Infective Endocarditis After Alcohol Septal Ablation for Obstructive Hypertrophic Cardiomyopathy

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SUMMARY

Infective endocarditis (IE) is a relatively rare but serious complication of hypertrophic obstructive cardiomyopathy. Currently, antibiotic prophylaxis is not generally recommended in these patients. We report a case of infective endocarditis in a patient after alcohol septal ablation for hypertrophic obstructive cardiomyopathy with residual left ventricle outflow tract obstruction. To the best of our knowledge, this is the first case in the medical literature demonstrating this complication in the late postprocedural period following alcohol septal ablation. (Int Heart J 2008; 49: 371-375)

Key words: Infective endocarditis, Hypertrophic cardiomyopathy, Alcohol septal ablation

HYPERTROPHIC cardiomyopathy (HCM) is a genetically determined disorder that is defined by the presence of unexplained cardiac hypertrophy. Although the majority of the patients with HCM are asymptomatic, some are symptomatic (dyspnea, angina, syncope) or have increased risk of cardiac complications. Infective endocarditis (IE) is one of the recognized complications.1) Agreement exists on increased risk of infective endocarditis in HCM in the presence of left ventricular outflow tract (LVOT) obstruction.1) Highly symptomatic patients with obstructive HCM are considered candidates for surgical ventricular septal myectomy,2) alcohol septal ablation (ASA),3) or dual-chamber pacemaker implantation.4) Long-term results of ASA for obstructive HCM are consistent, attributing a number of favourable effects to ASA that generally match that of surgery, including gradual and progressive reduction in outflow pressure gradient over 3 to 12 months and alleviation of symptoms. Unfortunately, mild to moderate residual obstruction is common in 10-20% of these patients.3,5)
Antibiotic prophylaxis in HCM was recommended by the American Heart Association in the past. However, recent guidelines do not recommend antibiotic prophylaxis in patients with HCM, which mainly illustrates the lack of data on this topic.

In the present case report we describe infective endocarditis in a patient with residual LVOT gradient following ASA.

CASE REPORT

A 75-year old female was admitted to our hospital for heart failure in March 2006. Echocardiography revealed left ventricular hypertrophy with preserved systolic function (left ventricular ejection fraction was 80%), severe impairment of diastolic function, mild mitral regurgitation, and severe left ventricular outflow tract obstruction (maximal pressure gradient was 80 mmHg). Owing to comorbidities (gastrointestinal bleeding in her medical history, chronic obstructive pulmonary disease, depression), we decided to perform ASA. The procedure resulted in an acceptable hemodynamic result (residual pressure gradient was 10 mmHg) and improvement of clinical symptoms (NYHA class IV to II). The

Figure 1. Echocardiographic parasternal long-axis view (arrows display vegetation on mitral valve and left ventricle outflow tract)
patient was clinically stable during a 6-month follow-up, but echocardiography showed a gradual increase of the outflow tract obstruction up to 50 mmHg.

In February 2007 the patient was admitted again for progressive dyspnea with a history of untreated cough lasting for two months and subfebrile body temperature. Bedside transthoracic echocardiography showed left ventricular hypertrophy with preserved systolic function (left ventricular ejection fraction was 70%), severe impairment of diastolic function, mild to moderate mitral regurgitation, suspicious vegetations on the mitral valve and in the left ventricular outflow tract (LVOT) (Figure 1), and LVOT obstruction with a pressure gradient of 50 mmHg. Laboratory tests revealed leucocytosis ($44 \times 10^9 /L$), renal insufficiency (creatinine 323 µmol/L), and elevated inflammatory markers (C-reactive protein 299 mg/L). Antibiotic therapy was commenced (ampicillin, gentamycin), but the status of the patient rapidly deteriorated. Despite intensive medical care (mechanical pulmonary ventilation, catecholamines), she died of septic shock 4 hours after admission. Autopsy confirmed a diagnosis of sepsis and infective endocarditis in both the mitral valve (Figure 2) and upper interventricular septum (Figure 3).

Figure 2. Histologic appearance of vegetation of infective endocarditis with extensive acute inflammatory cells and fibrin on mitral valve (haematoxylin-eosin, magnification 100x)
DISCUSSION

The present case illustrates the seriousness of infective endocarditis in obstructive HCM. Unlike the cases published to date, the patient was treated for the obstruction by ASA one year before this episode with a suboptimal result (residual obstruction in the follow-up). The presence of an infective site in the upper interventricular septum is also unique. Studies examining patients with infective endocarditis in HCM found vegetations most commonly on the left ventricular aspect of the anterior mitral valve leaflet, presumably caused by mitral-septal contact during systole.\(^8\) A hypothesis relating the infective focus in LVOT to ASA was considered. However, the infective focus appeared outside of the scar tissue by ASA (Figure 3), and the long period of time between the procedure and infective endocarditis provided further evidence against this. Her clinical history suggested that infective endocarditis was secondary to infective exacerbation of chronic obstructive pulmonary disease.

Infective endocarditis complicating HCM is not commonly reported. It occurs predominantly in patients showing evidence of LVOT obstruction. Spirito, \textit{et al} followed 810 patients for 7 years and reported an infective endocarditis incidence of 1.4 per 1000 persons/year. The main risk factors were the presence of LVOT obstruction (\(>\) 50 mmHg) and left atrial dilation.\(^1\) The risk of infective endocarditis in HCM is moderate according to previous guidelines.\(^6\) The ration-
ale for prophylaxis was based largely on expert opinion and what seemed to be a rational and prudent attempt to prevent a life-threatening infection. Recent guidelines recommend antibiotic prophylaxis only for patients at high risk (prosthetic cardiac valve or prosthetic material used for valve repair, previous IE, some patients with congenital heart disease, and transplanted heart). Although we agree with the recent recommendation on the prevention of infective endocarditis, there is an ongoing debate about the prophylaxis and the strategy of antibiotic treatment in patients with HCM with outflow tract obstruction. In our opinion, antibiotic prophylaxis is not generally recommended in the prevention for dental, respiratory, gastrointestinal, and urogenital tract invasive procedures; on the other hand, our case gives evidence that well-timed antibiotic treatment of other infective diseases is strongly advised.

**Conclusion:** This case suggests an enhanced risk of infective endocarditis in HCM with obstruction not only in the natural history of the disease, but also after ASA with residual outflow tract gradient. The relation between scar caused by ASA and the IE is not well described. The reduction of LVOT obstruction by ASA may reduce the risk for IE, but residual obstruction and the interventricular septal tissue changes by ASA may increase the risk. Careful management of the infection should be considered in these patients.

**REFERENCES**