

Aortic Valve Calcification Induced Mitral Stenosis

Tony Bechara*

Cardiology and Echocardiography Departments, Notre Dame des Secours University Hospital, Byblos - Lebanon and Central Military Hospital, Beirut, Lebanon

***Corresponding Author:** Tony Bechara, Cardiology and Echocardiography Departments, Notre Dame des Secours University Hospital, Byblos - Lebanon and Central Military Hospital, Beirut, Lebanon.

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Abstract

Aortic valve calcification is a very common disease, especially in the elderly population; in fact, valvular calcification can become severe enough to extend beyond the aortic valve, float inside the outflow tract of the left ventricle (LVOT), and even reach, although rarely, the anterior leaflet of the mitral valve and provoke its dysfunction. I aim by the presentation of this case report to reveal that one valve disease could be the cause of another valve dysfunction. In fact, the patient described in this article has a severe aortic valve calcification extending toward the anterior mitral valve which acts like a stopper, compromising its opening and inducing a mitral stenosis.

Keywords: *Aortic Calcification; LVOT Floating Calcification; Mitral Stenosis*

Learning Objective

This case report teaches us that severe aortic valve calcification can affect other nearby cardiac structures, and provoke, in addition to the initial aortic valve dysfunction, as in this case, a mitral valve stenosis.

Introduction

Aortic valve disease is the most common valve disease; it affects mostly the aging population; aortic sclerosis is seen in 29% of people of more than 65 years of age, with an annual progression rate of 1.7%/year to aortic stenosis, and an increasing incidence of myocardial infarction and cardiovascular death of more than 50% [1].

Mitral valve calcification may be seen in rheumatic mitral stenosis or as a result of degenerative changes in elderly patients. The latter are often associated with mitral valve annulus (MVA) calcification. Extensive degenerative calcification of the MVA may extend to the mitral valve leaflets (MVL), resulting in a variety of valvular diseases, including mitral valve stenosis [2].

The extension of aortic valvular calcification sometimes reaches the LVOT, seldom the mitral valve compromising its normal opening.

Case Presentation

An 85 year-old female patient, known to have hypertension and dyslipidemia (well treated), obese (BMI = 30), old smoker, presented for a new onset of mild lower leg edema; no other cardiovascular symptoms; his physical exam revealed a mild bilateral lower limb pitting edema, a right parasternal 2nd intercostal space systolic murmur (intensity of 4/6) in addition of a diastolic apical murmur (intensity of 3/6); lungs are clear and the physical exam of the abdomen is normal; no hepatojugular reflux.

Chest XR showed a mild cardiomegaly with clear lungs.

Laboratory tests are normal; an Echocardiography has been requested for cardiac evaluation.

Echocardiography

The cardiac ultrasound revealed a moderate aortic disease (mild aortic regurgitation associated with a moderate aortic stenosis (Figure 1)); LV is mildly hypertrophied with good overall contractility.

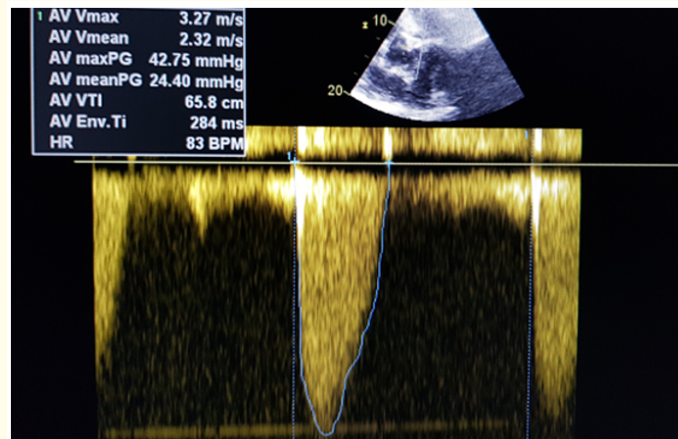


Figure 1: Transaortic flow showing a moderate aortic stenosis with a mean gradient of 24 mmHg.

The posterior mitral annulus is moderately calcified; no significant calcification of the mitral valve leaflets nor commissural fusion; one distal nodular calcification of a mitral chordae (Figure 3); however, the aortic valve calcification extends toward the LVOT, creating a rigid fixed rod like calcified tissue floating in the LVOT, approaches the anterior mitral leaflet (Figure 2), which, when it opens, becomes jammed at its middle third portion, blocking its full opening by acting like a stopper (Figure 3), causing a moderate mitral stenosis with a trace MR (mean transmitral pressure gradient = 5 mmHg; Figure 4).

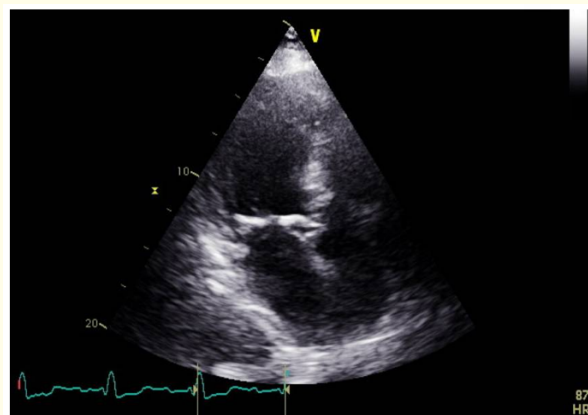


Figure 2: Aortic calcification tissue floating in the LVOT and approaching the anterior mitral leaflet.

