# Do all critical aortic stenosis with chest pain need aortic valve replacement? A case report

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## Abstract

Aortic valve replacement (AVR) remains the cornerstone of treatment for symptomatic critical aortic stenosis (AS). It is a Class I indication that symptomatic patients with critical AS undergo either surgical or transcatheter aortic valve replacement (TAVR). We present a patient with critical AS and new angina that was managed successfully with percutaneous coronary intervention (PCI) of the Right coronary artery. Physicians should consider that not all patients with critical AS and angina necessarily require AVR. Concomitant pathology leading to the symptoms should be carefully ruled out. This leads to a less invasive, cost effective care plan especially in patients with advanced age and comorbidities for which any type of surgical valvular intervention may pose high risk.

## Introduction

AS is the most common cause of left ventricular outflow obstruction in children and adults. It is most common type of valvular heart disease in Europe and North America, occurring in 2–7% of the population over 65 years of age.[1] Medical therapy alone is not effective for the long-term management of aortic valve disease, thus valve replacement remains the standard of care in patients with an acceptable risk profile. Symptomatic patients with critical AS are highly recommended to undergo surgical AVR or TAVR.[2,3] We present a case of critical AS with significant symptom manifested as chest pain that was managed successfully without surgical AVR or TAVR. We will also briefly review incidence, etiology, grading of AS and current guidelines for AVR.

## Case Report

A 91-year-old gentleman with history of hypertension, dyslipidemia and AS presented to our office on 7/2015 with complaints of new onset sub sternal burning pain of 6 weeks duration. This pain was worse with exertion and was relieved by rest. Patient would have pain on walking even around 100 yards. Prior to 6 weeks patient could walk 2 blocks on level ground without shortness of breath or chest pain. Patient denied any history of radiation or referral of pain. It was not related to breathing or positional changes. On examination, Blood pressure was 140/90 mm Hg and Pulse was regular at 60 beats per minute. Cardiovascular exam revealed a Grade 4/6 ejection systolic murmur best heard in the right second intercostal region radiating bilaterally to the neck. ECG revealed left ventricular hypertrophy (LVH) with non specific ST-T wave changes in inferior leads (Figure 1). Stress test was precluded due to critical AS.

Echocardiography (ECHO) done in May of 2014 had revealed critical AS with aortic valve area of 0.6-0.7 centimeter square and ejection fraction of 65 percent. The Aortic valve (AV) was heavily calcified. Aortic valve peak velocity (AV Vmax) 4.66 m/s (Figure 2). Notably, in 2014 patient was asymptomatic. In view of his new symptoms, the patient was directly referred for a cardiac catheterization to evaluate his coronary anatomy prior to AVR.

Complete heart catheterization was performed. Hemodynamic measurement revealed Left ventricle (LV) pressure 240/0 with left ventricular end diastolic pressure (LVEDP) of 10. Central Aortic pressure was 166/59. There was a 55 mm mean gradient across the aortic valve. Pulmonary artery wedge pressure (PAWP), mean of 24, Right atrium (RA) mean of 8, Right ventricle (RV) 60/10 with an Right ventricular end diastolic pressure (RVEDP) of 14, Pulmonary artery (PA) 60/17. The aortic valve area was calculated at 0.6 centimeter square, which was unchanged from ECHO done May1, 2014.

The left ventriculogram in the right anterior oblique (RAO) view revealed normal LV systolic motion and ejection fraction of 55-60 percent. Left main coronary artery revealed a common ostium-giving rise to the Left anterior descending (LAD) and the circumflex. Right coronary artery (RCA) revealed a 99 % ostial narrowing (Figure 3). LAD revealed a 60 % narrowing in its proximal segment and a 90% ostial diagonal narrowing (Figure 4). Circumflex coronary artery had a 90 % stenosis (Figure 5).

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After a detailed discussion involving the patient, his son, interventional cardiologist and the cardio thoracic surgeon, patient requested that only PCI of the RCA be considered, as the chest pain was of recent duration with no change in aortic valve finding. Thus, a successful PCI was done in the proximal RCA with a bare metal stent (Figure 6). Patient was recommended anti-platelet therapy only for 6 months.

