

A Case of Rheumatic Heart Disease Complicated by Acute Infective Endocarditis and Valve Leaflet Perforation

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Abstract

Rheumatic Heart Disease (RHD), abbreviated as rheumatic heart disease, is a heart disease caused by repeated attacks or further deterioration of Acute Rheumatic Fever (ARF), which is mostly caused by valve damage (Zhang P & Wang P., 2021). The production of ARF is the result of a series of autoimmune reactions by group A streptococci that invade the pharynx of a susceptible host. Clinically, rheumatic heart disease mostly involves valves, which can cause patients to have a variety of manifestations such as incomplete valve closure or stenosis, structural remodeling of the heart, etc. If patients do not seek medical attention in time to delay the best time for treatment, they often develop acute heart failure, malignant arrhythmias and other acute complications of the cardiovascular system in the later stages of the disease. Clinically, rheumatic heart disease complicated by infective endocarditis is not common, and valve leaflet perforation due to severe infection is even rarer! A case of acute infective endocarditis complicated by atypical rheumatic heart disease and simultaneous valve leaflet perforation in our hospital is reviewed and reported below.

Keywords: rheumatic heart disease, acute infective endocarditis, valve leaflet perforation, case report

1. Case Information

The patient is a 54-year-old female who was admitted to the hospital with "exertional shortness of breath with bilateral lower

extremity edema for more than 8 months, aggravated for more than 10 days". The patient felt shortness of breath with bilateral lower extremity edema and generalized weakness

during activity 8 months ago, which could be relieved after rest, and had no special symptoms. He was admitted to a Chinese medicine hospital, where a cardiac ultrasound showed rheumatic heart disease, and he was taking diuretics (the name and dosage of the drug are unknown). In the last 10 days or so, he felt that his activity tolerance was significantly reduced, the edema of both lower limbs was further aggravated, and he could not lie down at night. D-dimer 3.0mg/ml, cTnI 0.36ng/ml, CK-MB 7.74ng/ml, Myo 79.62ng/ml, NT-proBNP 19064.2pg/ml. Cardiac ultrasound: 1. severe mitral stenosis; 2. left atrial right atrial enlargement, right ventricular fullness; 3. tricuspid valve massive regurgitation; 4. moderate pulmonary hypertension; 5 The left ventricular thrombosis was possible; 6. hyposystolic left ventricular function (LVEF: 35%). He was admitted to our department as an emergency patient with "heart failure". He was previously found to have abnormal blood glucose for more than 8 months and had been taking oral metformin 1 tablet/time 1 time/day. Also, his blood sugar was not regularly tested, and no special medical history, no history of alcohol or tobacco. On admission: Vital signs: temperature: 36.0°C; heart rate: 120 beats/min; respiratory rate: 20 breaths/min; blood pressure: 115/84 mm Hg. Examination: acute illness, clear consciousness, autonomic position, clear respiratory sounds in both lungs, no dry and wet rales heard. The heart borders were enlarged to both sides, the rhythm was definitely irregular, the heart rate was 127 beats/min, the pulse rate was 120 beats/min, the first heart sound was unequal in strength, a grade 4/6 systolic murmur could be heard in the mitral valve auscultation area, no pathological murmur could be heard in the remaining valves, no pericardial fricative sound could be heard, both lower limbs were heavily depressed edema, physiological reflexes existed, pathological reflexes were not elicited. He was admitted to the hospital and given symptomatic supportive treatment such as cardiac monitoring, oxygen, anticoagulation, diuresis, improvement of ventricular rate, and correction of electrolyte disturbance.

2. Disease Evolution

The patient's condition was recurrent and worsened after 1 week of hospitalization, with several episodes of sleepiness during the illness, transient delirium, chills and hyperthermia, Tmax 40.0°C, sustained shock blood pressure,

