment. For a long time, the electrocardiogram (ECG) has been the primary diagnostic tool for identifying STEMI due to its cost-effectiveness and widespread availability. However, in cases with prominent symptoms such as abdominal pain, as seen in our patient, a comprehensive physical examination and consideration of alternative differential diagnoses that can mimic STEMI become crucial. Examples of cardiac conditions that can mimic STEMI include myocarditis, pericarditis, acute pulmonary thromboemboli, left bundle branch block, and coronary artery dissection. Non-cardiac etiologies of ST-segment elevation encompass pancreatitis, intracranial hemorrhage, and acute cholecystitis [1,2]. Several reported cases of gastric perforation (3 cases) and abdominal distention (8 cases) mimicking myocardial infarction have been published [3-9]. In comparison with previous cases of gastrointestinal tract perforation, our patient was notably younger. In all previous cases, none had elevated troponin levels nor received reperfusion therapies. Coronary artery angiography was performed in 6 patients, and 5 of them exhibited normal coronary arteries or non-significant lesions (Table 1). Similar to our patient, ECG patterns returned to normal after addressing the underlying condition (Figure 1 and 3). To minimize mortality and morbidity rates, current guidelines recommend initiating reperfusion therapy as early as possible in patients presenting with evidence of STEMI without the need for additional evaluations such as echocardiography [10]. In our patient, the presence of chest discomfort and ST-segment elevation in anterior leads, along with positive cardiac enzyme levels, led our colleagues to diagnose STEMI even in the absence of echocardiographic data. However, after fibrinolytic therapy, echocardiography revealed no evidence of myocardial involvement, and the presence of abdominal free fluid raised suspicion of alternative diagnoses, including hemoperitoneum as the primary cause. As the ECG changes reverted to normal after splenectomy, splenic rupture, hemoperitoneum and it's compressive effect on the heart were identified as the causes of ST-segment elevation in our patient.

The pathophysiologies that can induce ST-segment elevation in patients with abdominal pathologies include a compressive effect on the heart or changes in cardiac positioning due to abdominal distention, Hypervagotonia resulting from the visceral-cardiac reflex, Takotsubo cardiomyopathy, and Prinzmetal angina [2]. Based on echocardiographic data, the patient's heart rate on admission and on the fourth day, Takotsubo cardiomyopathy, Hypervagotonia, and Prinzmetal angina are less likely in our patient. Therefore, the only plausible cause of ECG changes in our patient based on abdominal distention in physical examination is a compressive effect on the heart.

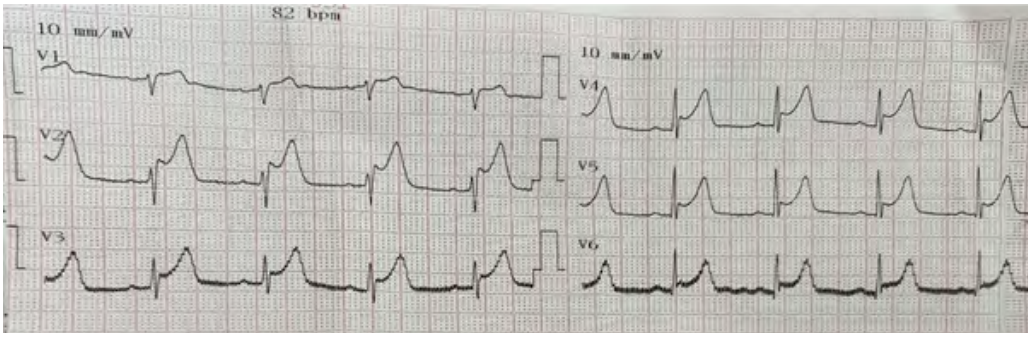

Figure 1: 12-lead ECG on the admission. 12-lead ECG with precordial ST-segment elevation after fibrinolytic therapy on our hospital admission.

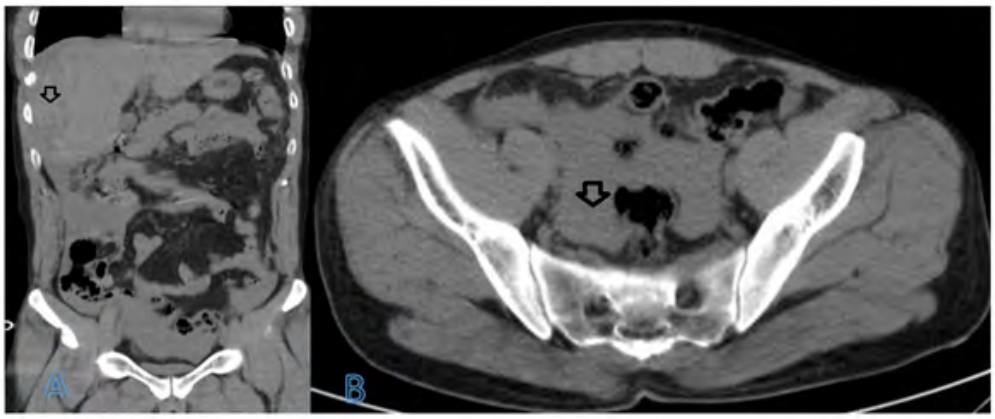

Figure 2: Abdominal CT scan without contrast. CT scan demonstrated heterogeneous density in right liver lob (arrow in picture A) and free fluid in abdominal space suggestive of blood (arrow in picture B).

