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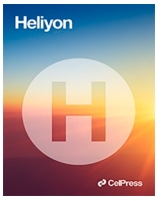

Case report

Post-infarction ventricular septal rupture complicated with cardiogenic shock and multiple organ hemorrhage: An autopsy case report

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A R T I C L E I N F O

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A B S T R A C T

Ventricular septal rupture (VSR) is a catastrophic mechanical complication of acute myocardial infarction (AMI) that can result in acute heart failure. Delaying operative intervention frequently leads to cardiogenic shock and multi-organ failure. Here we report a case of massive anterior MI complicated with VSR that was discovered through cardiac Doppler ultrasound and suspected multiple organ hemorrhage. The patient showed signs of rapid cardiogenic shock and eventually died. The morphological changes of VSR and MI were identified during necropsy, and microscopic examinations of the heart, brain, and kidney revealed multiple organ hemorrhage. This autopsy case suggested that the complication of VSR caused by AMI results in a reduction of oxygen and nutrient content of the circulating blood throughout the body and, eventually, functional failure of multiple organs. We provide clinical and pathological evidence elucidating changes in multiple organs under the severe condition of post-infarction VSR and demonstrate the consequences of a lack of immediate surgery and sufficient medical intervention for a patient suffering from AMI with VSR.

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1. Introduction

Post-myocardial infarction (MI) ventricular septal rupture (VSR) is a rare but potentially catastrophic mechanical complication [1], which specifically occurs in 0.21 % of patients with ST-segment elevation myocardial infarction (STEMI) and 0.04 % of patients with non-ST-segment elevation myocardial infarction (NSTEMI) [2,3]. Post-MI VSR is a medical emergency with a mortality rate of 70 %–80 % during the 2 weeks following diagnosis, and guidelines recommend urgent surgical closure irrespective of the patient's hemodynamic status [1]. VSR leads to increased pulmonary blood flow and left ventricular volume overload due to the left-to-right intracardiac shunt, which, if severe, can lead to acute heart failure [1]. Symptoms of VSR include chest pain, shortness of breath, and low cardiac output and shock [4]. Here we report a patient with MI complicated with VSR who demonstrated cardiogenic shock symptoms at admission, declined surgical repair, and eventually died suddenly. Autopsy examination revealed a pathological profile of post-MI with VSR, consistent with clinical findings. This case report enhances our understanding of post-MI with VSR and provides evidence supporting clinical therapeutic considerations.

2. Case presentation: clinical data

A 63-year-old Chinese male was admitted to the emergency department with a main complaint of "dyspnea after exercise for 10 days." After a couple of days, the condition worsened to the point that one or two steps of walking could result in the patient sweating and feeling suffocated. The patient's cardiac function was scored to grade IV (Killip grade) and the full day urine volume was 400 ml, with elevated serum troponin I (320.8 pg/ml). In terms of history, the patient had been diagnosed with coronary heart disease without treatment of revascularization 2 years prior and had suffered from "cerebral infarction" but had fully recovered 1 year previously. The patient had also drank alcohol for decades and had smoked 20–40 cigarettes per day for 40 years.

Physical examinations at admission: the patient showed acute facial features, wheezing, and restlessness. The skin of the patient's limbs was cold, with distension of jugular veins on both sides, and thickening of the breathing sound in both lungs without moist rale. The heartbeat was regular, with a harsh, loud holosystolic murmur along the left and right sternal borders radiating toward the base and apex. The muscle strength and tone of the extremities were generally normal, with no edema in the lower limbs. On the first day of administration, electrocardiogram showed "complete right bundle branch block, ST segment elevation of lead V2–V5" (Fig. 1A). Bedside echocardiogram showed nodular dilation at the apex of the heart, reverse movement near the apex of the ventricular septum, shunt from left to right, and local interruption of continuity, which was approximately 1.4 cm long (Fig. 1B), indicating perforation of the ventricular septum.