emergency blood gas analysis suggesting type II respiratory failure (PCO₂ 154mmHg), blood cell analysis results returned WBC: 34.59*10⁹/L, NT-proBNP32358.00pg/ml, CRP: 34.27mg/ml, PCT: 8.58ng/ml, creatinine: 120.0umol/L, chest CT showed focal inflammation in both lungs, localized solid lung tissue in the middle lobe of the right lung, and multiple dense shadows in the valve area. The patient's blood pressure, renal function, and coagulation abnormalities were combined with septic shock (Alrikabi, Saad Khalaf et al., 2020), norepinephrine combined with dopamine and epinephrine micro-pump (1.0ug/kg/min) was given to maintain blood pressure, active bedside hemofiltration, fresh frozen plasma transfusion to correct the patient's coagulation function, acid suppression to prevent stressful gastrointestinal bleeding, and again. The patient was given fresh frozen plasma to correct coagulation, acid suppression to prevent stressful gastrointestinal bleeding, and antibiotics (cefoperazone sulbactam imipenem cistatin sodium combined with vancomycin). 1 day later, a repeat cardiac ultrasound indicated hyperechoic masses in the anterior mitral leaflet. Surgery was proposed. On October 15, 2022, the surgery was performed, during which the pericardium was incised and heparinized to establish extracorporeal circulation, and after cardiac arrest, the left atrium was incised, probed, and a large amount of thrombus was seen in the left atrium. "Mitral valve replacement + tricuspid valvuloplasty + left atrial thrombus removal + left auricular suture", intraoperative delivery of a mitral valve-like tissue, size of about 3.0*3.0*1.0cm, visible calcification, 4 pieces of material, postoperative with tracheal intubation and returned to the monitoring ward, 5 days later pathology diagnosis return: mitral fibrous tissue with bosom and calcification.



Figure 1. Intraoperative delivery of mitral

valve-like tissue (approximately 3.0*3.0*1.0 cm in size)



Figure 2. Visible under the microscope (HE stain 10x microscope formalin solution fixed)

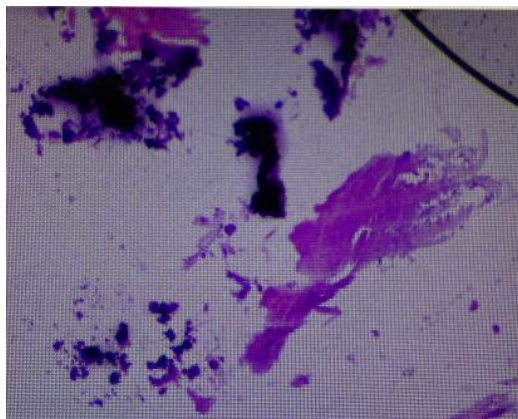


Figure 3. Visible under the microscope (HE stain 10x microscope formalin solution fixed)

3. Infective Endocarditis

3.1 Basic Overview of Infective Endocarditis

Infective endocarditis (IE) is an inflammatory disease caused by a direct attack of the endocardium or valves by fungi, bacteria, and other microorganisms (e.g., spirochetes, chlamydia, etc.). The disease can be accompanied by the formation of redundant organisms causing structural remodeling of the heart. The atrial surface of the mitral or tricuspid valve and the ventricular surface of the aortic and pulmonary valves are usually favored locations for these pathogens, with heterogeneity and, in severe cases, defective,

perforated, or ruptured tendons (Lei Hao, Cheng Shanglin, Shang Bin et al., 2021). The global incidence of IE is not high, ranging from 3 to 9 per 100,000 per year, with a male-to-female ratio of approximately 2:1 and a recurrence rate of 5% to 10%. However, it has a mortality rate of up to 20% and an annual mortality rate approaching a staggering 30% (Zhigalov K, Sá MPBO, Kadyraliev B et al., (2020). In clinical practice, the majority of patients with IE present with fever, chills, and reduced BMI; 85% of patients have a heart murmur, and the incidence of embolism can be as high as 25% at diagnosis. Therefore, IE must be suspected in any patient presenting with fever and embolic manifestations (Liang F, Hu DY, Shen ZJ et al., 2017).

3.2 Diagnostic Criteria

Using the Modified Duke Diagnostic Criteria (Liang F, Hu DY, Shen ZJ et al., 2017): The main criteria: (1) positive blood cultures, i.e., 2 different blood cultures with typical pathogenic organisms of infective endocarditis or persistent positive blood cultures of microorganisms consistent with infective endocarditis or non-pathogenic organisms as described above; (2) positive single blood culture for Cocksackie or stage 1 IgG titer >1:800; (3) positive echocardiographic findings of infective endocarditis, such as redundancies, abscesses, new valve perforations, or regurgitation. (4) Positive echocardiographic findings of infective endocarditis, such as bulges, abscesses, new valve perforations or regurgitation. Secondary criteria: (1) predisposing factors, such as underlying heart disease or intravenous drug addiction; (2) temperature above 38°C; (3) signs of vascular damage; (4) signs of immune abnormalities; (5) positive blood cultures but failure to meet the primary criteria, or active bacterial infection consistent with infective endocarditis. Two major criteria, or one major criterion + three minor criteria, or five minor criteria were met to determine the diagnosis.

