Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

Ventricular septal rupture with right hypochondrial pain mimicking acute cholecystitis



JOURNAL of CARDIOLOGY CASES

Volume 22 Ju

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A R T I C L E I N F O

Article history: Received 20 September 2021 Revised 17 December 2021 Accepted 7 January 2022

Keywords: Intensive care medicine ST elevation myocardial infarction Ventricular septal rupture Diagnostic error

ABSTRACT

A 67-year-old man was admitted for anterior acute ST elevation myocardial infarction (STEMI) management. He developed a severe acute right subcostal pain with normal cardiac tests. On day 5 of hospitalization, cholecystectomy was performed for suspected acute cholecystitis, but the pain intensified with hemodynamical instability. Transthoracic echocardiography revealed ventricular septal rupture (VSR). After emergency operation was performed, the pain diminished with improved hemodynamics. Right subcostal pain associated with heart disease can be referred from STEMI or liver congestion with right heart failure. VSR and right heart failure may be considered as a cause of right subcostal pain of uncertain etiology.

<Learning objective: Patients presenting with signs suggestive of acute cholecystitis may be misdiagnosed, and we highlight the importance of considering ventricular septal rupture as a differential diagnosis.>

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Introduction

Cases of acute cholecystitis mimicking ST elevation myocardial infarction (STEMI) with typical chest symptoms have been reported. However, no case of ventricular septal rupture (VSR) with typical abdominal pain for acute cholecystitis has been reported. We present a case of VSR with right subcostal pain mimicking acute cholecystitis after anterior acute STEMI treatment.

Case report

A 67-year-old man with untreated hypertension and diabetes was admitted to our hospital with acutely increasing chest and back pain. The patient's electrocardiogram (ECG) assessment revealed ST segment elevation in V1-6 (Fig. 1A), and transthoracic echocardiography (TTE) demonstrated wall motion abnormality in the anterior region (Fig. 2A); thus, he was diagnosed with an anterior acute STEMI. The maximum creatinine kinase (CK) level was 5021 U/L, and the maximum CK-MB level was 535 U/L. Coronary

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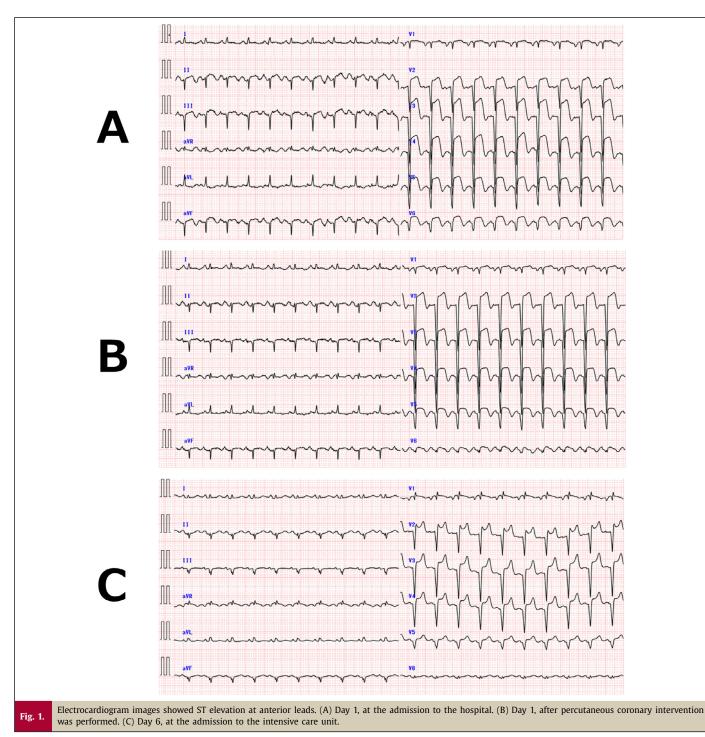
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angiography revealed total occlusion at the middle section of the left anterior descending artery, after which percutaneous coronary intervention (PCI) was performed (Fig. 2B). It probably took at least 12 h from onset to PCI, because chest pain occurred on a previous day and CK level was already high, 5021 U/L at the admission. However, 5 h after PCI, he consistently presented with acute strong right subcostal and epigastric pain, abdominal computed tomography (CT) was immediately performed because acute cholecystitis was suspected. Gallstones were detected on CT but the liver enzyme level did not increase in the laboratory tests; therefore, he was treated conservatively for biliary colic. He did not have symptoms of left heart failure, such as dyspnea during exertion or orthopnea, but had symptoms of right heart failure including fatigue and loss of appetite. After PCI, the myocardial enzyme level decreased, no heart murmur was heard, and there was no evidence of new ECG abnormalities (Fig. 1B). Daily TTE did not demonstrate new signs of decreased systolic function, myocardial ischemia, or mechanical complications. On the 5th day of hospitalization, he developed stronger right subcostal and epigastric pain and a positive Murphy sign without heart murmur. Laboratory tests showed an increase in C-reactive protein to 10.82 mg/dL, and new CT revealed an edematous gallbladder. Meanwhile, the white blood cell count increased to 12600/µL and

