ACUTE MYOCARDIAL INFARCTION IN AN ELDERLY PATIENT WITH SEVERE AORTIC STENOSIS AND ANGIographically NORMAL CORONARY ARTERIES

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SUMMARY

Aortic stenosis (AS) is common in the elderly and is associated with an increased risk of death from cardiovascular events. Nevertheless, acute myocardial infarction (AMI) in patients with severe AS and “normal” coronary arteries is very rare. We present an elderly male with severe AS and angiographically normal coronary arteries who experienced AMI. Platelet hyperaggregability, activation of blood coagulation, coronary microcirculatory dysfunction, imbalance of supply and demand in the hypertrophied myocardium, and subendocardial ischemia predisposed by AS are possible mechanisms. The relevant literature is reviewed and discussed in the report. [International Journal of Gerontology 2010; 4(3): 157–160]

Key Words: aortic stenosis, acute myocardial infarction

Introduction

Previous studies demonstrated that older persons with aortic stenosis (AS) have a higher prevalence of coronary risk factors, a higher prevalence of coronary artery disease, and a higher incidence of new coronary events1–3. Nevertheless, acute myocardial infarction (AMI) in patients with severe AS and “normal” coronary arteries is very rare. We present a 79-year-old male with severe AS and left ventricular hypertrophy who suffered an AMI. Coronary angiography excluded coronary obstruction as the cause of the AMI in this patient. Platelet hyperaggregability, activation of blood coagulation, coronary microcirculatory dysfunction, imbalance of oxygen supply and demand in the hypertrophied myocardium, and subendocardial ischemia predisposed by AS are possible mechanisms to explain this serious event.

Case Report

A 79-year-old male with a medical history of hypertension and chronic renal insufficiency presented to our emergency department with complaints of chest discomfort and heaviness for 3 hours. Two months prior to visiting our emergency department, he had been admitted to our ward of cardiology for 1 week because of angina pectoris and heart failure. Echocardiography showed severe AS with estimated valve area 0.8 cm² and low left ventricular ejection fraction. Aortic valve replacement with preoperative coronary angiography was suggested but the patient refused. He was then discharged and received regular outpatient follow-up. Antihypertensive medications and aspirin were prescribed. On arrival at our emergency department, his electrocardiogram revealed regular sinus rhythm with ST-segment depression in lead II, III, aVF and V4-6, which was the same as that from his previous visit. Initial biochemical data of cardiac enzymes were creatine kinase (CK), 224 U/L; CK-MB, 13.6 U/L; troponin-I, 4.96 ng/mL. He was admitted to our intensive care unit under the impression of non-ST segment elevation...
myocardial infarction. Intravenous infusion of nitroglycerin and subcutaneous injection of low molecular weight heparin were prescribed in the intensive care unit. Aspirin and clopidogrel were also prescribed. Coronary angiography was performed by an interventional cardiologist 18 hours after onset of the symptoms and revealed normal coronary arteries with nonsignificant obstructive lesions (Figure 1). The peak cardiac enzymes recorded at 20 hours after appearance of the symptoms were CK, 516 U/L; CK-MB, 30.8 U/L; troponin-I, 7.80 ng/mL. After medical treatment, he was discharged 6 days later with no further event.

Discussion

In developed countries, AS is the most prevalent of all valvular heart diseases. A clear increase in the prevalence of AS is seen with age: 1.3% in patients aged 65–75 years, 2.4% in those aged 75–85 years, and 4% in those older than 85 years. Previous studies indicated that AS, even in its early phases, may be associated with the pathogenesis of acute coronary syndromes. About 20–50% of subjects with AS and angina pectoris have significantly obstructive coronary artery disease (CAD). Although most patients with AS are of an age at which CAD is prevalent, AMI with severe AS and “normal” coronary arteries is rarely reported. On the other hand, the overall rate of AMI in patients with normal coronary angiography ranges from 1% to 12%. Disparate etiologic factors have been implicated, including transient occlusion of the infarct-related artery owing to platelet hyperactivity and thrombosis, coronary artery spasm, cocaine abuse, acute alcohol intoxication, ingestion of ephedrine-containing drugs, withdrawal from calcium channel blockers, and coronary embolization. Patients in this group appear to be younger and less likely to have traditional risk factors for atherosclerosis than those with documented coronary disease. It is of interest that, in this report, the 79-year-old male with severe AS and common CAD risks suffered from an AMI despite angiographically normal coronary arteries. Mechanisms other than obstructive CAD predisposed by severe AS perhaps play an important role in such events.