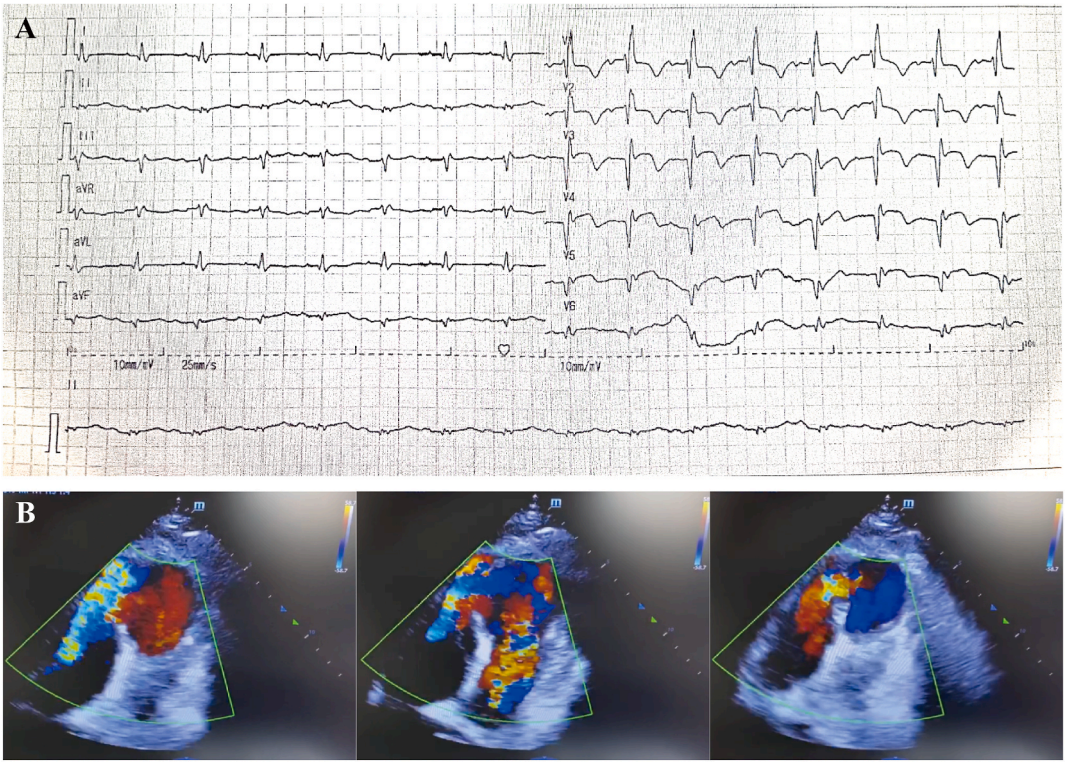

Fig. 1. (A) The 12-lead electrocardiogram performed on admission showed ST segment elevation in the V2–V5 leads. (B) Echocardiogram showing a left-to-right shunt near the apex of the ventricular septum, indicating perforation of the ventricular septum.

During hospitalization, the patient continued without urination and showed significantly increased levels of creatinine and urea nitrogen and electrolyte disturbances in the serum. The patient's condition continued to deteriorate through treatment of appropriate correction of electrolytes and diuresis but the dosage unclear, the patient leaved the hospital without any other drug treatment or surgery. Finally, the patient was found to have passed away at home, and the body tissue was donated for research according to the will before his death.

3. Case presentation: autopsy findings

The postmortem delay was 9.5 h. The entire heart and thoracic aorta was harvested from the body, at a total weight of 575 g. Macroscopic examination revealed a scar in the anterior wall of the heart (Fig. 2A and B, white box), indicating severe MI. The left ventricular outflow tract and tricuspid artery appeared normal.

Microscopic examination showed that the myocardial specimen exhibited typical histological features of MI without reperfusion [5]. Hematoxylin and eosin staining of the infarcted left ventricle showed contraction band necrosis [6] (Fig. 2C), heavy inflammatory cell infiltration (Fig. 2D), and intramyocardial hemorrhage with diffuse and confluent packed red blood cells spread between myocytes (Fig. 2E). Sirius red (Fig. 2F) indicates severe fibrotic adverse remodeling of the left ventricular infarcted area. Histological staining of the adjacent non-infarcted left ventricle revealed infiltration of inflammatory cells and necrosis of cardiomyocytes (Fig. S1A),

