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A review of aortic stenosis: an anesthetic perspective

Abstract

Anesthetists face many challenges in the operating room. Many patients have severe comorbidities that the anesthetic plan must be tailored to. In the United States, aortic stenosis (AS) is the most common valvular disorder.[1–4] AS affects nearly two to four percent of the population over the age of 65.[5] Patients that are diagnosed with severe AS have a three year mortality of 75 percent unless the valve is replaced.[3] Aortic stenosis is characterized by narrowing of the aortic valve (AV) causing a litany of symptoms. This review will discuss the anatomy and physiology, etiology, pathophysiology, and symptoms associated with AS. Additionally, anesthetic considerations for the care of a patient with AS will be reviewed.

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Description of disease

Anatomy and physiology

The closed AV serves as a barrier to prevent blood from backflowing into the left ventricle of the heart during diastole and also the forward flow of blood into the aorta to allow ventricular filling. The AV's structural significance helps aid forward unidirectional flow of blood through the heart and to body. The AV consists of three leaflets that are named according to their location in relation to the coronary ostia: left, right, and noncoronary.[4] When describing the severity of AV stenosis, clinicians will discuss the transvalvular gradient and the valvular surface area.[3] Sources may vary, but the normal adult aortic valve surface area is around two to four centimeters squared.[4] AS is considered severe when the transvalvular pressure gradient is higher than 50 millimeters of mercury and the valvular surface area is less than 0.8 centimeters squared.

Etiology

Two major factors are associated with development of AS.[3] One such factor is the calcification of the AV leaflets leading to stenosis. Advancing age is the primary cause of the calcification of the AV leaflets. The second factor is the presence of a valvular abnormality from birth. A congenital bicuspid valve that replaces the normal tricuspid AV can lead a patient to develop AS earlier in life.[6] Other causative factors include rheumatic heart disease from group A streptococci rheumatic fever and infective endocarditis. Risk factors for AS include systemic hypertension, hypercholesterolemia, age, male gender, diabetes mellitus, cigarette smoking, metabolic syndrome, and end-stage renal disease.[3,4,6]

Pathophysiology

The major pathophysiologic concerns with severe AS include concentric hypertrophy, decreased diastolic compliance, and a narrow balance of myocardial oxygen supply and demand.[3,4,6] The pathological consequences of AS are contributed to left ventricular outflow obstruction caused by the stenotic valve. Over time, the AV becomes more calcified and thick which worsens the outflow obstruction. The calcification is progressive and degenerative, but also thought to be an inflammatory atherosclerotic process similar to coronary artery disease.[6] The outflow of blood through the AV during systole progressively decreases over time as the AV narrows. Thickening of the left ventricle (LV) occurs due to the increased force of contraction needed to eject the appropriate amount of blood through the narrowed AV. The changes in the LV are considered concentric in nature due to the thickening of the ventricle myocardium. AS can also be associated with a small degree of aortic regurgitation. Aortic regurgitation can lead to decreased coronary artery filling pressure resulting in ischemia.[3]

Signs and symptoms

The classic symptoms of AS have similarities with coronary artery disease: angina, syncope, dyspnea on exertion, and congestive heart failure (CHF).[3,4] The angina felt with aortic stenosis is related to the increased myocardial oxygen demand without corresponding increase in oxygen supply.[3] Syncope is usually attributed to the dilation of the systemic vascular system and cardiac output cannot compensate leading to a drop in mean arterial blood pressure. Dyspnea and CHF is due to the increased pressure applied to the pulmonary system from the outflow obstruction of AS.

Treatments

If one of the classical symptoms including dyspnea, fatigue, angina pectoris, or syncope occurs, the patient's prognosis worsens dramatically, and valve replacement is indicated.[3] Gold standard therapy for AS is surgical valve replacement.[5] AV replacement via percutaneous transcatheter implantation, or AV bypass are methods of surgical intervention.[1,7] Valvular replacement usually includes cardiopulmonary bypass (CPB) which requires additional anesthetic implications that are outside the scope of this review. AV replacement is not without risk for the patient though. A valve replacement exposes patients to the risks associated with sternotomy or thoracotomy, cardiopulmonary bypass, cardiac arrest and general anesthesia.[3,5] The risks associated with an AV replacement are especially worrisome in the elderly population and those with medical comorbidities. According to the Euro Heart Survey on Valvular Disease, up to one third of AS patients are over the age of 75 and have contraindications for conventional surgery.[2] Also, a heavily calcified aorta, previous mediastinal radiation, and redo valvular surgery are frequent causes of concern for providers.[5] The stated risks lead providers to delay AV replacement in up to one third or more of patients with severe symptomatic AS and try to manage without operative intervention because of the high risk involved with surgery.

Medical management of AS includes antihypertensives, beta blocking drugs, vasodilators, antianginals, and hydration.[4] Antibiotics are used to prevent worsening endocarditis if present.[6] Recent studies have found that statin therapy can decrease the progression of AS. However, patients with AS treated with medical therapy still have a poor prognosis. It is important to note that asymptomatic patients with AS have a good prognosis, but must be reevaluated on a regular basis for worsening of their disease. An evaluation using echocardiography (ECHO) is indicated every one to five years.