Chirkov et al. reported that AS was associated with platelet hyperaggregability, irrespective of the presence and/or absence of CAD. Thrombin generation and platelet activation were enhanced in patients with AS, and a maximal transvalvular pressure gradient was the only independent predictor of these hemostatic processes. All three platelet activation markers, i.e., soluble CD40 ligand, P-selectin, and beta-thromboglobulin were higher in AS patients than in healthy individuals (more than 2-fold increase in solubleCD40 ligand and P-selectin). Also, levels of all the hemostatic variables were higher in AS patients than in patients with hypertrophic obstructive cardiomyopathy and sinus rhythm, indicating that higher transvalvular pressure gradient is a much more potent trigger for blood clotting. The mechanism underlying thrombin generation and platelet activation in the AS patients appeared to be closely linked to altered hemodynamic properties of blood flow in the presence of severe aortic valve stenosis. However, it is still uncertain that antiplatelet or anticoagulant therapy are effective in preventing...
thrombin generation and platelet activation predisposed by AS.

In addition to thrombin generation and platelet activation, left ventricular hypertrophy predisposed by AS can lead to microcirculatory dysfunction, even when the coronary arteries are normal. In a recent study reported by Miyagawa et al., who used myocardial contrast echocardiography to evaluate the deterioration of blood flow in the hypertrophied myocardium of AS patients, the myocardial blood flow in the subendocardium was significantly lower than that in normal individuals, whereas there was no significant difference in the subepicardial blood flow. Decreased subendocardial blood flow implies myocardial microcirculatory dysfunction and reduced coronary vasodilator reserve. This impairment could be secondary to perimyocytic fibrosis and reduced capillary ingrowth into the hypertrophied myocardium. Additionally, the increased filling pressure needed to distend the thickened ventricular wall compresses the endocardium, further impairing blood flow to that layer of the myocardium. These abnormalities may contribute to the induction of subendocardial ischemia in AS patients who have normal epicardial coronary arteries. According to the report by Miyagawa et al., the reduced subendocardial blood flow and microcirculatory dysfunction recovered significantly at 2 weeks after aortic valve replacement and this improvement was still present at a 1-year follow-up. Although aortic valve replacement is indicated in symptomatically severe AS, it remains unclear whether it can reduce the rate of AMI in patients with severe AS.

In our report, a 79-year-old male with a history of symptomatically severe AS and hypertension suffered from AMI with typical ischemic angina and elevation of cardiac enzymes. Coronary angiography was performed and unexpectedly revealed normal coronary arteries. It reminded us that AMI can occur in AS patients by means of altered hemostatic and hemodynamic properties even without obstructive CAD. To our knowledge, this is the first case report in Taiwan of AMI occurring in a patient with severe AS and angiographically normal coronary arteries.

In conclusion, thrombin generation and platelet activation can be enhanced by AS despite antiplatelet or anticoagulant therapy. Left ventricular hypertrophy predisposed by AS can lead to decreased blood flow in the subendocardium and impaired coronary vasodilatory reserve. A high filling pressure produced by the hypertrophied myocardium compresses the endocardium and contributes to subendocardial ischemia. These abnormalities are possible mechanisms to explain AMI occurring in elderly patients who have severe AS and common CAD risks but normal coronary arteries. The effect of aortic valve replacement in the prevention of this serious event requires further investigation.

References

