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Clinical Review

EVALUATION AND MANAGEMENT OF AORTIC STENOSIS FOR THE EMERGENCY CLINICIAN: AN EVIDENCE-BASED REVIEW OF THE LITERATURE

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☐ **Abstract—Introduction: Aortic stenosis is a common condition among older adults that can be associated with dangerous outcomes, due to both the disease itself and its influence on other conditions. Objective: This review provides an evidence-based summary of the current emergency department (ED) evaluation and management of aortic stenosis. Discussion: Aortic stenosis refers to significant narrowing of the aortic valve and can be caused by calcific disease, congenital causes, or rheumatic valvular disease. Symptoms of advanced disease include angina, dyspnea, and syncope. Patients with these symptoms have a much higher mortality rate than asymptomatic patients. Initial evaluation should include an electrocardiogram, complete blood count, basic metabolic profile, coagulation studies, troponin, brain natriuretic peptide, type and screen, and a chest radiograph. Transthoracic echocardiogram is the test of choice, but point-of-care ultrasound has been found to have good accuracy when a formal echocardiogram is not feasible. Initial management should begin with restoring preload and ensuring a normal heart rate, as both bradycardia and tachycardia can lead to clinical decompensation. For patients with high blood pressure and heart failure symptoms, nitrate agents may be reasonable, but hypotension should be avoided. Dobutamine can increase inotropy. For hypotensive patients, vasopressors should be used at the lowest effective dose. The treatment of choice is valve replacement, but extracorporeal membrane oxygenation and percutaneous balloon dilatation of the aortic valve have been described as temporizing measures. Conclusion: Aortic stenosis is an important condition that can lead to dangerous outcomes and requires prompt recognition and disease-specific management in the ED. Published by Elsevier Inc.**

☐ **Keywords—aorta; aortic stenosis; aortic sclerosis; cardiogenic shock; cardiac; valve; valvular**

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INTRODUCTION

Background

Abnormalities of the aortic valve are frequent among older patients, with aortic stenosis (AS) the most common valvular disease in the developed world (1–4). The Cardiovascular Health Study found that 26% of patients aged ≥ 65 years demonstrated aortic sclerosis, with 2% demonstrating AS (5). Patients over 80 years of age demonstrate rates of AS approaching 10% (4,6,7). Symptoms depend on the degree of valvular stenosis and aortic flow gradient and typically follow a defined course (4,8). Once symptoms develop, rapid clinical

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deterioration can occur, commonly within 2–3 years, unless treated (9–14).

These patients can present to the emergency department (ED) with either known or undiagnosed disease and can present with symptoms ranging from earlier disease to cardiogenic shock (4,8). This review seeks to provide emergency clinicians with an understanding of relevant anatomy and pathophysiology, history and physical examination, and the ED evaluation and management of critical AS.

METHODS

The authors searched PubMed and Google Scholar for articles using a combination of the keywords "aorta," "aortic," "valve," "valvular," "sclerosis," and "stenosis." The literature search was restricted to studies published in English. Authors decided which studies to include for the review by consensus. A total of 96 articles were selected for inclusion in this review.

DISCUSSION

Anatomy and Underlying Causes

AS occurs as a result of inflammation from endothelial damage and mechanical stress, followed by lipid penetration, fibrosis, thickening of the valve leaflets, and eventually, calcification (4,8,9). The most common causes of AS are calcific AS, congenital AS, and rheumatic valvular disease (4).

Calcific AS develops from a previously normal tricuspid aortic valve, which becomes stenotic through a process similar to atherosclerosis (15–17). Risk factors include advanced age, male sex, hyperlipidemia, and inflammation, which can lead to valvular stenosis, typically occurring beyond the sixth decade of life (15–17). The process begins with mild calcification of the valve cusps, termed calcific sclerosis, which worsens over time (4). Calcific sclerosis is independently associated with a higher risk for cardiac death (16). Bicuspid aortic valves (1–2% of the population) are also at higher risk for calcification (4,18,19). Therefore, there is a greater prevalence of AS in these patients, and they demonstrate AS two decades earlier than the normal population (4,18,19).

Congenital AS is usually detected in childhood. The congenital form is typically not associated with a bicuspid valve, but rather a unicuspid, unicommissural valve (4,18,19). Affected children often die at a young age or develop symptoms that lead to an early valve replacement (18,19). Sudden death in this population is common, and unfortunately, patients with this form of AS often do not demonstrate angina or heart failure (20,21). Sudden death is correlated with left ventricular (LV) strain on electrocardiogram (ECG) and an ejection fraction (EF) that is supranormal (4,20,21).

Rheumatic valvular disease is another cause, although this is exceedingly rare in developed countries (4,8,22). Patients with this form of AS usually display mitral valve involvement as well (22). In rheumatic AS, fusion of the valve commissure is present, similar to the congenital type (4,8,22).

Regardless of the underlying etiology, the stenotic valve results in increased stiffness and a narrowed valve orifice, increasing the pressure gradient over the aortic valve (8,23). Progressive valve narrowing and an increased pressure gradient leads to LV pressure overload and hypertrophy (4). These changes cause the classic triad of AS with angina, syncope, and heart failure (3,4,7). Despite the severity of the stenosis, asymptomatic patients typically have improved mortality (4,24). However, once the onset of symptoms begins, the mortality rate increases by approximately 25% (4,24).

History, Physical Examination, and Pathophysiology

History and physical examination are essential in the diagnosis and prognostication of this disease. The condition is typically clinically latent for years in the form of aortic sclerosis (4,7–9). However, the development of symptoms such as angina, syncope, or heart failure is associated with poor prognosis, with survival decreasing to 2–3 years (3,4,7,11–14). Advanced symptoms are associated with extensive myocardial damage (25–28). Unfortunately, symptoms can be subtle in some patients, and sudden death may occur without preceding symptoms suggestive for severe disease (3,4,7,11–14). Symptoms develop in one-third of patients within 3–5 years, with the rate of progression of valvular disease approximating 0.1 cm^2 per year (29–36).

A normal aortic valve orifice approaches 3 cm^2 (4,8). Once this is reduced by half, the pressure gradient across the valve increases drastically (3,4,7,8). As a result, the LV pressure and afterload increases, resulting in LV hypertrophy (37–40). Although hypertrophy helps to maintain the EF, it impairs coronary blood flow reserve and worsens diastolic function (41–47). As the oxygen extraction in the heart approximates 100% (unlike other organs), increasing oxygen demand in times of stress can only be matched with increased oxygen flow in the heart (4,8). Hypertrophy reduces this ability in AS, and the increased filling pressure further impairs blood flow to the endocardium (43,44). These two components can lead to symptoms of angina. Although the degree of angina symptoms does not correlate well with the extent of the hypertrophy, it does correlate moderately