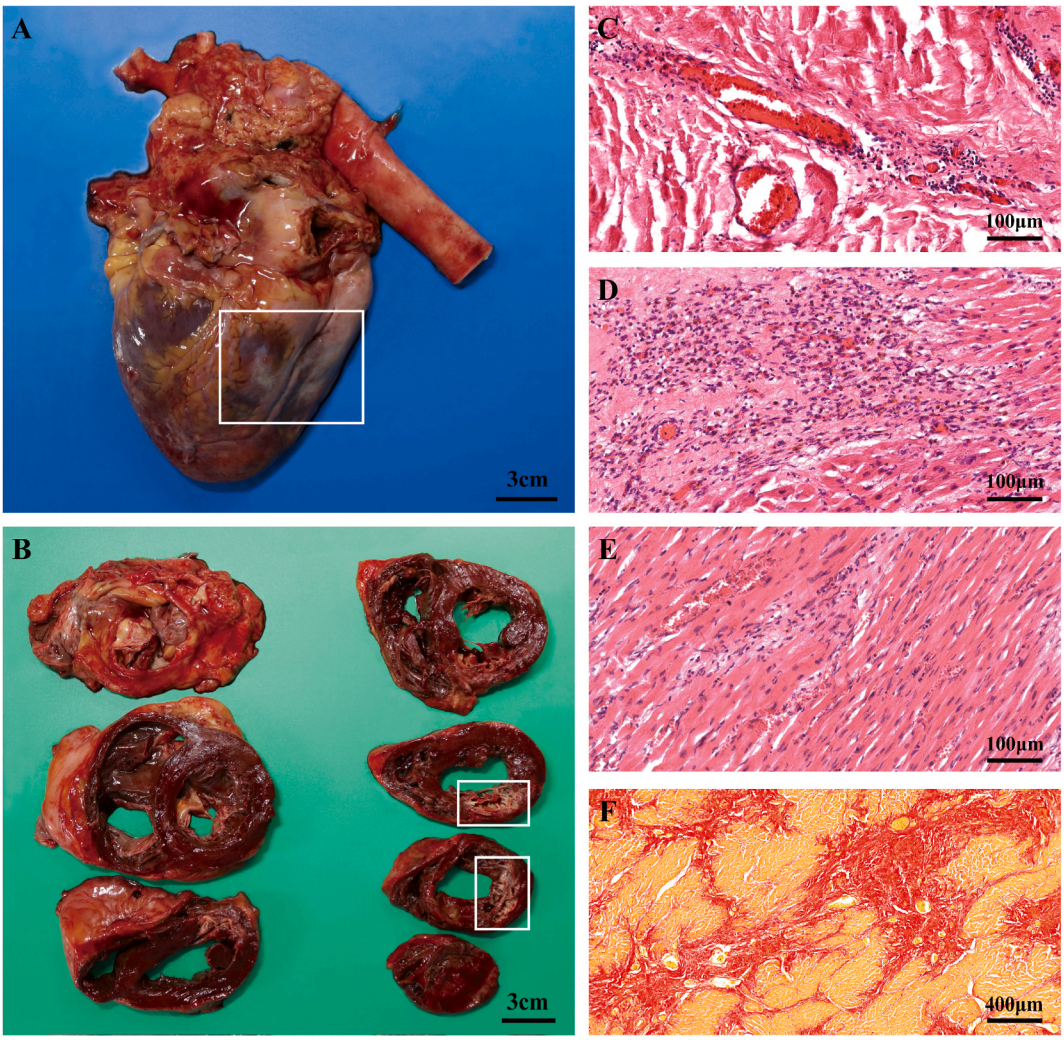

Fig. 2. Histopathological findings of the infarcted heart tissue. (A) Gross view of the heart and aorta. (B) Autopsy myocardial specimens, sequentially from the base to the apex, 0.5–1 cm per slice. Hematoxylin and eosin staining of the infarcted area showed (C) contraction band necrosis, (D) heavy inflammatory cell infiltration, and (E) intramyocardial hemorrhage with diffuse and confluent packed red blood cells spread between myocytes. Sirius red indicates (F) severe fibrotic adverse remodeling of the left ventricular infarcted area. Scale bar for (A and B): 3 cm; scale bar for (C–E): 100 μm; scale bar for (F): 400 μm. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

interstitial edema (Fig. S1B), myofiber waviness (Fig. S1C), and mild microvascular fibrosis (Fig. S1D–F).

Staining of the ventricular septum reflected microvascular damage (Fig. 3B), severe inflammation (Fig. 3B), fat infiltration (Fig. 3C), interstitial and microvascular fibrosis (Fig. 3D), which correlated with bedside echocardiography showing "ventricular septal perforation". The right ventricular tissue showed microvascular obstruction, interstitial fibrosis, and inflammatory infiltration (Fig. S2).

The brain and kidney were also collected for pathological examinations. The whole brain weighed 1246 g, with no obvious external atrophy. Moreover, no evident hemorrhages or infarction lesions were found in the cerebrum, cerebellum, and brainstem after general observation (Fig. 4A and B). Microscopic examination showed mild neuronal loss and moderate perivascular dilatation in the gray matter of the frontal (Fig. 4D) and motor cortices (Fig. 4E). Moderate perivascular dilatation and mild microbleeds were observed in the pons (Fig. 4F), while congested blood vessels with microbleeds were found in the medulla (Fig. 4G). The left kidney weighed 161 g, and its surface was slightly grainy (Fig. 4C). Widespread red blood cell exudation, inflammatory cell infiltration, and congested dilated blood vessels were observed (Fig. 4H&I), whereas the glomerular morphology appeared normal (Fig. 4H).

