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Calcific Aortic Stenosis

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Introduction

Degenerative calcific aortic stenosis is a progressive disease that becomes more severe with age and is a direct result of an active inflammatory process. The life expectancy of the United States' population is increasing and with it, the incidence of aortic stenosis has increased over the past several decades. Surgical data suggests an rise in prevalence of the disease by over 25% in the past ten years (Bonow & Greenland, 2015). Advanced Practice Nurses (APNs) are more frequently encountering patients of an advanced age and recent technologic advancements have created more treatment options for aortic stenosis. APNs should be aware of this disease process and should be knowledgeable about these options so appropriate care can be provided.

While 25% of the population has sclerotic aortic valve disease, only an estimated two percent demonstrate calcific aortic stenosis at age 75 (Novaro, 2014). Development of this type of aortic stenosis arises from the same process associated with atherosclerosis and its risk factors include hyperlipidemia, hypertension, smoking, and diabetes (Maganti, Rigolin, Sarano, & Bonow, 2010). Congenital bicuspid valves appear to undergo this change more rapidly (Ray, 2010).

- Calcification of the aortic valve cusps leads to the restriction of the valve's mobility and it loses the ability to freely open and close (Carey & Pearce, 2013).
- The left ventricle is unable to effectively pump blood due to this obstruction. The left ventricle compensates for the increased work of pumping blood through a restrictive valve during systolic contraction and left ventricular hypertrophy ensues (Carey & Pearce, 2013).
- The left ventricle's compensatory mechanism can lead to detrimental ramifications due to the increase in afterload. These effects can include increased myocardial workload, increased oxygen consumption, and decreased supply of blood to the coronary arteries.
- Later manifestations can result in a shrunken left ventricular chamber size that contributes to decreased preload and poor systolic function (Carey & Pearce, 2013).
- The resulting symptoms of dyspnea on exertion, angina, syncope, and heart failure become apparent (Maganti et al., 2010). Progression of these symptoms can occur in a dramatically short period of time, however some patients with severe aortic stenosis may exhibit few if any symptoms of the disease.
- Replacement of the valve is the only definitive treatment. Patients with moderate to severe aortic stenosis should be evaluated by a comprehensive heart valve team for optimal management (Hull, 2012).

Pathophysiology

Calcific aortic stenosis goes beyond the process of wear and tear on the valve, and the development of calcium on the valve cusps can be segmented into two phases: the "initiation phase" and the "propagation phase" (Pawade, Newby, & Dweck, 2015, p. 561). During the initiation phase, the extremely thin valve cusps are subject to mechanical stress from turbulent blood flow and can be subject to injury. Endothelial damage causes lipoprotein(a) and oxidized low-density lipoprotein to deposit on the valve (Pawade et al., 2015). Further injury and lipid deposition creates an inflammatory response and macrophages migrate to the area. The products of cell death lead to formation of microcalcifications (Pawade et al., 2015). Formation of calcium crystals form sites for additional calcium to deposit, and in a cyclical fashion, additional inflammation occurs. In the propagation phase, collagen is deposited and the valve cusps become fibrotic and osteoblast-like cells develop. While this process may not be fully understood, one explanation involves myofibroblasts, or valve interstitial cells, differentiating into the osteoblast-like cells (Pawade et al., 2015). Ossification of the valve also requires angiogenesis and a rich blood supply provides additional macrophages and pro-inflammatory cytokines to mediate the process.

Progression of a sclerotic valve can be viewed on a continuum and can turn into hemodynamically significant aortic stenosis. Fusion of the cusps occurs due to fibrosis and calcification causing narrowing of the valve orifice (Dweck, Boon, & Newby, 2012). Blood flow out of the left ventricle is obstructed and a pressure gradient between the ventricle and the aorta is created (Ray, 2010). The left ventricle compensates for the increased work of pumping blood by increasing its myocardial muscle mass and concentrically changing its shape (Rader, Sachdev, Arsanjani, & Siegle, 2015). The myocardial microvasculature must work to keep up with the increased oxygen demanded by the ventricle to maintain cardiac output. The ventricular chamber can ultimately become smaller, which reduces the amount of preload and loss of stretch in the muscle resulting in deterioration of ventricular function. This results in an ineffective stroke volume and cardiac output. The backwards pressure in the lungs can cause pulmonary arterial hypertension (Carey & Pearce, 2013).

