Aortic valve disease on computed tomography examination of the chest: a missed opportunity

Introduction

Aortic valve thickening or stenosis is a highly prevalent heart valve lesion seen in 2–7% of adults over 65 years (Freeman and Otto, 2005). The diagnosis can be overlooked in the acutely ill patient because the signs may be masked and difficult to detect, and yet untreated, when severe, it carries a very grave prognosis (Horstkotte and Loogen, 1988). Even in its milder forms aortic stenosis requires expert evaluation including regular surveillance and follow up as the rate of progression to clinical disease is variable. The risks of aortic valve surgery increase as left ventricular performance deteriorates and therefore timely diagnosis is essential. A feature of both early and advanced aortic valve disease is increasing calcification of the valve leaflets and annulus. This can only sometimes be detected on plain radiography but is readily seen on any form of computed tomography scanning of the chest.

This article presents two cases where the clinical diagnosis of aortic stenosis was initially not considered and in which computed tomography scanning of the chest had been performed for other indications. The failure to comment on the aortic calcification in both cases resulted in a significant and unnecessary delay in diagnosing aortic stenosis.

Discussion

Aortic stenosis is a leading causes of cardiovascular disease, the prevalence of which is increasing as the population ages (Nkomo et al, 2006). The spectrum ranges from mild aortic valve thickening without obstruction to the left ventricle outflow, termed aortic valve sclerosis, to severe aortic valve obstruction requiring urgent surgical intervention. Severity of valve obstruction is the strongest predictor of clinical outcome.

Even without obstruction aortic sclerosis doubles the risk of cardiovascular death (Otto et al, 1999). Patients with mild aortic valve disease show around 10–22% risk of progression to moderate or severe aortic stenosis within 8 years (Faggiano et al, 2003). Aortic valve calcification is a major component of aortic stenosis formation, appearing early in valve lesions and accumulating through active and highly regulated mechanisms similar to ossification (Rajamannan et al, 2003). Presence of aortic valve calcification is a marker of cardiovascular morbidity or mortality and a high aortic valve calcification score in severe aortic stenosis negatively affects clinical outcome (Rosenhek et al, 2000).

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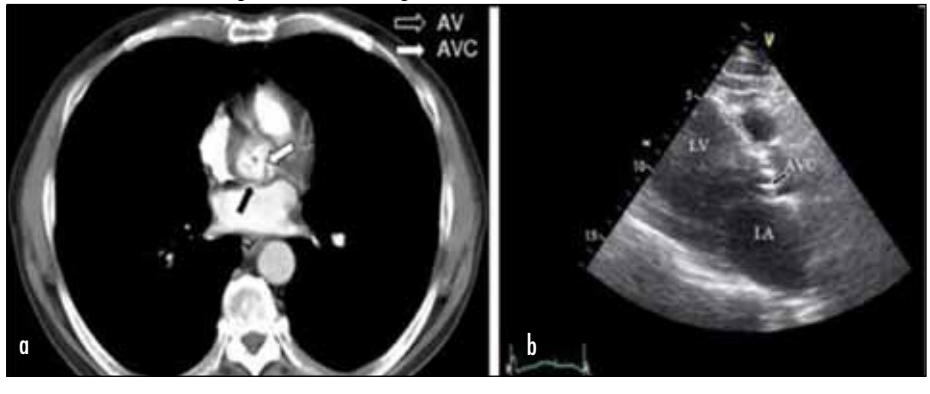
Case Report 1

A 62-year-old Caucasian man with a history of type 2 diabetes and chronic obstructive pulmonary disease who smoked regularly underwent work up for abdominal discomfort under the surgical team. As part of the general work up, a computed tomography scan of chest and abdomen was performed, and was reported as showing mild cardiomegaly but no other significant abnormality. He was discharged clinically improving but without a formal diagnosis. Six months later, he re-presented with breathlessness and significantly reduced exercise tolerance. There was no history of cough, chest pain, palpitations, dizziness or syncope. Clinical examination revealed a regular pulse, supine blood pressure 102/68 mmHg, oxygen saturation 98% on air, the jugular venous pressure was raised and the apex beat displaced, a soft ejection systolic murmur at the right upper sternal edge with no radiation to carotids and the second heart sound was soft. The electrocardiogram showed sinus tachycardia 110/min. Full blood count and serum urea, creatinine and electrolyte levels were also within normal limits.

