The Heart Surgery Forum 2021-3617
24 (2), 2021 [Epub April 2021]
doi: 10.1532/hsf.3617

Online address: http://journal.hsforum.com

Infective Endocarditis with Recurrent Epistaxis in a Young Patient: A Case Report

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ABSTRACT

Epistaxis is a common emergency, and its main causes are hypertensive crisis and trauma. Nasal packing is the primary treatment. After active symptomatic treatment, the symptoms of epistaxis effectively can be controlled. In this case report, the patient was treated with epistaxis many times in the outpatient department. After nasal examination, there was a clear bleeding point, and it was treated with gauze packing or silver nitrate cauterization. The symptoms of epistaxis gradually got worse and was accompanied with fever and progressive anemia. After blood culture and color Doppler ultrasound examination, it was confirmed that it was endocarditis caused by defective hypoxic bacterial infection. After active antibacterial and surgical treatment, the symptoms of epistaxis, fever and anemia were relieved.

INTRODUCTION

Infective endocarditis is caused by bacteria and other microorganisms infecting the endocardium, especially the heart valve. Its characteristic lesion is the formation of vegetation, which often occurs in the heart valves, congenital cardiovascular malformation or after artificial valve replacement [Gungor 2012]. Fever and anemia are common clinical manifestations. Atypical infective endocarditis often is covered by the clinical symptoms of complicated diseases. Mild cases only show bleeding from the nose, while severe cases can lead to hemorrhagic shock [Wang 2014]. The causes also can include local and systemic factors. In this case, the etiology of recurrent epistaxis is caused by two factors. After the control of infective endocarditis, the epistaxis was cured dramatically.

CASE REPORT

A 21-year-old male patient had the habit of blowing his nose, digging at it, and staying up late; he had no clear history of congenital heart disease. In May 2020, he suffered from a moderate amount of epistaxis after nasal excavation, which was relieved after treatment in the clinic near his home (specific drugs are unknown) and self tamponade (toilet paper). After three times of intermittent epistaxis, the patient was relieved after self tamponade, and no rhinoscopy was performed. On September 30, he felt sore limbs, his body temperature was 380 C, and blood routine examination in other hospitals showed that WBC was 7.67×10^9/L, RBC was 3.56×10^{12}/L, HGB was 98g/L, MCV was 85.5fl, MCH was 27.4pg, MCHC was 321g/L, PLT was 148×10^9/L. After symptomatic treatment, he left the hospital. One week later, his epistaxis reoccurred. Nasal endoscopy showed active bleeding points in the right and left nasal septum. The blood routine examination showed that WBC 12.79×10^9/L, RBC 3.56×10^{12}/L, Hgb 96g/L, MCV 82.9fl, MCH 27.1pg, MCHC 327G/L, PLT 223×10^9/L. After the examination, Azithromycin tablets 0.5g was given orally 1 tablet per day (course of 5 days) and furosemide nasal drops.

On October 16, epistaxis with high fever reoccurred again. The body temperature was 380C ~ 390C. Rhinoscopy examination in other hospitals showed that the nasal septum was deviated, the right side of the nasal septum has bleeding, left side of the patient's area had bleeding spots, and blood routine examination showed that WBC was 8.67×10^9/ L, RBC was 2.76×10^{12}/L, HGB was 78g/L, MCV was 88fl, MCH was 28.3pg, MCHC was 321g/L, and PLT was 190×10^9/L. His chest CT showed that his spleen was enlarged, and the density was not uniform. Direct antiglobulin test was positive, Ana 1:100; anti dsDNA was weakly positive; bone marrow biopsy smear showed that the proliferation of granulocytic and erythrocyte lineage was significantly active, with a slightly higher proportion of plasma cells. Some nucleated cells were degenerated and mature red blood cells were dissolved. There was no evidence of abnormal immunophenotype associated with acute leukemia, NHL and high-risk MDS.

On October 28, the blood routine examination showed that WBC 12.58×10^9/L, RBC 2.26×10^{12}/L, Hgb 61G/L, MCV 89.8fl, MCH 27pg, MCHC 300g/L, PLT 203×10^9/L. Considering autoimmune hemolytic anemia, we mainly give prednisone acetate short-term shock therapy, assisted by nasal packing and (compound paracetamol and amantadine capsules) cooling treatment, but the curative effect was not good. The frequency of epistaxis and high fever gradually increased, mainly in the morning and at night, accompanied by cardiac fatigue and chest tightness after activities.