4. Discussion

This case presented with acute onset with dyspnea as the main manifestation. The patient's activity tolerance decreased most significantly before admission, with no orthopnea or severe chest pain noted. Troponin showed a downward trend at the time of treatment (Fig. S3). Combined with all of the clinical manifestations, the exacerbation of dyspnea before admission was considered to be combined with ventricular septal perforation, low cardiac output, systemic circulation congestion, and heart failure.

VSR usually presents as rapid-onset clinical deterioration with acute heart failure or cardiogenic shock, and a shunt may result in signs and symptoms of acute right heart failure [7,8]. The only definitive treatment is surgical repair [8], while medical therapy still lacks standardized recommendations. Through the autopsy, we found that the patient complicated with multiple organ hemorrhage, while severe congestion was observed beside the infarcted heart. Additionally, there was mild edema and hyperemia in the brain and kidney, highlighting the importance of carefully considering thrombolysis and anticoagulation therapy for patients with AMI and VSR, as well as the need to conduct a close follow up. However, currently, no coronary computed tomography angiography [9] results could have helped the doctors to obtain a clearer picture of the patient's condition and improved the efficiency of clinical decision making [10].

Anatomical and pathological evidence of microbleedings in the brain and kidney consistently indicates hemorrhage in multiple organs. Laboratory disseminated intravascular coagulation (DIC) testing performed the day before the patient's death showed hypofibrinogenemia [11]. According to the International Society of Thrombosis and Hemostasis (ISTH) DIC score [12–14], the patient was compatible with overt DIC (platelet count: 191 $\times$ 10^9/L, 0; D-Dimer: 7.812 mg/L, 3; prothrombin time (PT) 39.8 s, 2; fibrinogen 1.48 g/L, 0; DIC score = 5). To the best of our knowledge, this is the first autopsy-confirmed case showing post-infarction VSR with DIC consequence. This case suggests that patients with VSR after MI may be accompanied by hemorrhage in multiple organs, and that anticoagulation therapy should be more comprehensively evaluated in these patients during clinical management. Further experiments should be conducted to observe the coronary arteries, which we did not focus on in this report.

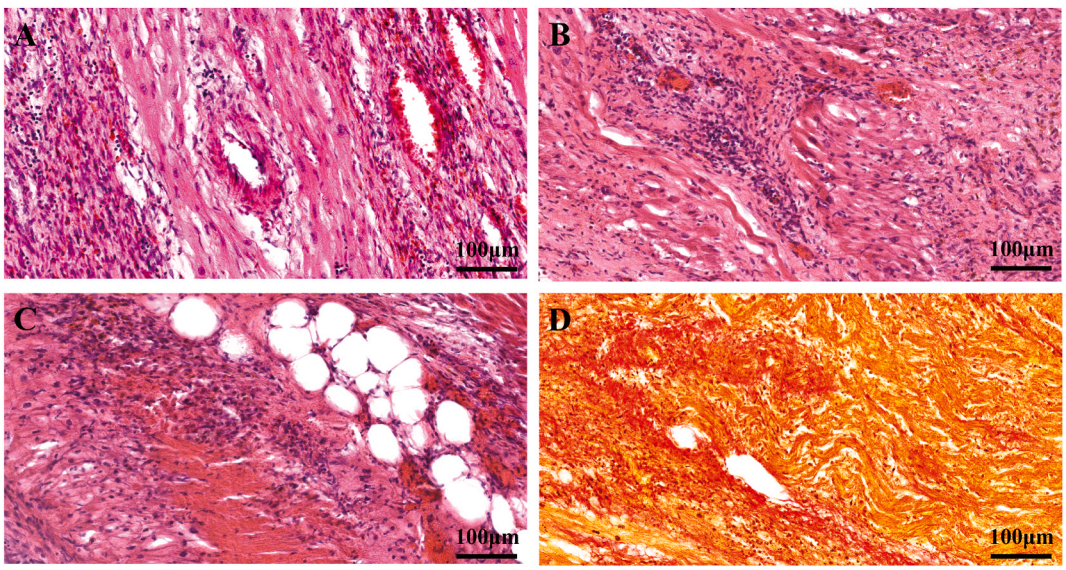

Fig. 3. (A) Microvascular damage and extravasation of erythrocytes. (B) Massive interstitial and perivascular infiltration of inflammatory cells (mainly mononuclear leukocyte). (C) Some fat infiltration stained as vacuoles. (A–C) H&E stain. (D) Microvascular congestion and fibrosis of the vessel wall; Sirius Red stain. Scale bar for (A–D): 100 μm. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