Patient did extremely well after PCI of the RCA and the patient has remained asymptomatic to date. Thus, this new onset chest pain in patient with critical AS was due to concomitant coronary artery lesion and was amenable to stenting and thus spared this elderly patient from the aortic valve replacement which would have been a high risk surgery for him.

## Discussion

AS is the most common valvular heart condition in the developed world, affecting 3% of people between ages 75 and 85 and 4% of people over age 85. Congenitally unicuspid, bicuspid, tricuspid, or even quadricuspid valves may be the cause of AS.[4] In adults who develop symptoms from congenital AS, the problem is usually a bicuspid valve.[5] The main causes of acquired AS include degenerative calcification and, less commonly, rheumatic heart dis-

ease.[6,7] Other, infrequent causes of AS include obstructive vegetations, homozygous type II hypercholesterolemia, Paget disease, Fabry disease, ochronosis, and irradiation. Based upon a variety of hemodynamic and natural history data, clinicians generally grade the severity of stenosis as mild, moderate, severe, or critical. Grading of AS are as follows:[8] i) mild: valve area exceeds 1.5 $cm^2$; transvalvular velocity 2.0 to 2.9 m/s; mean gradient <20 mmHg; ii) moderate: valve area of 1.0 to 1.5 $cm^2$; transvalvular velocity 3.0 to 3.9 m/s; mean gradient 20 to 39 mmHg; iii) severe: valve area is less than 1.0 $cm^2$; transvalvular velocity ≥4 m/s; mean gradient ≥40 mmHg.

The term *critical* stenosis was defined based upon theoretical considerations showing that the aortic valve area must be reduced to one-fourth of its natural size before significant changes in circulation occur. As a result, since the triangular orifice area of the normal (adult) aortic valve is approximately 3.0 $cm^2$, an area exceeding 0.75 $cm^2$ would not be defined as critical. AVR and TAVR remain the only treatment proven to reduce the rates of mortality and morbidity in this condition.

Under current guidelines, the onset of symptoms of exertional angina, syncope and dyspnea in a patient who has severe AS is a class I indication for surgery.[9] High-gradient, severe AS that is asymptomatic often poses a dilemma.[7] The annual rate of sudden death in patients with this condition is estimated at 1% to 3% but the surgical mortality rate in AVR has been as high as 6%.[10-15] With improvements in surgical techniques and prostheses, mortality rates have been reduced to 2.42% making a case for earlier intervention.[16] TAVR has become widely available, but further investigation into its use in this patient cohort is warranted.[17] While assessing the cases of asymptomatic AS we have both traditional as well as novel markers at our disposal now. Left ven-