Received January 16, 2021; accepted February 7, 2021

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Blood routine examination on November 6 in our hospital showed that WBC was 11.51 × 10^9/L, RBC was 2.05 × 10^{12}/L, HGB was 53g/L, MCV was 86.3fl, MCH was 25.9pg, MCHC was 299G/L, PLT was 219 × 10^9/L. Urine routine occult blood + + +; our hospital outpatient find he had severe anemia and he was admitted to our department. His examination showed anemia, sublingual vein thickening, arrhythmia, apical area and aortic valve second auscultation area can hear with systolic murmur. Color Doppler echocardiography showed infective endocarditis, severe regurgitation of perforated plate of anterior mitral valve (Figure 1 and Figure 2), formation of mitral valve vegetations (Figure 3), multiple vegetations in left atrium (Figure 4), mild tricuspid regurgitation, a small amount of pericardial effusion, and left ventricular false chordae tendineae. (Figure 1) (Figure 2) (Figure 3) (Figure 4)

According to the improved Duke diagnostic criteria, infective endocarditis was clear, in this case the simple anti-inflammatory effect was not good, then the patient was surgically operated under hypothermic cardiopulmonary bypass with thoracoscopic mitral valve replacement. Postoperative pathological section of mitral valve membrane showed that the valve tissue had hyaline degeneration, lymphocyte infiltration with necrosis. After operation, anti-inflammatory and warfarin anticoagulation therapy were continued, and his epistaxis and fever were relieved.

On December 6, the blood routine examination showed that WBC was 10.19 × 10^9/L, rbc3.57 × 10^{12}/L, HGB was 96g/L, MCV was 82.6fl, mch26.9pg, mchc325g/L, plt262 × 10^9/L. Urine routine occult blood was negative.

DISCUSSION

Infective endocarditis is a rare and serious disease caused by infective foci in the heart. It refers to the inflammation of the heart valve or ventricular wall caused by direct infection of bacteria, fungi and other microorganisms. In the research of Huang et al. [Huang 2020], Streptococcus and Staphylococcus are still the main pathogens of infective endocarditis, and 3.92% of them are deficient hypoxic bacteria. The main reason is that dextran expressed in the cell wall can enhance the ability of bacteria to adhere to the surface of the endocardium. Deficient hypoxic bacteria are present in normal flora in human oral cavity, upper respiratory tract, and intestinal tract. When immunity is low, it can cause bacteremia and infective endocarditis. Gungor et al. [Gungor 2012] reported endocarditis of valve prosthesis caused by nasal packing without systemic prophylaxis of antibiotics. Boumis et al. [Boumis 2018] reported that prophylactic use of antibiotics should be considered to avoid the occurrence of infective endocarditis after long-term nasal bleeding, nasal packing, or other nose trauma intervention. In this case, the patient with multiple epistaxis was given nasal packing, no other possible cause of bacteremia was identified, which can speculate that the occurrence of bacteremia is related to this. Wang et al. [Wang 2014] concluded that the analysis of the medical records of infective endocarditis with heart failure and bacteremia are difficult to

