

An Echocardiogram Image of Massively Dilated Aortic Root and Ascending Aortic Aneurysm Secondary to Giant Cell Arteritis

Dear Editor,

A 77-year-old Indian man who was an ex-smoker has a medical history of hypertension, hyperlipidaemia, previous cerebral lacunar infarct and temporal (giant cell) arteritis which was complicated by optic neuritis. The giant cell arteritis was diagnosed 12 years ago. He has received chronic steroid treatment in the past. He presented with congestive heart failure in the current hospital admission.

Case Report

Clinical examination showed collapsing pulse at rate of 95 per minute. Bilateral basal crepitations were heard on chest auscultation. The jugular venous pressure was raised. Auscultation of the heart revealed a loud, grade 4/6 early diastolic murmur. The cardiac apex was displaced to the 6th intercostal space.

Electrocardiogram (ECG) showed left ventricular hypertrophy and sinus tachycardia.

Chest X-ray (CXR) showed cardiomegaly and moderate pulmonary congestion. The essential laboratory parameters are shown in Table 1.

Echocardiogram showed severe aortic regurgitation with massively dilated aortic root and ascending aorta with a diameter of 92 mm (Fig. 1). The left ventricular ejection fraction was preserved at 50%. The left ventricle was dilated with left ventricular end systolic diameter of 45 mm and end diastolic diameter of 62 mm. The left atrium was dilated at 45 mm. The pulmonary artery systolic pressure was



Fig. 1. Parasternal long axis view of transthoracic echocardiogram showing the massively dilated aortic root and ascending aorta.

moderately raised at 54 mmHg. The aortic regurgitation was secondary to dilated aortic root and malcoaptation of aortic valves from aortic annular dilatation.

The patient has declined coronary angiogram and aortic surgery and opted for medical therapy. He was treated with digoxin 125 mcg, amlodipine 5 mg om, bisoprolol 5 mg om, frusemide 40 mg bd, potassium chloride 600 mg bd and simvastatin 40 mg on. He and his family members were informed of the very high-risk of aortic rupture and sudden death.

Discussion

Mildly dilated aortic root and ascending aorta are common findings during echocardiography examination of older patients. Older age, hypertension, smoking and aortic atherosclerosis are the common predisposing risk factors for the development of aortic aneurysm. Combination of these risk factors can lead to gradual development of aortic dilatation and formation of aortic aneurysm. Hence, medical therapies involving antihypertensive and lipid lowering treatment can reduce the risk. However, a massively dilated aortic aneurysm over a size of 90 mm as seen in our patient would rarely be caused by the traditional risk factors alone.

Table 1. Essential Laboratory Parameters

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|---|--------------|
| N-terminal prohormone of brain natriuretic peptide (NT Pro BNP) | 2271 pg/mL |
| Creatinine | 94 mmol/dL |
| Haemoglobin | 13 g/dL |
| Low density lipoprotein (LDL) | 2.2 mmol/L |
| High density lipoprotein (HDL) | 1.74 mmol/dL |
| Erythrocyte sedimentation rate (ESR) | 9 mm/Hr |
| C-reactive protein (CRP) | 1.1 mg/L |
| Anti-cardiolipin IgM and IgG | negative |
| Lupus anticoagulant | absent |
| Anti-nuclear antibody and anti double stranded DNA antibody | negative |
| Hepatitis B surface antigen | non-reactive |
| Anti-hepatitis C virus | non reactive |

The most significant contributing factor for the development of aortic aneurysm in our patient was systemic arteritis, essentially giant cell arteritis which can also be called temporal arteritis. Giant cell arteritis is an immunological systemic inflammation disorder commonly affecting the temporal arteries and ophthalmologic system. Rarely, it can also cause aortitis and results in massive aortic dilatation and formation of aortic aneurysm. The causes of the massive aortic aneurysm in our patient were likely to be multi-factorial. His risk factors include smoking, hypertension, atherosclerosis, male gender, older age and giant cell arteritis. Aortic aneurysms are known to be more common in men than women and in patients with chronic lung disease secondary to smoking.

Patients with aortic aneurysms are often asymptomatic until the aneurysm expands. The most common presenting symptom is pain. Pain can be acute, implying impending rupture or dissection, or chronic, from compression or distension. The location of pain may indicate the area of aortic involvement. Ascending aortic aneurysms tend to cause anterior chest pain, aortic arch aneurysms tend to cause pain radiating to the neck and descending aortic aneurysms tend to cause pain radiating to the back between the scapulae.

The most common and feared complications are acute rupture and dissection which normally result in sudden death. The patients should undergo surgical repair on emergent basis.

According to the natural history of aortic aneurysm, surgical repair for ascending aorta aneurysms at 5.5 cm should be offered to patients without any familial disorders such as Marfan syndrome. The recommendation is based on the incidence that rupture and dissection could increase exponentially once the size of the ascending aorta reaches 6 cm (31% risk of complications) and 7 cm (43% risk) respectively. It is important to note that a lower threshold of 5 cm has been used for consideration of prophylactic aortic root replacement in patients with Marfan syndrome when annulo-aortic ectasia is the cause of aneurysm.⁴

The joint task force from the American College of Cardiology Foundation and the American Heart Association produced guidelines for the surgical management of this disease as below.

- Aortic size
- Ascending aortic diameter ≥ 5.5 cm or twice the diameter of the normal contiguous aorta
- Descending aortic diameter ≥ 6.5 cm
- Subtract 0.5 cm from the cutoff measurement in the presence of Marfan syndrome, family history of aneurysm or connective tissue disorder, bicuspid aortic valve, aortic stenosis, dissection, patient

undergoing another cardiac operation

- Growth rate ≥ 1 cm per year

Conclusion

We presented a rare case of massive aortic root aneurysm attributable to giant cell arteritis.

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