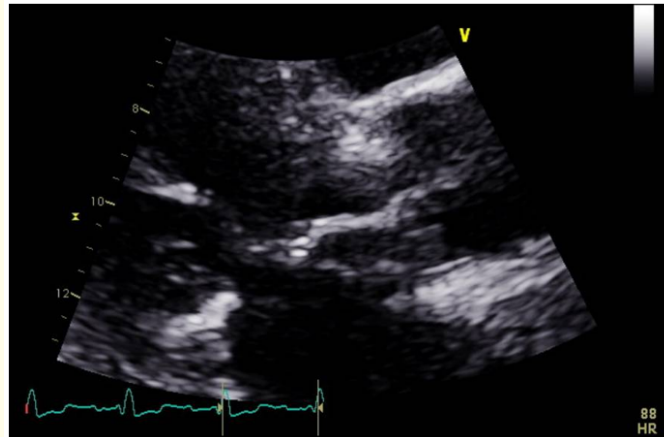


Figure 3: Extended aortic valvular calcification blocking the anterior mitral leaflet opening (like a stopper); notice also a nodular calcification of one mitral chordae.

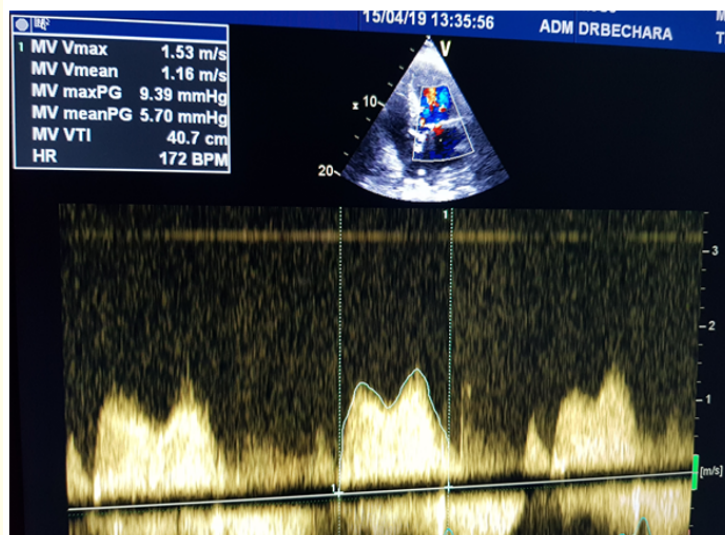


Figure 4: Transmittal flow showing a moderate mitral stenosis with a mean gradient of 5 mmHg.

No arterial pulmonary hypertension; the IVC is not dilated with good respiratory variability; no pericardial effusion nor constriction.

Discussion

Valvular calcification is an active process involving transformation of the valvular interstitial cells to osteoblast-like forming cells, which leads to the formation of microcalcific nodules, lipid deposition and infiltration of inflammatory cells and downregulation of local and circulatory inhibitors of calcification and neoangiogenesis [1].

The LVOT is analyzed for the presence, amount, and location of calcification. The distribution of calcification and extent can be assessed in a semiquantitative fashion as follows: mild, 1 nodule of calcium extending < 5 mm in any dimension and covering < 10% of the perimeter of the LVOT; moderate, 2 nodules of calcification or 1 extending > 5 mm in any direction or covering > 10% of the perimeter of the LVOT; severe, multiple nodules of calcification of single focus extending > 1 cm in length (which applies to this patient) or covering >20% of the perimeter of the LVOT [3].

Mitral annular calcification and stenotic or non-stenotic aortic valve calcification have a high incidence of atherosclerotic risk factors, suggesting they should be considered as manifestations of generalised atherosclerosis [4].

Mitral stenosis is a narrowing of the mitral valve orifice. The most common cause of mitral stenosis is rheumatic fever. Uncommon causes of mitral stenosis are calcification of the mitral valve leaflets and congenital heart disease. Other causes of mitral stenosis include infective endocarditis, mitral annular calcification, endomyocardial fibroelastosis, malignant carcinoid syndrome, systemic lupus erythematosus, Whipple disease, Fabry disease, and rheumatoid arthritis [5].

In degenerative mitral disease, mitral valve calcification is a pertinent indicator of mitral sclerosis or stenosis, whereas mitral valve annular calcification is not suited to reliably determine the presence of mitral valve disease [2].

In this case report, although there is a moderate calcification of the mitral valve annulus, the mitral leaflets are not calcified and there is no mitral commissural fusion, disfavoring a rheumatic mitral disease, even though there is a nodular calcification of one mitral chordae; the cause of this moderate mitral stenosis seems to be most probably extrinsic to the valve itself; in fact, the aortic calcification floats in the LVOT toward the anterior mitral leaflet and it acts like an anvil for which the moving mitral valve is the hammer; this creates a stopper for the anterior mitral leaflet opening; by reviewing the literature, I have not found any article describing a similar mechanism of mitral stenosis, which makes of this case a special and interesting one.

Conclusion

In conclusion, although degenerative mitral and aortic diseases can coexist and may share similar physiopathology, aortic valve calcification by itself can extend far enough so that calcified tissue can float inside the LVOT and approach the mitral valve inducing its dysfunction.

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