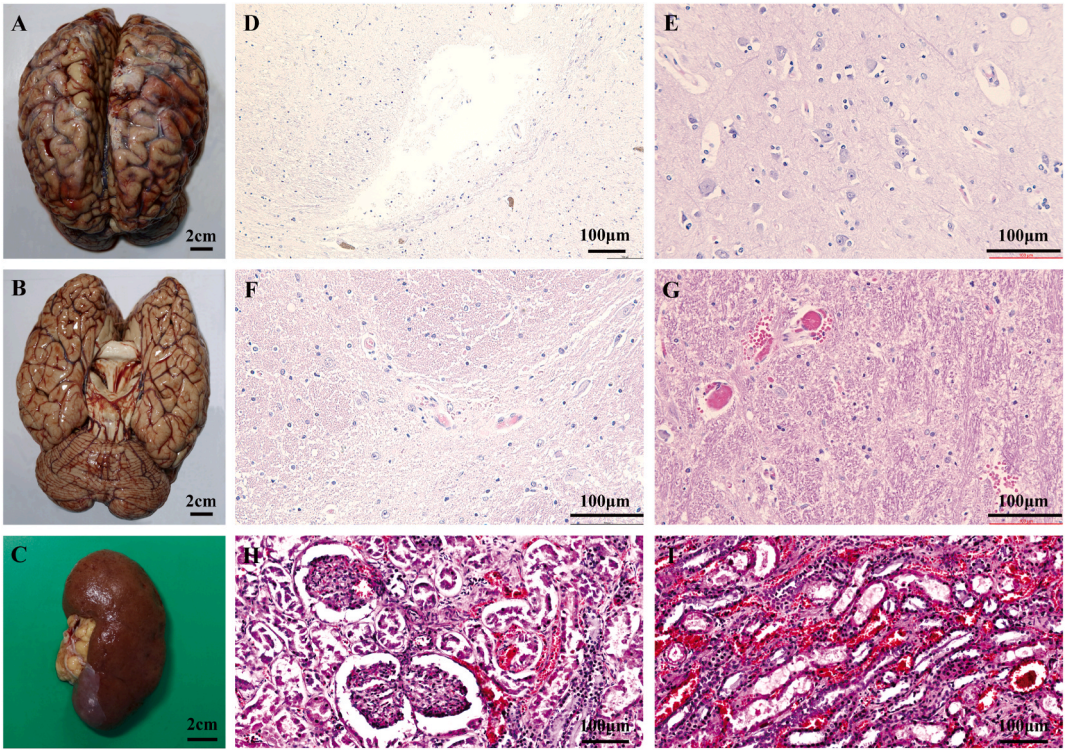

Fig. 4. Hemorrhage of multiple organs. Gross view of the brain from (A) superior and (B) basilar aspects. The whole brain was approximately normal in size without atrophy; the color of the whole brain tissue was dark. An old infarction lesion is observed in the midbrain (D). Moderate perivascular dilatation and mild neuron loss were observed in the gray matter of the motor cortex (E). Mild microbleeds were observed in the basal ganglia (F) and medulla (G). (C) Gross view of the left kidney: the surface is grainy and no severe pathological changes in glomerular morphology were noticed (H), while inflammation cell infiltration, congestion, mild dilation of the blood vessel, and exudation of red blood cells were found pervadingly (I). (D–I), H&E stain. Bar for (A–C): 2 cm; bar for (D–E): 100 μm; bar for (G–F): 50 μm; bar for (H–I): 100 μm. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

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CRediT authorship contribution statement

Bing-Jie Xue: Writing – original draft, Data curation. **Wen-Zheng Hu:** Writing – original draft, Data curation. **Chong-You Lee:** Investigation. **Qing Yang:** Data curation. **Li-Xin Jia:** Methodology, Investigation, Data curation. **Yuan Wang:** Investigation. **Yue Huang:** Writing – review & editing, Supervision, Investigation. **Bo-Kang Qiao:** Writing – review & editing, Writing – original draft, Methodology, Funding acquisition, Data curation, Conceptualization. **Jie Du:** Writing – review & editing, Funding acquisition, Conceptualization.

Declaration of competing interest

None.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e25315.

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