Anesthetic considerations

The anesthetic plan can vary for the type of procedure and also the severity of AS. A different anesthesia approach could be needed for cardiac and non-cardiac surgery even though they have many similarities.[6] Cardiac surgery usually pertains to a major surgical intervention of an AV replacement and CPB. Non-cardiac surgery, for a variety of reasons, can be performed on a patient with AS. The complexity of non-cardiac surgery can fluctuate depending on the type of procedure.

Preoperative evaluation

The preoperative evaluation and examination is essential to establish the patient's fitness for surgery. A thorough review of the patient's history and physical exam is important to determine any comorbidities or considerations for the anesthetic plan. A physical exam is essential as well to evaluate the patient's current physical status. It is important to evaluate the severity of AS prior to surgical.[3] Transvalvular pressure gradient and the valvular surface area that was determined in preoperative testing from ECHO with doppler should be noted. ECHO is considered the gold standard for diagnosis of AS and also severity of the disease.[6] Cardiac catheterization might be necessary to determine the severity of AS if an ECHO is not definitive.[3] Other factors to consider, when determining the severity of AS, would be the severity of symptoms associated with the condition and current physical status.[6]

On assessment, a grade 3 or 4 systolic murmur can be heard with signs of CHF.[6] Antibiotics are a must for a patient that has a history of rheumatic heart disease or previous valve replacement. Fluid status must be optimized on patients with AS due to their decreased LV compliance. An exercise stress test can give the clinician insight into the patient's stress tolerance prior to surgery. Baseline laboratory values should be considered before surgery including basic metabolic panel, complete blood count, ECG, and coagulation studies.

Intraoperative management

The anesthetic for an AS intervention is typically a general anesthetic due to the detrimental effects of the sympathectomy produced by a spinal or epidural.[3] Severe AS is a contraindication for regional anesthesia.[4,6] Alternatively, certain procedures such as cardiac catheterization or percutaneous valve implantation could be done under deep sedation.[2] The goal for intraoperative management is to maintain the patient's vital signs at baseline.[4] The main cardiovascular goals for AS include full preload, maintain afterload (SVR), maintain contractility, avoid bradycardia and tachycardia, and maintain normal sinus rhythm.[3] It is important to maintain sinus rhythm when managing AS since atrial kick may account for up to 30 or 40 percent of LV end diastolic volume.[4] Intraoperative monitoring of a patient with AS includes the standard monitors of a general anesthetic: blood pressure, heart rate, electrocardiograph, pulse oximetry, end tidal carbon dioxide and temperature.[3] An arterial catheter is often used to closely monitor the patient's blood pressure to provide beat to beat assessment. Other monitors used could include a central line with central venous pressure capabilities, pulmonary artery catheter, entropy monitor, and transesophogeal echocardiography.

Induction of anesthesia for a patient with AS should focus on avoiding a drastic decrease in the patient's SVR causing a decrease in systolic and diastolic blood pressure while also avoiding bradycardia or tachycardia.[3] A high opioid induction with a benzodiazepine can be beneficial for the AS patient when LV function is decreased. Ketamine as a solo agent should be avoided due to the incidence of tachycardia and hypertension, but in low concentrations, it can be combined with propofol and narcotics for a suitable induction combination. Maintenance of anesthesia can be in the form of low dose inhaled anesthetics or a total intravenous anesthetic with propofol and an opioid.[3] Ideal vital sign goals are the same as pre-induction goals of normotensive, maintain SVR, maintain contractility, adequate preload, and sinus rhythm. Hypotension should be aggressively treated with an alpha agonist. Tachycardia can be treated with a beta blocking agent like esmolol. For valvular replacements under general anesthesia, hemodynamically stable nondepolarizing muscle relaxants are best for paralysis. If CPB is used, the patient must be heparinized and coagulation studies must be drawn by the perfusionist or the anesthetist at frequent intervals.

Postoperative management

Postoperative care is focused on ensuring the patient's vital signs return to baseline. The AV replacement patient usually recovers in the intensive care unit following the procedure. Hydration is important and hypotension should be avoided. Pain management is essential since uncontrolled pain can lead to hypertension, tachycardia, and worsening LV outflow obstruction causing increased myocardial oxygen demand.

Conclusion

Patients with AS can be complicating for anesthesia providers that do not understand the pathophysiology of the disease. A detailed understanding of AS is essential for all anesthesia providers, considering the correlation of our aging population and AS, to provide high quality care. AS is a disease of the cardiac AV that is caused by a calcification or thickening of the leaflets leading to a narrowing of the outflow area. Severe AS has an extremely high mortality rate. The gold standard treatment is surgical intervention with AV replacement. A good anesthetic plan can lead to positive outcomes if consideration is given to the pathophysiology of AS, patient comorbidities, and strict vital sign maintenance throughout the case. Smooth inductions, with appropriate agents that avoid drastic swings in vital signs, are ideal. AS is a disease process that could be associated with many of the surgical patients anesthetists encounter. With the appropriate training, AS can be managed successfully in the operative setting.

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Conflicts of interest

Author declares that there is no conflict of interest.

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