

Embolization for infective endocarditis with spontaneous spleen rupture: A case report

Shuchong Mei; Ruiyan Xu; Yibing Wang*

*Corresponding Author: Yibing Wang

Department of Emergency, the Second Affiliated Hospital of Nanchang University, Jiangxi, China.

Email: house911cuddy@126.com

Abstract

Infective Endocarditis (IE) can occasionally induce spleen rupture and bleeding, leading to haemorrhagic shock [1]. We report a case in which a patient was admitted to the hospital due to fever, with a history of rheumatic heart disease and heart valve replacement. The patient exhibited sudden abdominal pain and went into shock during the course of the disease. Our examination revealed anemia and blood culture showed the presence of *Staphylococcus aureus* accompanied by a ruptured spleen. After anti-shock treatment, embolization to stop bleeding, and antibiotic treatment, the patient was discharged after 47 days of hospitalization. This case reveals that for patients with IE and spontaneous splenic rupture, embolization can achieve positive results and may result in less trauma and faster recovery than open splenectomy.

Keywords: Infective endocarditis; Spleen rupture; Haemorrhagic shock; Embolization; Antibiotic treatment.

Case Presentation

The patient was a 63-year-old male. He was admitted to the hospital after five days of fever. In May 2018, he underwent mitral valve and aortic valve replacement with left atrial appendage ligation in our hospital for rheumatic heart disease. The patient began to experience nasal congestion and rhinorrhea on April 7, 2019 and developed a fever (temperature not measured) accompanied by chills, general weakness, chest tightness, poor appetite, lower extremity pain, mild cough, and production of a small amount of white sputum. On April 12, 2019, the patient felt his symptoms worsening as well as chest tightness after minimal activity. He arrived at our hospital for a physical exam and exhibited the following: Blood pressure 120/80 mmHg, body temperature 39.7°C, heart rate 126 beats/min, pulse 100 beats/min, and 30 breaths/min, minimal atrial fibrillation, no significant murmur, scattered wet crackles in the left lower lung, soft abdomen without tenderness upon applied pressure or rebound, and no edema in the lower limbs. Routine blood work showed the following: $12.83 \times 10^9/L$ white blood cells, 110 g/L hemoglobin, $48 \times 10^9/L$ platelets;

coagulation function: INR 1.28, APTT 54 s; liver function: Total bilirubin 54.3 $\mu\text{mol/L}$, direct bilirubin 39.1 $\mu\text{mol/L}$, indirect bilirubin 15.2 $\mu\text{mol/L}$, alanine aminotransferase 55.0 U/L, aspartate aminotransferase 65 U/L, C-reactive protein 54.15 mg/L, PCT 16.01 ng/ml; renal function: creatinine 119.92 $\mu\text{mol/L}$, NT-pro-BNP 8651.10 pg/ml, no abnormalities in troponin, myocardial enzymes, blood coagulation, and routine urine results; ECG: Rapid atrial fibrillation; emergency chest CT: Inflammation of the lower left lobe; cardiac ultrasound: Mitral and aortic valve replacement, enlarged left atrium, slightly larger left ventricle, and fullness in the right ventricle, thickened ventricular septum, thickened anterior wall of the right ventricle, and widened aorta, mild reflux in the tricuspid and pulmonary valves, and diminished left ventricular diastolic function. These observations led us to consider the possibility of IE and to improve blood bacterial culture, ceftriaxone sodium (Rocephin), amoxicillin, and potassium clavulanate were used to prevent infection and to treat symptoms. The patient experienced sudden upper abdominal pain on April 14 around 15:20, with profuse sweating, blood pressure of 86/63 mmHg, body temperature of 38.1°C, heart rate of 124 beats/min, abdominal distension mainly in the left upper quadrant, and upper abdominal tenderness, and was transferred to the emergency room. The ECG data revealed tachycardia, atrial fibrillation, and ST-T changes. The patient's abdominal pain was slightly relieved upon producing tan stools. The blood test showed $16.97 \times 10^9/\text{L}$ white blood cells, 78 g/l hemoglobin, and $64 \times 10^9/\text{L}$ platelets. Planar CT of the chest and abdomen indicated splenic rupture and haemorrhage, abdominal pelvic effusion and haemorrhage, postoperative changes in heart valves, enlarged heart, subpleural inflammation on the dorsal side of both lungs, and small pleural effusion on both sides of the chest (Figure 1). Diagnostic abdominal fluid draw revealed the presence of non-coagulated blood. The patient was transfused with 4 U red blood cells and emergency splenic artery angiogram and embolization was performed. During surgery, retention and extravasation of contrast agent was observed from the inferior splenic artery, which led to the consideration of inferior spleen haemorrhage. Polyvinyl alcohol particles were administered and a coil was applied to embolize the inferior splenic artery. This was followed by angiography showing blockage of the inferior splenic artery and the absence of bleeding (Figure 1). Blood culture was performed again that night and showed the presence of Gram-positive bacteria (*S. aureus*). The patient was then treated with vancomycin combined with piperacillin and tazobactam to treat the infection and symptoms. The patient was transferred from the ICU ward to the general ward on the 11th day of initial disease presentation, and was discharged on the 47th day.

