CASE REPORT

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Infective Endocarditis in the Setting of Early-0nset Valvular Disease

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Abstract

In the scope of cardiac valvular disease, aortic stenosis accounts for approximately one-fourth of chronic cases[1]. Valvular disruption may lead to a range of complications, from heart failure to infection. In the setting of bacteraemia, stenotic valves may serve as a nidus for microorganisms, and thus infective endocarditis (IE) may ensue. When arriving at the diagnosis of IE, it is imperative to remember the pathophysiology, particularly when diagnostic tests are inconclusive. We present a case of early-onset symptomatic aortic stenosis and subsequent IE.

Key words: Aortic valve; Rheumatic heart disease; valvular abscess.

Introduction

While primary valvular disease ranks below other chronic medical conditions in prevalence, it contributes significantly to morbidity and mortality. Aortic stenosis occurs due to degenerative calcification, most often in the setting of congenital anomalies, rheumatic heart disease, or chronic deterioration. The pathophysiology of aortic stenosis is progressive, resulting in an average 0.1 cm^2 annual decrease in valvular area[2]. Given this indolent progression, patients typically become symptomatic after the sixth decade. Likewise, the patient population affected by IE has also steadily increased during the past 40 years, with more than half of all IE cases in the United States and Europe occurring in those over the age of 60. At the time of IE development, approximately three-fourths of patients are identified with a pre-existing structural cardiac abnormality[3].

Case report

A thirty-six-year-old Ethiopian male, originally presenting to hospital for aortic valve replacement, reported high grade fever for twelve days. Patient denied associated symptoms, including sore throat, cough, abdominal pain, diarrhoea, vomiting, and burning micturition. On presentation, patient was febrile (101° F) with remaining vital signs within normal limits. Carotid pulses averaged 80 beats per minute, were slow rising, and palpable as thrills bilaterally. On auscultation, a grade 4/6 systolic ejection murmur was heard, most prominent at the right upper sternal border. The remainder of the systemic examination was unremarkable. The patient was started on vancomycin and ceftriaxone and admitted for further management.

Routine blood tests revealed a normocytic, normochromic anaemia with haemoglobin 12.9 gm/dl and MCV 85.4 fl. Acute phase reactants were elevated, with erythrocyte sedimentation rate (ESR) 110 mm in Ist hour and C-reactive protein (CRP) 115.75 mg/l. Microscopic haematuria was detected, as routine urine culture and microscopy revealed > 50 RBCs/high power field. Two sets of blood cultures were negative and all other lab tests, including CBC, liver function tests, and kidney function were within normal limits. A transesophageal echocardiography (TEE) done on admission was suggestive of severe aortic stenosis, with no vegetations. CT chest and abdomen revealed linear, hyperdense focal parenchymal lesions in the splenic parenchyma, suggestive of splenic infarcts as well as evidence of glomerulonephritis.

Discussion

The physical examination of this patient featured classic findings of aortic stenosis. Aortic stenosis is the result of progressive calcification, often in the setting of a congenital anomaly, rheumatic heart disease, or chronic deterioration[1]. The progressive narrowing in aortic stenosis is cited as the most common cause of left ventricular outflow tract obstruction in both children and adults. The systolic ejection murmur heard with aortic stenosis is the result of increased turbulence, due to increased flow across a narrowed lumen.

Given the relationship between the indolent narrowing of the valvular lumen and symptom presentation, aortic stenosis is typically seen in a patient population greater than 60 years of age. The most common presenting symptom is dysponea on exertion, with other common complaints including angina and syncope[2]. The young age of the patient in this case warrants consideration of an

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underlying cardiac defect. In a case series of 932 adults undergoing surgery for isolated aortic stenosis, 54 per cent exhibited an anatomically abnormal valve. Of this, 49 per cent had a bicuspid valve, and 4 per cent a unicuspid valve. When looking specifically at the seven per cent of patients less than 50 years of age undergoing surgery, approximately two-thirds had a bicuspid valve (BAV)[4]. BAV is the most common congenital valve defect, with a prevalence of 0.5 - 1.4 per cent. Given its predilection for early onset valvular dysfunction, BAV may present with symptoms almost two decades earlier[5].

While working this patient up for his persistent fever, given his history of valvular abnormalities, IE was placed logically at the top of the differential diagnosis. IE is characterised by endocardial infection, typically involving either heart valves or an intracardiac device. Risk factors for IE can be categorised into cardiac and noncardiac aetiologies. Cardiac risk factors include prior IE history, valvular disorder, congenital heart disease, or presence of a foreign device. Noncardiac risk factors include immunosuppression, recent procedure, intravenous drug use, or indwelling intravenous catheter. The most common chief complaint associated with IE is fever. Other clinical manifestations of IE are two-fold, based on patient presentation. Acute IE is characterised by a hectic febrile state. This manifestation of IE may result in rapid destruction of cardiac structures as well as infiltration of extracardiac sites, and may progress to death if left untreated. Subacute IE, on the other hand, presents with a more indolent course, gradually causing cardiac damage.

Predisposition and aetiology of IE vary, based on geographic location. In the developed world, congenital heart disease previously had the highest association with IE. However, most recently this association has shifted towards illicit intravenous drug use, degenerative valve disease, and intracardiac devices. In developing countries, on the other hand, rheumatic heart disease continues to be the mainstay[6]. While this patient's early onset presentation and geographic location mimic the description of rheumatic heart disease, the isolated involvement of the aortic valve suggests other aetiologies.

Metastasis of the valvular vegetation in IE may result in a wide array of systemic complications, thus diagnosis can be challenging. The majority of the lab values of physical exam findings typical of infective endocarditis were not present in this patient. In lieu of these inconclusive findings, radiographic investigations supported the diagnosis. Given the patient's chief complaint of persistent fever in the setting of microscopic haematuria, splenic infarction, and glomerulonephritis, infective endocarditis was diagnosed and treatment was switched to vancomycin and meropenem. A later TEE obtained approximately two weeks after admission revealed small flagellar structures, likely to be vegetations, on the aortic valve.

Conclusion

Given the severity of this patient's murmur, the lack of involvement of other valves, and the relatively young age of presentation, it is likely this patient has a congenital valvular finding, possibly bicuspid aortic valve. Additionally, considering the patient's persistent fever, elevated inflammatory markers, microscopic haematuria, and radiographic evidence of both splenic infarction and also glomerulonephritis, infective endocarditis was the ultimate diagnosis. Although the initial TEE revealed no signs of vegetations, it was imperative to keep infective endocarditis in the differential diagnosis.

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