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CLINICAL VIGNETTE

A Case of Acute Pulmonary Edema from Severe Aortic Stenosis

Neil Patel, M.D.

Case Report

A 70 year-old male with a history of hypertension and hyperlipidemia was brought to the Emergency Department by ambulance from home in significant respiratory distress. The patient was only able to communicate in 3-4 word sentences between breaths and history was assisted by his family members. The patient reported progressively worsening shortness of breath with exertion over the past 2 months, but stated that it was much worse and present at rest for the past 2-3 days. His family also reported the patient had experienced several episodes of exertional lightheadedness and syncope over the past month without preceding palpitations. He denied any chest pain/pressure, recent immobilization, cough, fever, tobacco use or history of previous pulmonary disease.

The patient's vitals were as follows: temperature 97.8°F, pulse 98, blood pressure 118/88, respiratory rate 22, pulse oximetry of 92% on room air. Physical exam revealed an elderly Caucasian male in moderate respiratory distress. JVP was elevated at 15 cm. The cardiac exam revealed a late-peaking crescendo-decrescendo murmur best appreciated at the right upper sternal border with radiation to the carotid arteries. It was further noted that the carotid upstroke was slightly delayed in relation to cardiac auscultation. Rales were appreciated over the bilateral posterior lung fields approximately halfway up. There was only trace bilateral lower extremity pitting edema with no calf tenderness.

An ECG revealed normal sinus rhythm with left axis deviation, evidence of left ventricular hypertrophy and "strain pattern" ST-T changes without definitive evidence of acute infarction or ischemia.

A portable chest x-ray revealed borderline cardiomegaly with a calcified aortic valve and evidence of significant pulmonary edema.

Given the above findings, a presumptive diagnosis of acute congestive heart failure in the setting of suspected severe aortic stenosis was made. The patient was started on a low dose nitroglycerin drip and BiPAP therapy was initiated with a dobutamine drip on standby to be used in case of significant hypotension. The patient was then admitted to the Cardiac Intensive Care Unit where a bedside ultrasound confirmed the diagnosis of severe aortic stenosis with a mean calculated valve area of 0.7 cm^2 and an aortic jet velocity of 4.8 m/s.

The patient underwent an urgent mechanical aortic valve replacement 2 days later with resolution of his symptoms and return to baseline functional status by 3 months after surgery.

Discussion

The above case illustrates the difficulty managing acute cardiogenic pulmonary edema in the setting of severe/critical aortic stenosis. Whereas most patients with acutely decompensated heart failure resulting from a non-valvular source can be liberally treated with preload reducing agents (loop diuretics, nitrates), patients with severe aortic stenosis are in a relatively preload dependent state, even when exhibiting signs of severe pulmonary edema [1-5].

This is due to the concentric hypertrophy that accompanies increased pressure work against a stenotic aortic valve. Not only does this reduce left-ventricular end-diastolic volume (LVEDV) but also increases end-diastolic pressure (LVEDP), leading to impaired pulmonary venous return and congestion[2,3,6,7]. Unfortunately, if loop diuretics such as furosemide were to be used liberally in such patients, the resultant decrease in preload could lead to severe hypotension as LVEDV is already low to begin with[3,7,8].

Conversely, rapidly providing intravenous fluids to hypotensive patients with severe aortic stenosis could lead to acute or flash pulmonary edema as the high LVEDP would not be able to accommodate the extra fluid, leading to congestion of the pulmonary vasculature and subsequently, the pulmonary parenchyma. Therefore, the management of both hypotension and pulmonary edema in patients with

severe aortic stenosis must be undertaken rather cautiously as it is fraught with peril[2,3,8,9].

It is therefore highly advisable to use rapidly titratable parenteral agents when treating aortic stenosis associated acute pulmonary edema[2]. Furthermore, medical management should only be undertaken as a temporizing bridge to surgical correction of the stenosis and only when symptoms are significantly distressing to the patient or when there is marked hemodynamic compromise[2,6]. Use of preload reducing agents, as stated above, is relatively contraindicated in patients with severe aortic stenosis[1]. However, they may be required in critically ill patients such as our patient. In these instances, it is best to use either nitroglycerin or nitroprusside at low doses with gradual upward titration. Both of these agents have rapid onset and offset and are frequently used in critical care[10].

If hypotension results from excessive preload reduction, the drip can simply be held and/or a dobutamine drip can be initiated. This latter drug acts as a positive inotrope, thereby increasing cardiac contractility and output[2,3].

The use of BiPAP in this setting is also a temporizing measure and serves to decrease preload by increasing intrathoracic pressure as well as reducing the work of breathing while providing a higher FiO_2. Should hypotension result, BiPAP can simply be discontinued with no lasting effect[2,3,8].

Unfortunately, there is a dearth of evidence for the above agents in managing acute pulmonary edema in patients with severe/critical aortic stenosis. However, expert consensus and several case reports support their use[2,3,8].

The patient with acute severe pulmonary edema resulting from critical aortic stenosis presents a clinical scenario that is fraught with peril as imprudent use of seeming "routine" heart failure medications can lead to frank cardiovascular collapse. By medically intervening only when absolutely necessary and judiciously using rapidly acting, readily titratable agents in a highly monitored setting, the medical clinician may safely deliver the patient to the operating theater for definitive management.

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