Hemodynamic Evaluation of Suspected Severe Aortic Stenosis

Leads to a Diagnosis of Renal Cell Carcinoma

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The evaluation of aortic stenosis is not always straightforward. When symptoms of severe aortic stenosis are present with supporting Doppler echocardiographic or cardiac catheterization data, replacement of the aortic valve is recommended. Occasionally, Doppler- and catheter-derived data are discordant; appropriate treatment in such cases becomes less clear.

We report a case in which a 66-year-old man's symptoms and Doppler data suggested severe aortic stenosis. However, heart catheterization data suggested otherwise, and ultimately it led to the diagnosis of a highly vascular renal tumor. Shunting within the tumor resulted in high cardiac output, which, in combination with a small aortic root, masqueraded as severe aortic stenosis. (Tex Heart Inst J 2015;42(1):77-9)

Key words: Aortic valve stenosis/classification/diagnosis/physiopathology; blood flow velocity; cardiac catheterization/diagnostic; cardiac shunt/evaluation; echocardiography, Doppler

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Degenerative aortic stenosis (AS), the most common acquired valvular lesion in the developed world, poses a significant problem in the elderly.^{1,2} Accurate evaluation of AS is important, because survival rates decline sharply once the disease becomes severe.³ Surgical aortic valve replacement is recommended in severe symptomatic AS, and transcatheter aortic valve replacement has recently emerged as an option for patients with prohibitive surgical risk.⁴

The proposed American College of Cardiology/American Heart Association criteria for severe AS include an aortic valve area (AVA) of $<1 \text{ cm}^2$, a peak transthoracic echocardiographic (TTE) Doppler velocity of $>4 \text{ m/s}$, and a mean gradient of $>40 \text{ mmHg}$.⁵ However, these guidelines do not specify which method, catheter or Doppler echocardiography, should be used for the evaluation. Frequently, the 2 methods correlate well, although on occasion discrepancies add to the complexity of the evaluation. Herein, we present an unusual case that emphasizes close attention to the hemodynamic evaluation of AS.

Case Report

A 66-year-old man with hypertension, hyperlipidemia, and obesity presented with chest discomfort and dyspnea. His heart rate was 51 beats/min and his blood pressure was 137/55 mmHg. Auscultation, although revealing a late-peaking 3/6 systolic murmur, was accompanied by a normal A_2 sound. Pulsus parvus et tardus was absent. An electrocardiogram lacked signs of ventricular hypertrophy. A TTE showed a normal left ventricular ejection fraction (>0.55) and a calcific trileaflet aortic valve. Other pertinent echocardiographic findings included an aortic root diameter of 2.3 cm, a peak velocity of 4.84 m/s, a mean aortic valve gradient of 56 mmHg, and a calculated AVA of 0.97 cm^2 , all of which were consistent with severe AS.

This lack of consistency between the echocardiogram and the physical examination prompted cardiac catheterization. A diagnostic coronary angiogram showed mild non-obstructive atherosclerosis. Catheterization yielded a mean gradient of 39.8 mmHg across the aortic valve, and a Fick-derived cardiac output of 8.6 L/min. An unexpected increase in pulmonary artery saturation (to 81%) was detected. No intracardiac shunt was found by means of oximetry, although a step-up of 9% in saturation between the lower and upper inferior vena cava was detected. Subsequent computed tomograms of the abdomen revealed a highly vascular renal tumor.

Catheter data indicated only mild-to-moderate AS and an AVA of 1.36 to 1.52 cm² (Hakki and Gorlin formulae, respectively). High cardiac output from intratumor shunting—due to increased vascularity and suspected arteriovenous malformations—resulted in increased Doppler gradients, which mimicked severe AS.

When the patient underwent nephrectomy, a histologic specimen confirmed a diagnosis of renal cell carcinoma with arteriovenous malformations. He did well for over a year and his aortic valve disease remained stable. He was eventually found to have metastatic disease and is now undergoing chemotherapy.

Discussion

This is an engaging case in which spuriously elevated gradients from echocardiography suggested severe AS. Further evaluation overturned that assumption and led to a diagnosis of malignancy. The presence of a small aortic root and high cardiac output were meaningful.

Doppler- and catheter-derived AVAs are obtained differently. The peak instantaneous gradient, used in echocardiography, reveals the effective area of the orifice. Catheter-derived pressure gradients, which approximate the anatomic area, yield the net difference in pressure across the valve, thereby accounting for recovered pressure distal to the stenosis.^{6,7} Because Doppler does not account for recovered pressures, Doppler AVAs are often smaller than those derived by catheterization. It is important to note that the magnitude of recovered pressure is greater when aortic roots are smaller and transvalvular velocities are higher.⁸ In our patient, these factors accounted for a pressure gradient that was 16 mmHg lower by catheterization.

In addition, the calculated AVA is dependent on flow, because velocity is proportional to volume flow rate.⁹ Therefore, in the presence of high flow, the AVA calculated by the continuity equation would be smaller than the true AVA. On the other hand, the degree of valve opening can be affected by the driving force applied to the valve.¹⁰ That is, high cardiac output applies greater force to the valve, which can result in a greater orifice area—thereby causing a severely stenotic valve to appear less stenotic. To resolve the influence of flow, we applied the ratio of the left ventricular outflow stroke volume to transaortic stroke volume (dimensionless index): a measure that better identifies patients with severe AS than does a Doppler gradient (sensitivity, 97% vs 81%, respectively).¹¹ We obtained a value of 0.3, which was inconsistent with severe AS.

Discrepant hemodynamic variables can lead to entirely different diagnoses. In this patient, higher-than-expected pulmonary artery saturation was an important clue to a shunt. We suspected an anomalous pulmonary venous connection between a pulmonary vein and the

inferior vena cava. However, the source of the shunting—a highly vascular tumor—became more apparent after a review of the computed tomographic scan.

It is known that states of high cardiac output can mimic severe AS,¹² although it is exceedingly rare to find that both high-output state and small aortic root size have led, in the same patient, to erroneous measurements.

Our case well illustrates the occasional difficulty of evaluating AS. An understanding of the hemodynamics of AS, together with a recognition of some subtle abnormalities, ultimately led to our reclassifying this patient's "severe" AS as mild-to-moderate. Aortic valve replacement was no longer necessary, and the alternative diagnosis of renal cell carcinoma was made.

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