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Lee, Melissa, "Pathophysiology of Aortic Stenosis" (2022). *Nursing Student Class Projects (Formerly MSN)*. 508.

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Pathophysiology of Aortic Stenosis

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Introduction

Aortic stenosis (AS) is one of the most common heart valve diseases encountered in clinical practice (Lindman et al., 2020)

Current research into the valvular pathophysiology of aortic stenosis has determined it is a disease of both the valve and the myocardium (Zheng et al., 2020).

Defined as the progressive narrowing of the aortic valve opening, secondary to calcification, that restricts blood flow from the left ventricle to the aorta (American heart association, 2020).

Stages of aortic stenosis disease progression are similar to the stages of heart failure, characterized by mild, moderate and severe – correlated with cardiac function (Kanwar et al., 2018)

Clinical Significance

Severe aortic stenosis has an estimated two-to-three-year survival rate, and when left untreated mortality rates reach 25% (Joseph et al., 2017).

In the United States, approximately 5.6 million adults have notable valvular dysfunction (Kanwar et al., 2018).

AS affects 2-5% of older adults, and ten percent of the population in the 80–89-year-old cohort (Joseph et al., 2017).

Early recognition and management of aortic stenosis is essential, untreated symptomatic disease is considered universally fatal (Kanwar, 2018).

Currently there is no effective pharmacologic treatment to slow disease progression (Joseph et al., 2017).

Broad-scale education is a public health necessity due to the rapidly growing elderly population and the insidious primary asymptomatic disease process (Zheng et al., 2020)

Aortic Stenosis is set to become a major health care burden (Zheng et al., 2020)

The hemodynamic consequences of aortic stenosis – increased afterload, left ventricular hypertrophy, heart failure are often unrecognized before the patient becomes symptomatic. Education on AS is important for providers to identify at risk patients to promote desired patient outcomes (Zheng et al., 2020)

Signs and Symptoms

Initial development of aortic stenosis involves an asymptomatic latent period lasting 10 to 20 years on average. Symptom onset is associated with a 90% mortality rate (Pujari & Agasthi, 2022)

Cardiac symptoms

Syncope Most common during exertion due to decreased cerebral perfusion. Stenosis causes a decrease in atrial pressure, systemic vasodilation and inadequate increase in cardiac output needed during activity (Pujari & Agasthi, 2022)

Angina Results from the increased oxygen demand from myocardial hypertrophy and reduced oxygen delivery to the myocardium secondary to compression of the coronary vessels (Pujari & Agasthi, 2022)

Signs of pulmonary venous hypertension Exertional dyspnea, paroxysmal dyspnea, orthopnea and pulmonary edema (Pujari & Agasthi, 2022)

★ **Hallmark sign:** Auscultation of a mid-systolic ejection murmur with a crescendo-decrescendo pattern that radiates to the carotids (Pujari & Agasthi, 2022)



Non-cardiac symptoms

Gastrointestinal bleeding. Often associated with severe aortic stenosis. Bleeding occurs from shear stress-induced platelet aggregation and reduction in von Willebrand factor (Pujari & Agasthi, 2022)

Risk Factors

Age- Each decade increase in age results in a 75% increase in risk for developing AS (Yan et al., 2017).

Gender- There are higher incidences of AS in male patients (Yan et al., 2017). The male sex is associated with a 32% excess risk (Kanwar et al., 2018)

Cardiac risk factors including hypertension, diabetes and dyslipidemia are correlated with higher incidences of aortic stenosis (Yan et al., 2017).

Patients who have a past medical history including congenital heart disease, valvular heart disease and coronary heart disease are considered high risk for the development of AS (Yan et al., 2017).

Pathogenesis

Aortic stenosis pathogenesis occurs in two distinct phases: Initiation phase and Propagation phase (Zheng et al., 2020).

Initiation Phase: Begins with an injury to the valvular endothelium, secondary to mechanical stress or other risk factors. Lipoproteins accumulate and undergo oxidative modification causing a chronic inflammatory response. Recruitment of inflammatory cells (macrophages, T-cells, mast cells) activate valve interstitial cells (VICs) (Zheng et al., 2020).