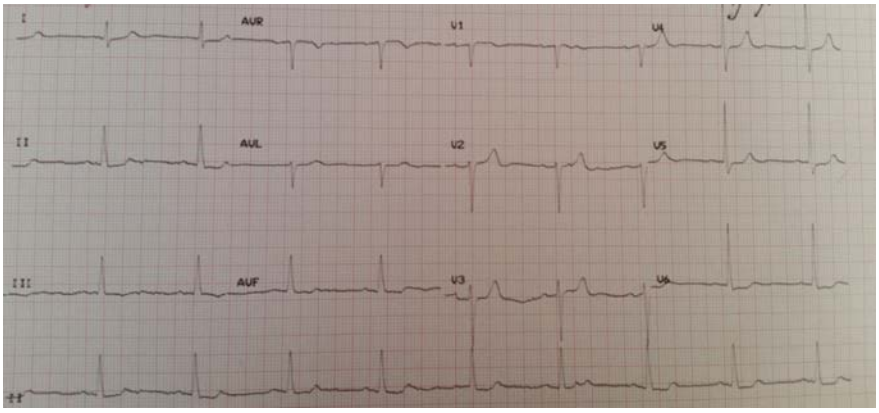


**Figure 1. Electrocardiogram.**

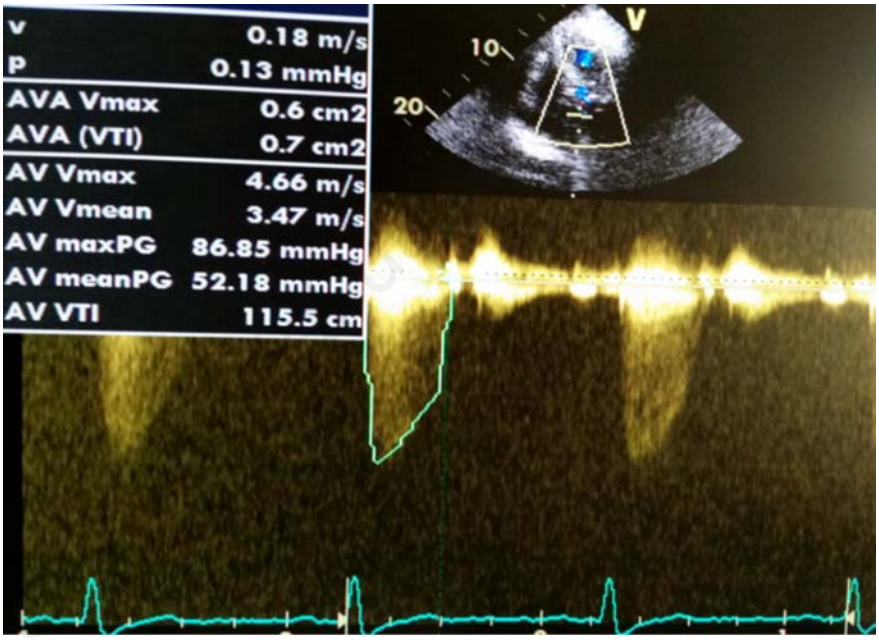


**Figure 2. Echocardiogram with peak velocities and calculated aortic valve area.**

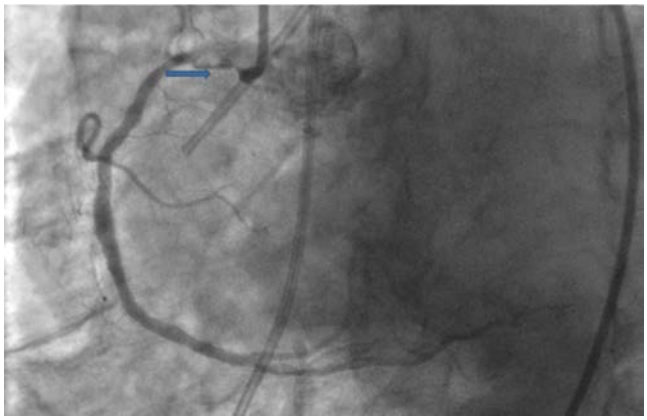

**Figure 3. Right coronary artery revealing a 99 percent ostial narrowing (shown by arrow).**

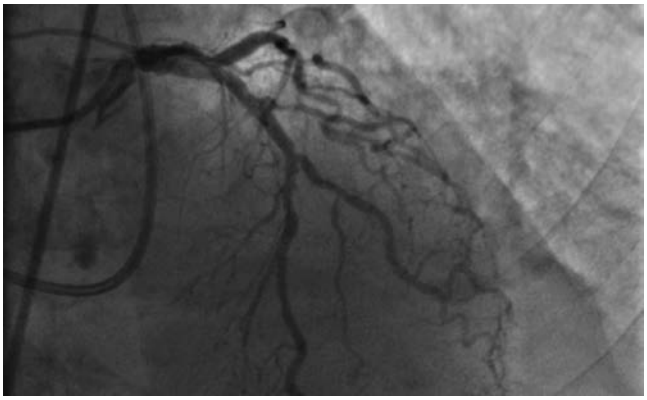

**Figure 4. Left anterior descending and left circumflex.**

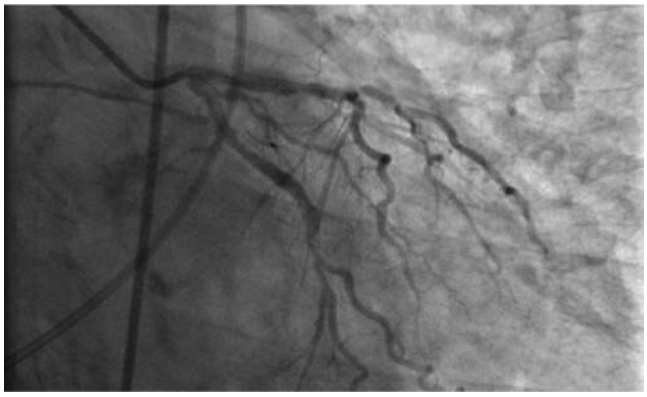

Figure 5. Circumflex coronary artery had a 90% stenosis.