A transthoracic echocardiogram was organized which demonstrated heavily thickened, calcified, severely restricted aortic valve with poor left ventricle systolic contraction (ejection fraction 17%). Peak velocity across the aortic valve was 3.83 m/sec, the mean gradient was 39 mmHg and the calculated aortic valve area was 0.4 cm^2. The clinical diagnosis of low flow aortic stenosis was made based on the florid morphology of the valve. After coronary angiography which demonstrated significant coronary artery disease, he underwent emergency aortic valve replacement and coronary artery bypass operation successfully. Retrospective review of the computed tomography scan performed 6 months earlier clearly showed grade III aortic valve calcification (Koos et al, 2006) (*Figure 1*) which had not been noted in the report.

Figure 1. a. Computed tomography chest with contrast without appropriate windowing to bone level. b. Transthoracic echocardiogram demonstrating aortic stenosis.



Electron beam computed tomography or conventional multislice computed tomography enable non-invasive, objective definition of calcification and quantification of calcification load (Feuchtner et al, 2006). Because of the clear difference in Hounsfield unit score between calcification and other structures even with intravascular contrast media, differentiation of aortic valve calcification from other structures is relatively easy on any computed tomography scan with appropriate windowing. The extent of aortic valve calcification is linked to the severity of aortic stenosis and provides additional prognostic information (Messika-Zeitoun et al, 2004). In a retrospective study of 402 patients, Koos et al (2006) found 18% of patients had incidental aortic valve calcification on routine computed tomography scans performed for non-cardiac reasons. The grade of aortic valve calcification correlated well with the haemodynamic severity of aortic valve disease as determined by echocardiography.

The two cases reported here demonstrate situations where although aortic valve calcification was apparent on the computed tomography chest, the finding was not reported and therefore a crucial opportunity to diagnose aortic stenosis was missed. In both cases the correct diagnosis was subsequently made; in case two this took only 5 days but in case one it took 6 months. In case one the aortic stenosis murmur was very soft because the left ventricular function had deteriorated significantly making the clinical diagnosis difficult. The computed tomography findings could have been crucial in alerting clinicians to the possibility of aortic stenosis as the diagnosis and allowing surgery before the onset of left ventricular dysfunction, thus allowing an operation at significantly lower risk.

Conclusions

Aortic valve calcification seen on computed tomography scan is a common finding and relates directly to the presence and severity of aortic valve disease. Although many patients with grade I and II aortic valve calcification will only have aortic sclerosis, this in itself results in an adverse prognosis and has a high risk of progression to clinically significant aortic stenosis.

The authors propose that it should be standard practice to report the presence and extent of aortic valve calcification with two objectives: to provide opportunistic screening for aortic valve disease – a common disease which may need long-term surveillance – and to help make the diagnosis in patients presenting acutely with aortic stenosis, particularly those with impaired left ventricle function where the murmur intensity may have reduced and are therefore more likely to be missed. **BJHM**

Faggiano P, Antonini-Canterin F, Erlicher A et al (2003) Progression of aortic valve sclerosis to aortic stenosis. *Am J Cardiol* **91:** 99–101

Feuchtner GM, Dichtl W, Friedrich GJ et al (2006) Multislice computed tomography for detection of patients with aortic valve stenosis and quantification of severity. *J Am Coll Cardiol* **47:** 1410–17

Freeman RV, Otto CM (2005) Spectrum of Calcific aortic valve disease: pathogenesis, disease progression, and treatment strategies. *Circulation* **111:** 3316–26

Horstkotte D, Loogen F (1988) The natural history of aortic valve stenosis. *Eur Heart J* **9:** 57–64