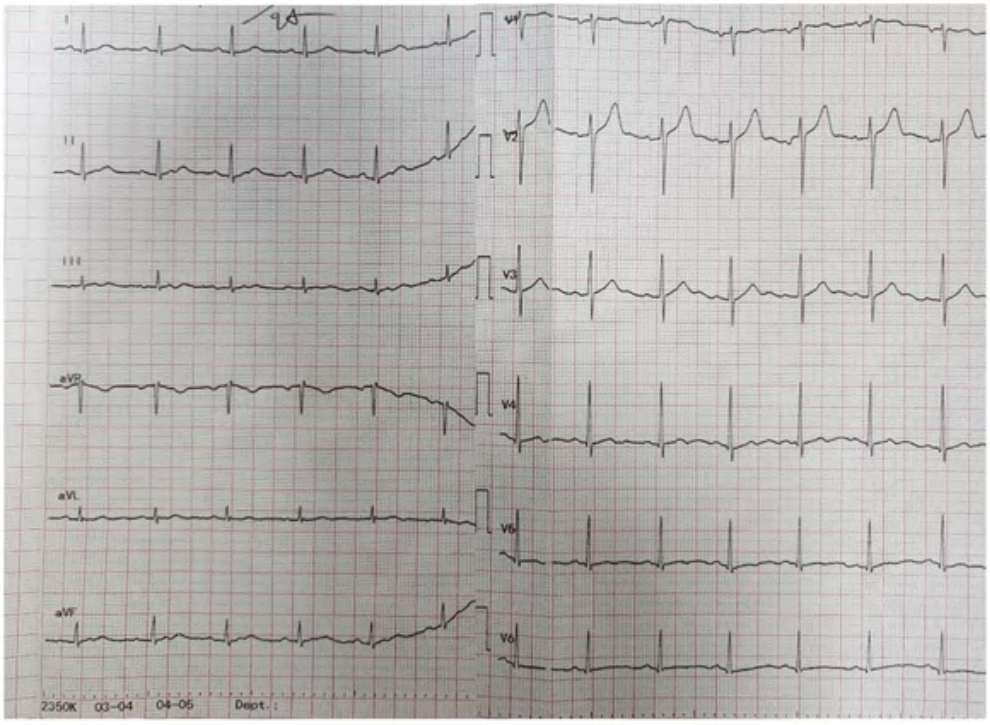

Figure 3: 12-lead ECG after splenectomy. The 12-lead ECG showed that ST-segment elevation returned normal after splenectomy

Table 1: Comparison of cases with gastro-intestinal disorders mimicking STEMI

Case	Chief complaints	ST-segment elevation leads	Troponin(ng/ml)	Diagnosis	Main mechanism of ST-segment elevation
Zhang et al.1	Dyspnea, fatigue, lower limbs edema	Extensive anterolateral	<0.01	Ileus	Compressive effect to the heart
Intant et al. 2	Epigastric discomfort	Anteroseptal	N/A(normal CK-MB)	Gastric perforation	Compressive effect of abdominal distention to the heart
Hoang et al.2	Dyspnea, chest pain	No data	No data	Peptic ulcer perforation	Pneumopericardium
Vutthirkraivit2	Epigastric pain	Anteroseptal	<0.01	Peptic ulcer perforation	Peritonitis
Parikh et al.3	Abdominal pain, vomiting	Inferior	0.04	SBO	Change in heart position
Hibbs et al.4	Abdominal pain, emesis, nausea	Inferolateral	<0.01	Ileus	Transient ion channel change
Hearth et al.5	Abdominal pain	Anterior	Negative	Colonic obstruction	Change in cardiac position
Asada et al.6	Chest discomfort	Inferior	Negative	Gastric distention	Compressive effect to the heart
Patel et al.7	Nausea, vomiting	Inferolateral	N/A	SBO	Compressive effect to the heart
Mixon et al.8	Abdominal pain	Anterior	Negative	SBO	Pericarditis/electrolyte change
Jawa et al.9	No symptoms	Inferolateral	Negative	Ileus , stomach distention	Change in heart position

SBO: small Bowl Obstruction

4. Conclusion

Splenic rupture should be regarded as an uncommon condition capable of inducing ST-segment elevation on an ECG. It is vital for physicians to recognize that acute abdominal conditions can result in ECG changes that resemble those seen in acute myocardial infarction. Delay in identifying and treating such cases can have serious, irreversible consequences.

5. Disclosures

The authors have nothing to disclose.

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