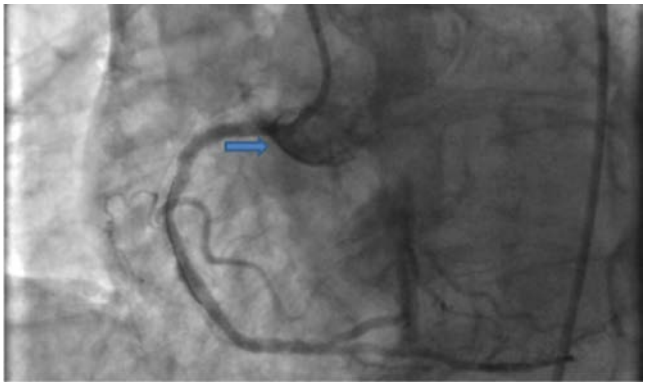

Figure 6. After percutaneous coronary intervention (shown by arrow).

tricular ejection fraction (LVEF) <50 percent, Peak aortic jet velocity >4.0 m/s, Valve area <1 $cm^2$ and Mean pressure gradient >40 mm Hg are the traditional markers to denote severe AS in asymptomatic patients. While, Indexed left atrial size >12.2 $cm^2/m^2$, LVH with wall thickness >15 mm, global left ventricular longitudinal strain <15.9, BNP (B-Natriuretic peptide) level >130 pg/mL and increase in mean pressure gradient of >20 mm Hg during exercise testing are the novel markers of asymptomatic severe AS. BNP level does not appear to be significantly associated with the degree of AS severity but does reflect heart failure status.[18]

The American College of Cardiology and American Heart Association (ACC/AHA) have issued the following recommendations for AVR, based on the severity of stenosis and on whether the patient has symptoms:[19-22] i) severe stenosis, with symptoms: class I recommendation (surgery should be done). Without surgery, these patients have a very poor prognosis, with an overall mortality rate of 75% at 3 years; ii) severe stenosis, no symptoms, in patients undergoing cardiac surgery for another indication (example coronary artery bypass grafting, ascending aortic surgery, or surgery on other valves): class I recommendation for concomitant aortic valve replacement; iii) moderate stenosis, no symptoms, in patients undergoing cardiac surgery for another indication: class IIa recommendation (*i.e.*, aortic valve replacement *is reasonable*); iv) very severe stenosis (aortic peak velocity >5.0 m/s or mean pressure gradient ≥60 mm Hg), no symptoms, and low risk of death during surgery: class IIa recommendation; v) severe stenosis, no symptoms, and an increase in transaortic velocity of 0.3 m/s or more per year on serial testing or in patients considered to be at high risk for rapid disease progression, such as elderly patients with severe calcification: class IIb recommendation (surgery *can be considered*).

On revisiting the above case description we realize that the patient did have critical AS but was asymptomatic. His chest pain was only due to concomitant coronary artery disease but was not due to AS *per se*. Age and comorbidity of the patient posed a high risk for Coronary artery bypass graft (CABG) with AVR. EuroSCORE II is not validated for patients above age 90. Besides, the patient's symptoms were of new onset and subsequent cardiac catheterization revealing critical RCA stenosis and the patient's preference for treating the cause of his recent symptoms, encouraged us to think otherwise. The decision to perform PCI alone with the belief that this chest pain and CAD would be amenable to the minimal risk procedure paid dividends. In addition, patient received a bare metal stent with the option of undergoing surgical AVR if symptoms were not relieved.

## Conclusions

Awareness amongst physicians about the fact that *all critical aortic stenosis with chest pain may not require aortic valve replacement* is important. This can lead to less invasive treatment tailored to the need of the patient especially in those with advanced age, significant comorbidities and an extremely high risk for CABG with AVR and can also result in decreased cost of care. Hence, careful history taking and physical examination is extremely important prior to intervention. Pharmacological nuclear stress testing may be another modality that can be used to differentiate etiology of the symptoms.

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