https://doi.org/10.1016/j.jccase.2022.01.002

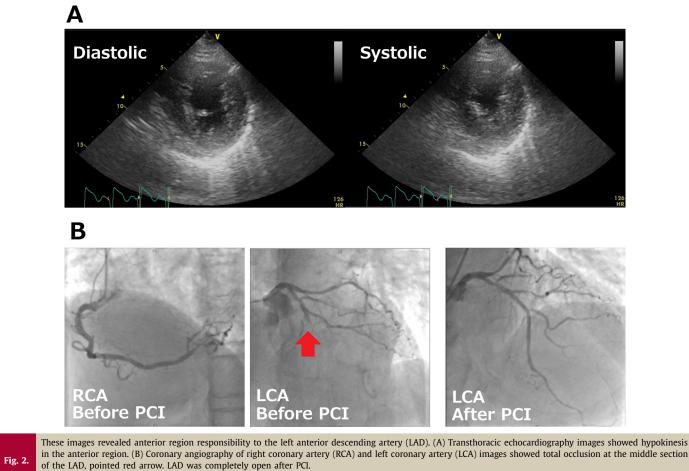
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some of serum liver and biliary enzymes were slightly high; the aspartate aminotransferase was 57 U/L, the alanine aminotransferase was 45 U/L, and the gamma-glutamyl transpeptidase was 77 U/L. The gallstones vanished on the CT. The abdominal symptoms were strongly suspicious of acute cholecystitis; therefore, laparoscopic cholecystectomy was performed for diagnostic treatment. However, in the pathological examination, chronic cholecystitis was identified by the slight infiltration of inflammatory cells, hypertrophy of the muscularis propria, and Rokitansky-Aschoff sinuses. Postoperatively, he presented with exacerbated pain and was admitted to the intensive care unit (ICU) due to restless-

ness, tachypnea, and shock without any new abnormality on TTE and ECG (Fig. 1C). He was managed for septic shock with broadspectrum antibiotics, tracheal intubation, invasive ventilation, and continuous renal replacement therapy. Norepinephrine (maximum 0.35 mcg/kg/min), dobutamine (maximum 7.6 mcg/kg/min), and epinephrine (maximum 0.2 mcg/kg/min) were administered continuously. Despite this therapy, the patient's hemodynamic state deteriorated. After admission to the ICU, 4 times TTE indicated no abnormalities. However, 10 h later, 5th TTE revealed a left-to-right shunt through the ventricular septum for the first time; thus, VSR was strongly suspected (Fig. 3A). He underwent immediate intra-



PCI, percutaneous coronary intervention.

aortic balloon pumping and venous-artery extracorporeal membrane oxygenation, with subsequent emergency patch closure of the VSR. Perforation at the apical septal region was confirmed by intraoperative transesophageal echocardiography (TEE) and by the surgeons' inspection (Fig. 3B). Postoperatively, his right subcostal pain diminished as the hemodynamics improved, and he left the ICU on the 21st day of hospitalization and was transferred on the 74th day. Informed consent to publish the information has been granted from the patient.