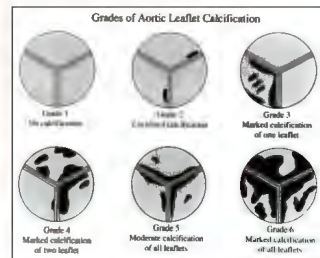
Propagation Phase: Activated VIC cells produce collagen which leads to fibrosis, valvular thickening and stiffening. VIC cells release calcifying vesicles that result in calcium accumulation. Progressive calcification of the aortic valve occurs when VIC cells differentiate into osteoblast-like cells, which follows similar pathways of bone calcification (Zheng et al., 2020).

Diagnostics

Diagnostic Criteria: Presence of a structurally abnormal valve (thickened or calcified), restricted motion, and doppler evidence of hemodynamic obstruction (Harris et al., 2022).

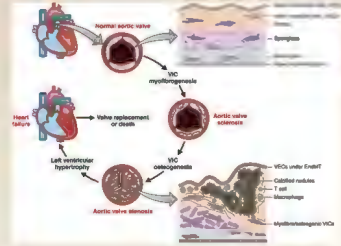
Transthoracic Echocardiogram (TTE) for symptomatic patients with unexplained systolic murmur, single heart sound, history of bicuspid aortic valve, and history of an infectious cardiac illness (ex. Endocarditis) (Harris et al., 2022)

Transcatheter Echocardiogram (TEE) for asymptomatic patients are repeated every three to five years to monitor inevitable progression from mild, moderate to severe valvular obstruction (Harris et al., 2022)



(Bahler et al., 2004)

Pathophysiological Process

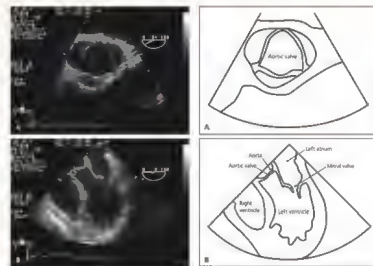


(Blaser et al., 2022)

The pathophysiological processes of aortic stenosis although similar, vary based on the primary cause of initial disease development.

- Causes of AS include a congenital heart defect that results in an aortic valve that has two cusps (bicuspid) instead of three (tricuspid) (Mayo Clinic, 2021).
- Rheumatic fever is a complication of strep throat that can form scar tissue on the aortic valve. The scar tissue creates a narrow aortic valve, and the rough surface promotes calcium deposit collection (Mayo Clinic, 2021).

(A) Normal TTE (B) Severe aortic stenosis



(AHA, 2021)

Grading the Severity of Aortic Stenosis

- Stage A:** Valve disease without hemodynamic effects (Svensson et al., 2021)
- Stage B:** moderate AS progressive disease, minor hemodynamic changes (Svensson et al., 2021)
- Stage C:** Asymptomatic severe AS, pressure gradient >40 mmHg
- Stage D:** Symptomatic severe AS, pressure gradient >40 mmHg (Svensson et al., 2021)

Pathophysiology

Aortic valve calcification to valvular obstruction occurs over a period of two to five years (Kanwar et al., 2018)

Fibrosis of the Aortic Valve: Fibrotic remodeling contributes to increased stiffness of the valve leaflets.

Increased production of extracellular proteins such as plasminogen activator inhibitor, regulate the fibrotic process (Zheng et al., 2020).

Renin-Angiotensin-Aldosterone System (RAAS): Chronic inflammatory response and recruitment of inflammatory mediators cause increased local levels of angiotensin-converting enzyme and directly contribute to the fibrotic remodeling of aortic valve leaflets (Zheng et al., 2020).

Left Ventricular Hypertrophy: To maintain cardiac function with progressive aortic valve narrowing and increased afterload, myocytes become hypertrophied to reestablish ventricular wall stress (Zheng et al., 2020).