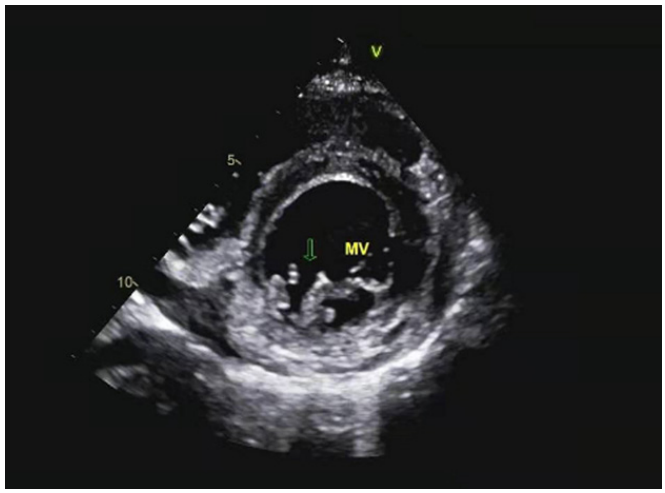

Figure 1. Perforation of anterior mitral valve

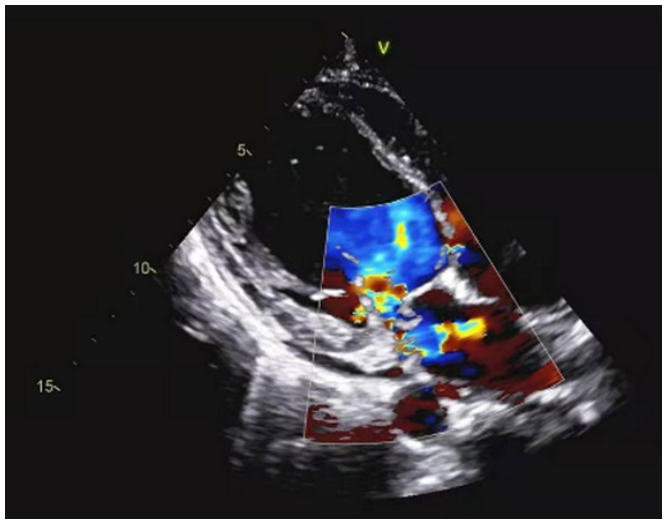

Figure 2. Mitral regurgitation bundle

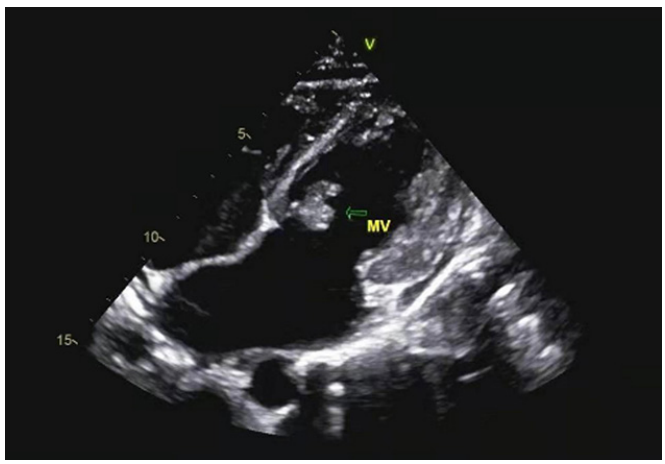

Figure 3. Mitral valve vegetations

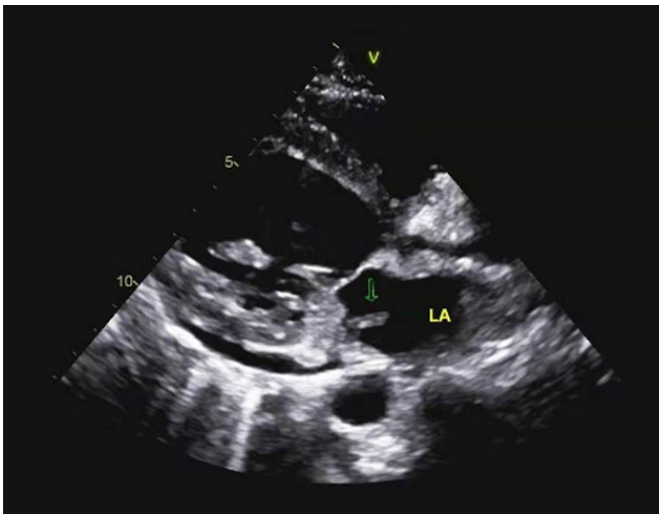

Figure 4. Left atrial vegetation

control by drugs, and these are the common manifestations of dead and unhealed patients. Early surgical treatment is the key to successful treatment.

In this case, the patient had a long course of disease and repeated epistaxis. It was speculated that it was related to the persistence of heart failure and bacteremia. Normally in a young man with good cardiac function, infective endocarditis before the onset is not easy to diagnose. But recurrent nose bleeds were an indication that endocarditis already was present. With the progress of the disease, there were symptoms of heart failure, such as cardiac fatigue and chest tightness after activity, with increased central venous pressure and improved vascular pressure in the drainage area of superior vena cava. This is the same reason that hypertensive patients seek emergency treatment with epistaxis [Lee 2020], which also can explain why epistaxis often occurs with patients in the morning and at night. Due to the persistence of bacteremia, it is easy to fix the value of bacterial thrombus at the damaged nasal mucosa, lymphocyte infiltration, tissue necrosis, normal anticoagulant and procoagulant balance are destroyed [Boeddha 2020], coagulation factors are consumed, and the activity of anticoagulant system is enhanced. Infective endocarditis is prone to secondary infective aneurysms, the incidence of which is 3% ~ 5%, which is more common in intracranial [Chen 2020]. It also is more prone to rupture and bleeding than aneurysms caused by other reasons. After disease control, secondary aneurysms tend to self-heal. This patient had recurrent epistaxis, which could not be excluded because of the absence of local angiography. Irregular high fever in the course of disease, high fever causes nasal mucosa dryness, local vasodilation and congestion, and is also related to the occurrence of recurrent epistaxis. In the course of analysis of epistaxis, nasal anatomy, bad living habits, improper use of drugs (antipyretic drugs, hormones), emotional changes will also lead to difficult control of epistaxis.

The clinical symptoms of infective endocarditis are persistent fever and progressive anemia. Vascular embolism is a serious complication of infective endocarditis, with an incidence rate of 13%-49%. Hemorrhagic complications are relatively rare, especially hemorrhagic stroke. From this case history, we can improve the understanding of atypical symptoms of infective endocarditis and reduce its clinical misdiagnosis and mistreatment.

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