Discussion

We present a case of VSR with right subcostal pain for several days after STEMI treatment. Cases of acute cholecystitis mimicking STEMI have been reported [1–3]. Right subcostal pain associated with heart disease is known to be referred pain from acute myocardial infarction or liver congestion with right heart failure [4]. In this case, the patient tended to have symptoms of right heart failure, including fatigue and loss of appetite, and the pathological examination revealed chronic cholecystitis. Therefore, we consider that he already had minor VSR and right heart failure before the right subcostal pain developed, but he was misdiagnosed with acute cholecystitis because the pain was strongly suggestive thereof, and because we could not identify the VSR on TTE until the rupture progressed after the cholecystectomy. Thus, in patients with right subcostal pain after STEMI onset, we must consider right heart failure due to VSR. VSR has a significant mortality rate; approximately 46% of patients die within the first week and 62% to 82% within two months without surgical treatment [5]. Occasionally, TEE may be better for delineating the VSR than is TTE [6]. In this case, the absence of abnormalities on TTE led to the misdiagnosis of septic shock; therefore, the diagnosis of VSR was delayed. When VSR is strongly suspected, even if it cannot be detected on TTE, invasive examinations such as TEE or a pulmonary artery balloon catheter to detect a leftto-right shunt should be considered [7]. We believe that an additional test was indicated immediately when hemodynamic instability developed.

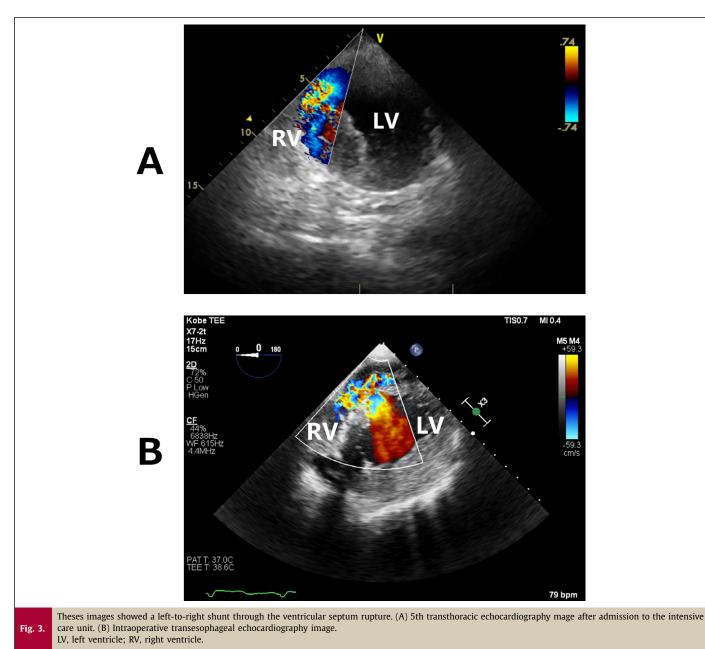
When patients have right subcostal pain after STEMI onset, they may have right heart failure from VSR, not acute cholecystitis. Thus, if the cause of the pain is unclear, we should consider VSR as a cause and diagnose it early.

Declaration of Competing Interest

Nothing

CRediT authorship contribution statement

Daichi Urabe: Writing – original draft. **Daisuke Kawakami:** Conceptualization, Writing – review & editing. **Haruna Nishigaki:** Visualization. **Yusuke Miyoshi:** Visualization. **Jiro Ito:** Writing – review & editing. **Hiroshi Ueta:** Writing – review & editing. **Takahiro**



Shimozono: Writing – review & editing. Hiroyuki Mima: Supervision.

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