**Infective endocarditis complicating hypertrophic obstructive cardiomyopathy**

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**KEYWORDS**
Endocarditis; Hypertrophic cardiomyopathy; Echocardiography

**Abstract**
Infective endocarditis is a rare complication of hypertrophic cardiomyopathy. It’s estimated incidence is 1.4 per 1000 person/year in all patients and it increases to 3.8 per 1000 person/year in patients with left ventricular outflow obstruction. The most common site of vegetation is the ventricular aspect of anterior mitral valve leaflet. We report a case of a 43-year-old man who was admitted for mitral infective endocarditis resulting in severe mitral regurgitation complicating a hypertrophic obstructive cardiomyopathy. The patient underwent mitral valve replacement. Post-operative outcome was good with relieve of symptom and resolution of left ventricular outflow obstruction. Literature data are reviewed.

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**Case report**
A 43-year-old man was admitted in our department with dyspnoea, fever and weakness lasting for two months. He had no history of hypertension or known heart disease, no family history of sudden death was reported and he had no symptoms before.

At presentation, the patient was pale with a temperature of 38.5 °C, a tachycardia of 100 beats/min. His blood pressure was 120/70 mm Hg. The auscultation revealed a systolic murmur suggestive of mitral regurgitation. There was no sign of heart failure and all peripheral pulses were present. The dental state was bad with multiple teeth decay. A 12 lead electrocardiogram showed a sinusal tachycardia of 100 /min, a left ventricular hypertrophy and abnormal lateral repolarisation. The chest X-ray showed a cardiothoracic ratio of 0.45. Laboratory investigation revealed a hypochromic microcytic anaemia (haemoglobin, 10 g/dl), leukocytosis of 13.7 x 10⁹ /l, raised inflammatory markers and normal serum creatinine. A set of three blood cultures grew viridans streptococci sensitive to ampicillin. Transthoracic echocardiography disclosed an asymmetric septal hypertrophy (18 mm), posterior wall thickness of 11 mm, hyperkinetic left ventricle, dilated left atrium (two-dimensional area of 30 cm²), systolic anterior motion of the mitral valve with obstruction.

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of the outflow tract (maximal gradient at 64 mm Hg). The mitral valve was thin with no signs of myxomatous degeneration. There was large mobile vegetation on the ventricular aspect of the anterior valve leaflet measuring 1.0/0.9 cm (Fig. 1), a perforation of anterior mitral leaflet resulting in massive mitral regurgitation (Fig. 2). The diagnosis of infective endocarditis was made according to the DUKE university criteria.1 Since renal function was correct and the patient was young, antibiotic therapy regimen consisted of ampicillin plus gentamicin for two weeks and ampicillin alone for another two weeks according to the guidelines of the European Society of Cardiology (ESC).2 This allowed resolution of fever within two days, but two weeks later the patient experienced acute heart failure resistant to medical therapy. He underwent urgent mitral valve replacement with a mechanical prosthetic valve. Post-operative outcome was excellent since heart failure was resolved. Control echocardiography showed relieve of outflow tract obstruction (maximal gradient at 6 mm Hg) and correct function of mitral prosthesis. The patient was placed on a lifelong vitamin K antagonist therapy. After five months of follow-up, he is free of symptom and echocardiographic parameters remain unchanged.

Discussion

Infective endocarditis is a rare complication of hypertrophic cardiomyopathy (HCM). During the last 20 years, only 33 cases have been reported in the English language literature.3,4 Spirito et al.3 followed 810 HCM patients between 1970 and 1997 and reported an incidence of endocarditis of 1.4 per 1000 person/year. The main risk factor was the presence of outflow tract obstruction (present in all patients who developed endocarditis) and left atrial dilation (>50 mm). Patients with both conditions, as in the reported case; carry the highest risk of endocarditis with an incidence of 9.2 per 1000 person/year.

Pathogenesis of infective endocarditis in obstructive HCM can be explained by endocardium damage of the mitral or aortic valve,3,5 consequence of turbulence of blood flow during ejection and of the contact between the mitral anterior leaflet and the septum during systole as well as mitral regurgitation.3,4

Endocarditis develops mainly in the mitral valve.3,5,6 The vegetations are typically located at the ventricular aspect of the anterior mitral leaflet.3,4 The predominant microbial causal agents reported in the literature are staphylococci and streptococci.3,7

Antibiotic therapy is the mainstay of the treatment. Surgery should be considered promptly whenever there is traditional indication (haemodynamic, emboli, persistent fever, abscess). Surgical procedure may consist of valve replacement or repair, and some authors reported relieve of outflow tract obstruction after mitral valve replacement which may be explained by the removal of systolic anterior motion of the mitral valve.8 Valve surgery combined with septal myectomy seems logical but requires great expertise and carries a higher operative mortality.4,9

The American Heart Association and the ESC recommended antibiotic prophylaxis for infective endocarditis in patients with HCM, especially when outflow tract obstruction is present at the basal conditions.5,10

References

Methemoglobinemia revisited: An important complication after transesophageal echocardiography

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KEYWORDS
Methemoglobinemia; Benzocaine; Transesophageal echocardiography

Abstract Methemoglobinemia induced by the use of benzocaine-containing topical anesthetics is a rare, but potentially lethal complication after transesophageal echocardiography (TEE). We report a patient who developed methemoglobinemia after TEE. A review of the literature was performed and the majority of cases of benzocaine-induced methemoglobinemia reported thus far have occurred in patients undergoing TEE, endotracheal intubation, esophagogastroduodenoscopy, and bronchoscopy. All of these procedures have become more frequent than before, and there is a need to reemphasize the potential problem and to reconsider the need for further use of topical anesthetics.

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