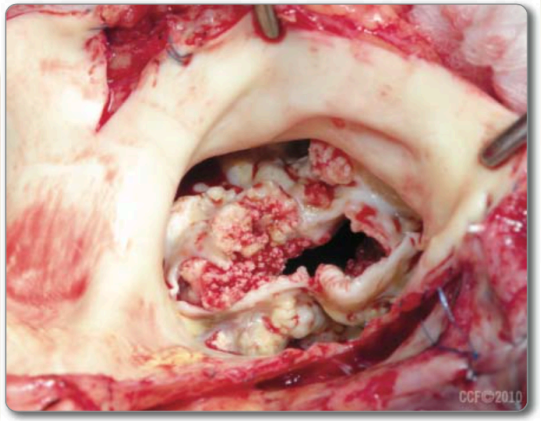

Figure 1. Severly calcified, stenotic aortic valve (Carey & Pearce, 2013)

Case Study

Mr. T is a 82 year old male with a history of hyperlipidemia, hypertension, obstructive sleep apnea, coronary artery disease with prior coronary artery bypass grafting (CABG) in 2014, atrial fibrillation, chronic kidney disease, diabetes, obesity, and is a former smoker. Mr. T reports chest tightness and dyspnea with minimal exertion. Echocardiogram reveals a normal left ventricular chamber size a preserved ejection fraction, and impaired relaxation. Aortic valve area measures 0.89 cm2. Mean gradient is 46 mm Hg. A left heart catheterization reveals patent grafts from prior CABG and pull back across the valve measures a 50 mm Hg gradient.

Mr .T's case is presented to a comprehensive heart valve team. Mr. T's aortic stenosis is classified as severe with a valve area less than 1 cm2 and pressure gradient greater than 40 (Panos & George, 2014). The decision is made to perform a transcatheter aortic valve replacement (TAVR) procedure due to his advanced age and comorbid conditions. His aortic valve is replaced with an Edwards Life Science SAPIEN valve via a right femoral arterial approach (Lachell & Henry, 2015). Mr. T's hospital course was uneventful and he is discharged home three days post procedure.

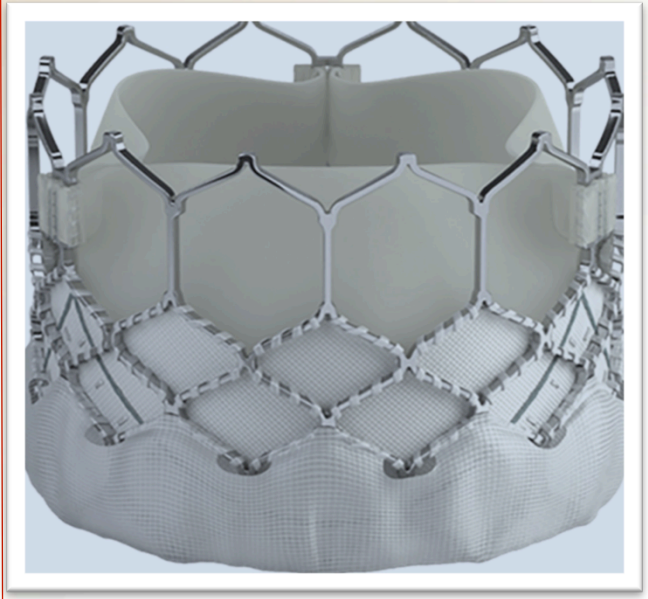

Figure 2. Edwards Life Science SAPIEN 3 aortic valve (Wöhrle et al., 2016)

Signs, Symptoms, and Diagnostic Evaluation

- poor exercise tolerance and exertional dyspnea (Maganti et al., 2010)
- Angina (Maganti et al., 2010)
- Syncope (Maganti et al., 2010)
- Classic features of heart failure: dyspnea, orthopnea, jugular vein distention, and rales (Carey & Pearce, 2013).
- crescendo-decrescendo systolic murmur can be heard along the left sternal border with radiation to the carotid arteries
- pulsus tardus (Maganti et al., 2010)
- EKG may show left ventricular hypertrophy (Maganti et al., 2010)
- Cardiomegaly on chest radiograph (Maganti et al., 2010)
- Echocardiogram is the gold standard diagnostic test and can aid in grading the severity of stenosis through Doppler by measuring the aortic jet velocity, the mean pressure gradient between the left ventricle and above the aortic valve, and measurements can be taken to determine valve area (Carey & Pearce, 2013)
- Heart catheterization can measure cardiac output, assess the aortic transvalvular gradient, and presence of coronary artery disease (Czarny & Resar, 2014)

Implications for Nursing Care

Patients with aortic stenosis are often complex and require a team approach for optimal management. APNs are integral in coordinating care and managing conditions that are associated with aortic stenosis such as hypertension, lipid abnormalities, diabetes, and obesity. Careful monitoring and treatment of these disorders may prevent secondary complications (Hull, 2012). Astute assessment of patients' symptoms can be critical in the prompt recognition of the disease with appropriate referral and optimal timing of valve replacement.

Conclusion

Calcific aortic stenosis is a progressive disease that can disrupt the quality of life for patients who suffer from symptoms of its pathology. Aortic stenosis requires early recognition and close monitoring of the disease progression to prevent a poor prognosis. The nature of the disease requires a variety of clinical specialists to assist in its treatment in order to provide the best outcomes. APNs can be central providers in diagnosing, monitoring, and providing appropriate pre and post valve replacement care.

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