4 Analysis of This Example

4.1 Surgical Indications for Active IE

Before the middle of the 20th century, antibiotic treatment was almost the only means of treating IE, but after the middle of the 20th century, surgical treatment of IE was no longer a fantasy due to the increasing maturity of cardiothoracic medicine and cardiac extracorporeal circulation. (Zhu LP, Zhao JL, Fu L et al., 2022) According to

the literature, the surgery rate in foreign countries is more than 50%, while the surgery rate in excellent cardiac centers in China is a staggering 88.3% (Chen X W, Liu Y X, Yu H, et al., 2018). To date, the accepted surgical indications for the treatment of progressive IE are (1) prosthetic valve endocarditis; (2) symptomatic heart failure, severe valve regurgitation, and recurrent embolic complications; (3) large bulky masses (>10 mm); and (4) bacteraemia and perivalvular complications lasting more than 3 days under anti-infective therapy.

4.2 Specific Analysis

The patient's condition suddenly worsened 1 week after admission, and the bedside cardiac ultrasound suggested hyperechoic masses of the anterior mitral valve leaflets were superfluous. Sun Yanrong et al. (Habib G, Lancellotti P, Antunes M J, et al., 2015) In a study to explore the application value of echocardiography in the clinical diagnosis of infective endocarditis, it was found that the detection rate of superfluous organisms in infective endocarditis by echocardiography was 100%, so the patient was considered to have a high possibility of IE, after which the patient sustained shock blood pressure and excluded embolism. After the embolic shock, hypovolemic shock, cardiogenic shock, anaphylactic shock and neurogenic shock, severe infectious shock was considered, and the patient's primary disease was rheumatic heart disease. Cheng Jun (Sun Y R, Wei L Y, Lin S P et al., 2022) et al. found in the study of the underlying etiology and causative agents of 802 patients with IE that although there was a decreasing trend of rheumatic heart disease leading to IE, it still belonged to the underlying cardiac disease of IE, based on this, it was not excluded that due to rheumatic Based on this, it cannot be excluded that acute severe infection due to rheumatic heart disease leads to symptoms and echocardiographic manifestations of IE. Some studies have pointed out that the positive rate of blood culture in the entire IE population is less than 70% (Cheng J, Hu H, Zhang H, et al., 2020), and since blood culture is the most reliable direct evidence for the diagnosis of IE, this patient was repeatedly left with negative blood culture, with the following possible reasons: (1) antibiotics were used before blood culture (Cahill T J, Baddour L M, Habib G, et al., 2017). (2) Under some conventional culture conditions, caustic bacteria

have difficulty growing. (3) Chemotherapy is being administered (Liu X Pei & Miao Q., 2021). This patient had a second cardiac ultrasound suggestive of mitral leaflet root perforation, which reaffirmed the serious adverse event of IE triggering a serious infection leading from a first cardiac ultrasound suggestive of a superfluous leaflet to a second leaflet perforation. According to the modified Duke diagnostic criteria, this patient was again considered to be associated with prophylactic antibiotic use, although multiple blood cultures were negative, and the case did not meet the primary diagnostic criteria, but still met the 5 secondary criteria, so the clinic again pointed to the diagnosis of IE. Since the patient had a concurrent leaflet perforation event at this time, and the patient had a severe infection and persistently elevated NT-Pro BNP, which were consistent with the indications for surgical treatment of progressive IE, the intraoperative incision of the pericardium revealed bacillary emboli, which again confirmed the concurrent severe infection based on wind cardiopathy leading to the acute phase of IE with concurrent leaflet perforation! In a retrospective study of 89 patients with infective endocarditis treated surgically, Li Dagang et al. (An Xiaoxia & Zhou Yunfang, 2008) concluded that aggressive surgical treatment of patients with IE improves the cure rate and reduces mortality, and is an effective method of curing infective endocarditis, and specifically noted that patients with IE involving valves and annuli require aggressive early surgical interventions. Based on this, timely surgical intervention in this patient could improve the long-term prognosis of the patient.

5. Discussion

The formation of valvular redundancy is the most common pathological damage in IE, and clinically it is most common in the left heart system. Once formed, the redundancy is easily dislodged into the body circulation, resulting in acute embolic events, especially cerebral embolism, which can cause death. As the disease progresses, complications such as valve defect or perforation can occur in IE, leading to acute heart failure (Li DAG, Meng CHY, Zhao P et al., 2020); therefore, result in the early and prompt identification of IE in clinical practice, i.e., early diagnosis can provide us with the conditions for early surgery, and early surgery can greatly improve the clinical cure rate of patients (An Xiaoxia & Zhou Yunfang, 2008). In this case, the

patient underwent mitral valve replacement, tricuspid valvuloplasty, left atrial thrombus removal, and left auricular suture immediately after diagnosis; moreover, the prognosis is good. By reporting on an exceptional case, this example aims to provide ideas and lessons for clinical treatment.

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