Asymptomatic Aortic Dissection Late After Aortic Valve Replacement

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ABSTRACT

Aortic dissection (AD) in patient with prostetic aortic valve is a rare but potentially fatal complication. Predictors of the occurrence of AD after aortic valve replacement (AVR) include fragility and thinning of the ascending aorta, aortic dilatation, aortic regurgitation (AR) and high blood pressure before AVR operation. AD is usually symptomatic, but rarely asymptomatic.

We presented a case of asymptomatic AD seen in routine echocardiographic examination at 15 years after the AVR surgery.

INTRODUCTION

Dissection of the ascending aorta has a high mortality rate of 1% to 2% per hour for the first 24 to 48 hours (1). Congenital and acquired factors, alone or in combination, can lead to AD. AD is more common in patients with hypertension, connective tissue disorders, congenital aortic stenosis, or a bicuspid aortic valve, as well as in those with first-degree relatives with a history of thoracic dissection. These diseases affect the media of the aorta and predispose it to dissection (2). AD is usually symptomatic, but rarely asymptomatic (3). We presented a case of asymptomatic AD seen in routine echocardiographic examination at 15 years after the AVR surgery.
CASE PRESENTATION

A 76-year-old male patient who had undergone a mechanical prosthetic AVR due to severe AR caused by dilatation of the ascending aorta because of aortoannular ectasia fifteen years ago was admitted to our clinic for measurement of routine coagulation parameters. He had no active symptoms. On physical examination, the pulse was irregular and heart rate was 86 beats/min, blood pressure was 130/80 mmHg and respiratory rate was 18 breaths/min. There was no jugular venous distension or hepatojugular reflux. His cardiac examination revealed a systolic 2/6 murmur loudest at the left sternal border and click sounds from the mechanic prosthetic valves. Peripheral pulses were symmetrical and palpable. Examination of the other systems was normal.

The patient was investigated for the risk factors of AD. He had hypertension controlled with drug treatment. He had no chest, back or abdominal pain at rest or exertion. The patient did not complain of diaphoresis, syncope, dizziness episodes, dyspnea or orthopnea. There were no symptoms suggestive of stroke and compression of large vessels and other structures within the chest. The electrocardiogram showed a rhythm with atrial fibrillation and there was no sign of myocardial ischemia.

The chest x-ray showed an enlargement of the ascending aorta and mediastinum (Figure 1). Transthoracic echocardiography (TTE) showed the aortic root diameter was 37 mm at level of sinus Valsalva and a giant ascending aortic aneurysm measuring 88 mm of maximum diameter and intimal flap suspicion after the sinotubular junction and mild intraprothetic AR. Opening of the aortic valve prosthesis was normal and no significant gradient was measured. The left and right ventricles were within normal size and function and concentric left ventricular hypertrophy was detected. The transesophageal echocardiography (TEE) revealed AD starting from the ascending aorta with an intimal flap arising 2.5 cm above the aortic valve prosthesis (Figure 2). The thoracoabdominal computed tomography confirmed double barreled aorta, intimal flap extending from ascending aorta to the level of the common iliac arteries with thrombosis at the false lumen (Figure 3). The patient was referred to the surgical department for operation but the patient denied surgical treatment.

DISCUSSION

AD may be observed in patients who underwent prior aortic valve replacement (4). AR, aortic size, bicuspid aortic valve, cystic medial necrosis, fragility of the aortic wall and systemic hypertension are some of the risk factors for late dissection of the ascending aorta (5,6). The aortic diameter at AVR is an independent predictor of dissection. Thus, in patients with moderate aortic dilation at AVR, additional factors play a role in the subsequent development of late AD (7,8). The aortic size and previous AVR have been supposed as predictors for late ascending AD in our case.

The mechanism which revealed an area of condensed resistance within the aortic wall
may have been the jet lesion which produced the post stenotic ascending aortic dilatation similar to aortic wall disease in aortic stenosis and regurgitation (9,10).

The interval between valve replacement and dissection varies greatly (9,10). The time interval varies from 2 months to 17 years in the cases studied. For example, in the report of Gooch AS the interval was 13 years, Nancarrow PA 12 years, Albat B 14 and 16 years and in case series of Modi A this time was 17 years (8,11).

AD is usually symptomatic and according to a report on 464 patients from the International Registry of Acute AD, 95% of patients reported pain, and 85% reported an abrupt onset (12). However, there is no typical pain in 5% to 15% of patients with AD (13). Our patient did not complain of pain. Several potential explanations have been offered as reasons for the absence of pain, but the exact mechanism is unknown. Slow or gradual dissection with less wall stretching may not cause pain and sparing of the adventitial layer, the site of aortic innervation may result as painless dissection (14).

TTE is a good noninvasive, fast and an easy method. TTE is convenient to start diagnostic evaluation with asymptomatic patients. Diagnosis of AD by TEE, helical CT and magnetic resonance imaging have similar and reliable diagnostic value. These tests are usually performed after TTE (15).

As a result, the frequency of asymptomatic aortic dissection after aortic valve replacement is extremely rare but very serious and fatal situation. Therefore, in particular patients with predisposing factors such as dilatation of the ascending aorta and systemic hypertension should be followed by transthoracic echocardiography with regular control visits.

**REFERENCES**