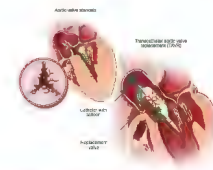
Surgical Options

Patients undergo aortic valve replacement (AVR) for improved quality of life and left ventricular function (Boskovski & Gleason, 2021)

Indications for AVR are stage C and D patients, classified as severe AS (Boskovski & Gleason, 2021)

Transcatheter AVR (TAVR) is a surgical approach that is transcatheter, minimally invasive with minor complications and requires no anticoagulation postoperatively (Boskovski & Gleason, 2021).

AVR with a mechanical or biological valve requires cardiopulmonary bypass. This includes cardioplegic arrest, midline sternotomy and removal of the calcified valve. This approach is most common for patients with moderate to severe aortic stenosis, or over the age of 65 (Boskovski & Gleason, 2021).



(Mayo Clinic, 2021)

Treatment

- Surgical intervention is the only treatment available for aortic stenosis
- Patients less than 50 years old are eligible for mechanical valve replacement (Svensson et al., 2021)
- Patients 50-65 years old, the decision to place a biological valve or a mechanical valve is individualized (Svensson et al., 2021)
- It is recommended that patients over the age of 65 receive a biological valve (Svensson et al., 2021)



(Medtronic, 2022)

Biological Valve



Mechanical Valve

(CTHSurgery, 2022)

Nursing Implications

- It is important for the nurse anesthetist to be aware of which type of valve replacement the patient has. Mechanical valves require lifelong anticoagulation, which is important for preoperative and perioperative monitoring due to increased risk for bleeding.
- Hypertension in patients with AS is associated with higher mortality rates. This is due to an increase in afterload, leading to decreased cardiac output and cardiac ischemia (Kanwar et al., 2018)
- To prevent increased ventricular filling pressures and pulmonary hypertension due to hypertension, vasodilatory therapy is paramount for symptom improvement (Kanwar et al., 2018).
- Patients with AS require a high preload. Therefore hypovolemia is poorly tolerated. It is important that nursing staff account for the patients baseline fluid status and administer intravenous fluids as needed (Whitener et al., 2021)
- Many anesthetic agents cause peripheral vasodilation, AS patients have higher rates of hypotension under general anesthesia and are unable to compensate. It is important to have vasoconstricting medications readily available to maintain baseline high afterload and increased systemic vascular resistance (Whitener et al., 2021)
- Slower heart rates are best tolerated in patients with AS because it allows for increased diastolic filling time and cardiac filling. The vasopressor of choice for patients with AS is Phenylephrine. Phenylephrine increases coronary perfusion without increasing the heart rate (Whitener et al., 2021).
- Regardless of the type of surgical procedure, it is important for patients with AS to have appropriate monitoring while under general anesthesia. This includes an arterial line to measure real-time blood pressure to quickly recognize hypotension, and a central line to monitor central venous pressure trends (Whitener et al., 2021).
- Although assessment accuracy of fluid volume status using a central venous pressure line is controversial, monitoring trends are useful when expecting large fluid volume shifts (Whitener et al., 2021).
- Due to the complex hemodynamic changes that occur with AS, multidisciplinary preoperative planning is essential for best patient outcomes. This includes educating the patient on the possible need for continued postoperative support and possible complications that can occur (Whitener et al., 2021).
- Postoperative nursing care includes monitoring for cardiac dysrhythmias, hypotension and symptoms of myocardial ischemia (Whitener et al., 2021).

Conclusions

- With the increasing population of aging patients, AS will be commonly encountered in the medical field. Due to the complexity of the pathological process of AS, often patients are not diagnosed until symptoms occur
- Intraoperative anesthetic management of patients with AS includes maintenance of normal sinus rhythm, fluid volume status, and rapid correction of hypotension (Whitener et al., 2021)
- The insidious asymptomatic disease progression makes it difficult for practitioners to predict when patients will become symptomatic (Whitener et al., 2021).
- Undergoing intervention before the symptomatic phase of disease progression is not recommended due to the increased risk of procedural complications (Whitener et al., 2021).

References



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