Discussion

IE refers to a series of infectious diseases characterized by inflammation caused by bacterial or fungal infection, or infection by other microorganisms. In recent years, with increased population age, there has been an increase in elderly patients with degenerative heart valve disease. At the same time, the use of artificial heart valve replacement, implants, and various endovascular intervention techniques, have been accompanied by an increasing number of IE occurrences. Its clinical manifestations and pathogenic microbial composition have also changed significantly [2,3]. The Duke criteria are currently used as the standard for the diagnosis of IE, with a diagnostic specificity of 99% and a sensitivity of 80% [4]. Although no typical neoplasms were seen in our patient's cardiac ultrasound, three secondary criteria from the Duke criteria were present, namely susceptible factors (heart valve replacement), fever (temperature > 38°C), and microbiological evidence (blood culture suggesting *S. aureus*) and as such, the patient was considered to have

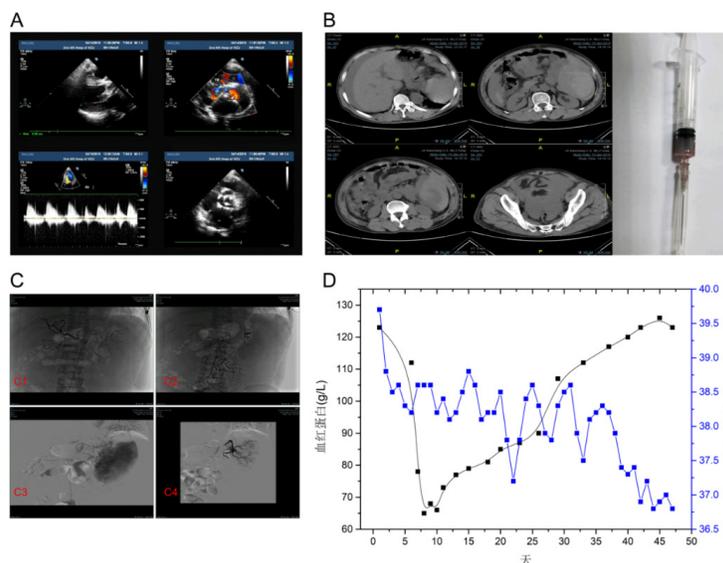


Figure 1: **A:** No obvious neoplasms were observed in the color Doppler ultrasound. **B:** Abdominal CT and diagnostic abdominal puncture suggest hemorrhaging in the abdominal cavity. **C:** **c1:** no contrast medium extravasation in hepatic arteriography; **c2:** no significant contrast medium extravasation in superior mesenteric arteriography; **c3:** contrast agent extravasation in splenic arteriography; **c4:** no contrast extravasation after inferior splenic artery embolization. **D:** Changes in the highest body temperature, hemoglobin, and mean radial arterial systolic blood pressure during patient treatment.

IE based on clinical observation.

IE embolism is a common complication secondary to heart failure. Embolism can occur from days to months after the onset of fever. The most common sites of embolism are the brain, kidney, spleen, and coronary arteries. A relatively large spleen embolism can cause sudden upper left abdominal or left shoulder pain, left pleural effusion and splenomegaly. In this case, on the seventh day after the initial fever, the patient experienced sudden upper left abdominal pain, significantly decreased hemoglobin compared to the previous test result. These observations, combined with whole abdomen CT of and aspiration of non-coagulated blood from the abdominal cavity, with no history of external trauma made the diagnosis of spontaneous splenic rupture clear. Spontaneous splenic rupture and bleeding are clinically rare. Renzull *et al.* studied the causes of spontaneous splenic rupture and found that they occur in cases with, in order of incidence: tumor, infection, non-infectious inflammation, drug-related and treatment-related, mechanical disorder, and normal spleen. Other causes include splenic infarction, coagulopathy, thrombocytopenia, splenic venous thrombosis, and focal spleen lesions [5]. Potential causes for comorbid IE and spontaneous splenic rupture include: local necrosis and rupture of the spleen due to focal spleen embolism or spontaneous rupture and bleeding of the spleen due to localized *S. aureus* aggregation and purulence [6].

The treatment for spontaneous splenic rupture should address both the primary disease and the ruptured spleen. For patients with hemodynamically stable splenic rupture, conservative treatment can be used [7]. During the treatment, routine blood tests, B ultrasound or CT should be performed dynamically to closely observe the hematoma. If the hematoma continues to increase and the splenic capsule ruptures, emergency surgery is required. The surgical method can be determined based on intraoperative exploration and may consist of partial splenectomy, spleen repair, total splenectomy, or arterial embolization. Our patient developed haemorrhaging in the abdominal cavity and was hemodynamically unstable and there-

fore had to be undergo emergency surgery. Based on the patient's underlying heart disease and the risk of infection after splenectomy, he was treated with splenic arterial embolization and his condition stabilized after surgery.

Anemia is one of the common symptoms of IE. Approximately 70% to 90% of patients have progressive anemia, which is mainly related to the infection suppressing bone marrow. In this patient, a significant decrease in hemoglobin occurred within a short period of time, which cannot be fully explained by IE. Therefore, when a change in a clinical indicator cannot be fully explained by the current etiology, we must actively search for other complicating factors. This patient presented with hypotension induced by haemorrhagic shock caused by splenic rupture, after embolization and blood transfusion, the patient's blood pressure stabilized.

Although IE itself can also cause shock, IE with spontaneous splenic rupture, bleeding, and shock is rare. In this case, the patient's shock status improved after embolization. Therefore, in our diagnosis and treatment of IE shock, we should consider the clinical presentation in a comprehensive manner to avoid missed diagnoses, misdiagnoses, and delayed treatments. At the same time, whether the use of embolization for spontaneous splenic rupture and hemorrhagic shock in IE patients is superior to splenectomy require additional clinical data to determine.

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Manuscript Information: Received: July 05, 2023; Accepted: August 17, 2023; Published: August 23, 2023

Authors Information: Shuchong Mei; Ruiyan Xu; Yibing Wang*

Department of Emergency, the Second Affiliated Hospital of Nanchang University, Jiangxi, China.

Citation: Mei S, Xu R, Wang Y. Embolization for infective endocarditis with spontaneous spleen rupture: A case report. Open J Clin Med Case Rep. 2023; 2097.

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