Koos R, Kohl HP, Mohlenbruch G et al (2006) Prevalence and clinical importance of aortic valve calcification detected incidentally on CT scans: comparison with echocardiography. *Radiology* **241**(1): 76–82

Messika-Zeitoun D, Aubry MC, Detaint D et al (2004) Evaluation and clinical implications of aortic valve calcification measured by electron-beam computed tomography. *Circulation* **110:** 356–62

Nkomo VT, Gardin JM, Skelton TN (2006) Burden of valvular heart diseases: a population-based study. *Lancet* **368:** 1005–11

Otto CM, Lind BK, Kitzman DW (1999) Association of aortic-valve sclerosis with cardiovascular mortality and morbidity in the elderly. *N Engl J Med* **341:** 142–7

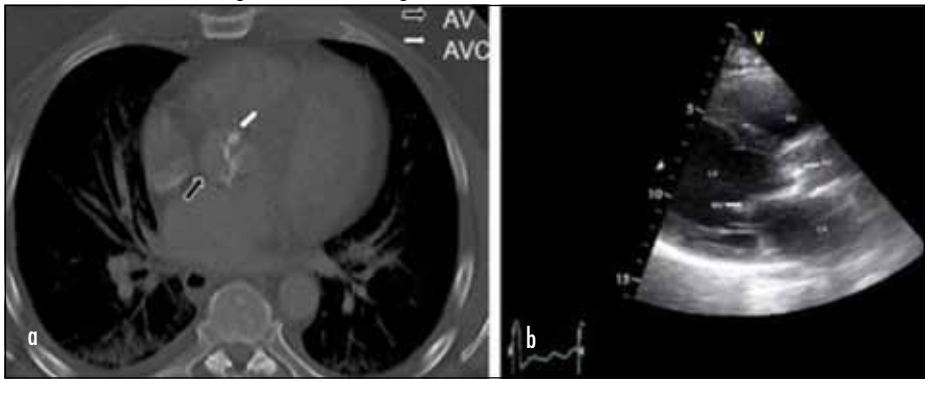
Rajamannan NM, Subramaniam M, Rickard D (2003) Human aortic valve calcification is associated with an osteoblast phenotype. *Circulation* **107:** 2181–4

Rosenhek R, Binder T, Porenta G (2000) Predictors of outcome in severe, asymptomatic aortic stenosis. *N Engl J Med* **343:** 611–17

Case Report 2

A 59-year-old Caucasian man presented with a 2-week history of breathlessness and feeling intermittently faint. He had longstanding hypertension treated with an angiotensin-receptor blocker (losartan) and calcium-channel blocker (amlodipine), suffered with obesity and was treated for nocturnal sleep apnoea with non-invasive ventilation. On examination, his body mass index was 41.5 kg/m^2, vital signs fell within normal limits, but a grade III ejection systolic murmur with soft second heart sound was noted. Full blood count and serum urea, creatinine and electrolyte levels were within normal limits. An elevated d-dimer level raised the possibility of a pulmonary embolism but a computed tomography pulmonary angiogram excluded the diagnosis. He was treated with antibiotics for a presumed community-acquired chest infection but a non-emergency transthoracic echocardiogram was ordered in view of the murmur. Transthoracic echocardiogram showed aortic valve peak gradient of 73 mmHg, mean gradient 48 mmHg, and aortic valve area of less than 1 cm^2. He was stabilized and underwent successful aortic valve replacement. Retrospective review of the computed tomography scan demonstrated grade III aortic valve calcification which was not mentioned in the report (*Figure 2*).

Figure 2. a. Computed tomography chest with contrast with appropriate windowing to bone level. b. Transthoracic echocardiogram demonstrating aortic stenosis.



LEARNING POINTS

- Aortic valve calcification is clearly seen in computed tomography of the chest.
- Aortic valve calcification should be mentioned in reports on computed tomography scans of the chest.
- Computed tomography of the chest can be one of the key screening investigations for aortic stenosis, which is highly prevalent and treatable.