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Heart Valve Disease: Classic Teaching and Emerging Paradigms

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Introduction

In the last century, the global epidemiology of valvular heart disease has evolved substantially. Rheumatic Heart Disease (RHD) was the most common cause of valvular ailment prior to World War II. In the second half of the twentieth century, however, the introduction of antibiotics and increased access to health care in industrialized countries led in a considerable decrease in the incidence of RHD. Life expectancy has grown at the same time, as has the prevalence of age-related valve problems (such as degenerative valvular disease). Degenerative valvular disease is the most common valve disease in affluent countries. In the United States, it is estimated that 2.5% of the general population, 8.5% of those 65 years-74 years of age and 13.2% of those ≥75 years of age have moderate to severe valvular disease.

For moderate to severe valvular heart disease, surgical surgery is the most common treatment. Although this alternative is costly, as the economy develops, so does access to advanced medical procedures. Brazil is an example of a country that has experienced significant growth as a result of policies implemented in the 1990s.This advancement resulted in an improvement in life expectancy. In 1988, the Brazilian public health system was established, which offers universal medical care. Between the 1980s and the 2000s, the average number of valve procedures conducted at Brazil's main public cardiovascular hospital in So Paulo climbed by 50%, from 400 to 600 per year.

As Brazil develops, it is projected that the prevalence of RHD will decline while the prevalence of degenerative valvular disease will rise. Few population-based researches, on the other hand, have looked at the burden of cardiac valvular disease in rising economies. We provide data from a population-based study conducted in Salvador, Brazil, to assess the incidence of valvular heart dysfunctions requiring surgical intervention and the most common etiologies. We also discovered risk indicators for in-hospital death after valve surgery.

Aortic Valve Disease

Aortic stenosis

Aortic stenosis is the most prevalent degenerative valve lesion in North America and Europe, affecting up to 2% of older persons with severe illness. Reduced leaflet excursion and an enhanced systolic gradient between the left ventricle and the aorta arise from calcific degeneration of a normal trileaflet or congenitally bicuspid aortic valve. Compensatory left ventricular hypertrophy causes systolic dysfunction and a mismatch in myocardial oxygen supply and demand. Angina, syncope, and heart failure are the hallmark symptoms of these conditions. The onset of symptoms heralds a rapid deterioration in prognosis, with individuals with heart failure having a median survival time of as little as two years. Sudden cardiac death is a dreaded consequence of aortic stenosis, yet it only happens in a small age of people who are truly asymptomatic. In aortic stenosis, physical examination reveals a systolic crescendo-decrescendo murmur at the base that radiates to the neck. As the disease advances, the intensity increases with handgrip, does not change with Valhalla, and peaks later. Both the murmur strength and the aortic component of S2 may soften in severe illness. The carotid pulse rises slowly and is delayed.

The first-line method for evaluating suspected aortic stenosis is Transthoracic Echocardiography (TTE). Severe stenosis is defined as a valve area of less than 1.0 cm2, a mean gradient of more than 40 mm Hg, and a peak velocity of more than 4 m/s. Gradient examination with a catheter has been linked to a high rate of subclinical stroke and should be avoided in general. A few patients have aortic stenosis symptoms and signs but have low gradients on echo. A dobutamine challenge combined with echocardiographic or invasive hemodynamic measurements can distinguish people with "pseudo stenosis" (low stroke volume due to non-severe stenosis) from those with actually severe aortic stenosis. For patients with pseudo stenosis, medical therapy for heart failure is usually sufficient, whereas patients with severe aortic stenosis require surgery. An activity test under close supervision is a viable way to elicit symptoms in asymptomatic or vaguely complaining patients. Age, male sex, hypertension, smoking, and dyslipidemia are all risk factors for aortic stenosis.

The evidence for particular medication to slow disease development has been inconclusive and disheartening. Statins, angiotensin-converting enzyme inhibitors, and bisphosphonates have all been researched, but they should not be used to prevent aortic stenosis at this time. Antihypertensive drugs are generally safe, but in patients with severe stenosis who have minimal reserve to enhance their cardiac output, hypotension must be avoided. The most obvious reason for aortic valve replacement is the start of symptoms. If the projected operative mortality is less than 1%, individuals with asymptomatic, severe aortic stenosis and unexplained reduced ejection fraction, provable symptoms, or very high peak velocity might consider surgery. Exercise testing of asymptomatic patients can indicate abnormally poor performance or high-risk characteristics. Aortic stenosis is to blame for the surgical referral. Despite traceable symptoms on chart review, up to 2 out of every 5 eligible patients are still not recommended for valve replacement. Failure to notice symptoms, senior age, and a perception of significant operative risk are all common reasons for inappropriate referral.

Mitral Valve Disease

Mitral stenosis

Mitral stenosis is a classic rheumatic fever sequel; however it is becoming less common in North America and Europe. Rheumatic valve disease and, in particular, mitral stenosis, continue to be a major public health concern around the world. Functional stenosis can be caused by severe intracardiac calcification in the elderly or people

with renal disease, although the mitral annulus, not immobile leaflets, is the primary barrier to left ventricular filling in these circumstances. Due to the development of post capillary pulmonary hypertension, patients often present with exercise intolerance and right heart failure. In the left lateral decubitus posture, physical examination indicates an opening snap and a low-pitched, diastolic rumble near the apex. The transmittal gradient and mitral valve area can be precisely measured using TTE, which is the first test of choice.

Severe mitral stenosis is defined as a mitral valve area of less than 1.0 cm2. Beta-blockers are used to improve diastolic ventricular filling time, while diuretics are used to lower left atrial pressure. Atrial fibrillation is frequent in this population, and it is linked to a 15% to 20% annual risk of embolic stroke, necessitating warfarin therapy. For valvular atrial fibrillation, newer anticoagulants have not been investigated or authorized. All patients under the age of 40 with rheumatic caritas should be seriously considered for chronic penicillin therapy to prevent recurring acute rheumatic fever. Percutaneous balloon valvuloplasty, which has an excellent success rate and a low risk of complications in skilled hands, should be considered for patients with at least moderate mitral stenosis, New York Heart Association class II-IV symptoms, and favorable morphology. With substantial pulmonary hypertension or new atrial fibrillation, a lower threshold is feasible. Valve replacement may be required if there is moderate-to-severe mitral regurgitation or substantial leaflet calcification.

Mitral Regurgitation

A structural defect of the valve, such as leaflet prolapse or endocarditis, causes primary mitral regurgitation. Secondary (functional) regurgitation is caused by dilated cardiomyopathy or papillary muscle tethering following a heart attack. Mild mitral regurgitation is extremely frequent, according to population studies, and usually does not require treatment. The left ventricle, on the other hand, responds to severe mitral regurgitation by dilatation, eccentric hypertrophy, and systolic failure. Pulmonary hypertension is caused by elevated left atrial pressures. Exceptional dyspnea and symptoms/signs of left ventricular failure develop in patients. A pan systolic, blown murmur at the apex may extend to the back or precordium with eccentric jets, according to physical examination. Mitral valve repair or replacement should be considered if symptoms or signs of left ventricular dysfunction (ejection fraction 60% or end-systolic diameter 40 mm) are present. Valve repair has a lower long-term morbidity and death rate than valve replacement, owing to the preservation of left ventricular shape and the avoidance of the thromboembolic risk associated with prosthesis. Because continuous alterations from ventricular dilation may render the original repair insufficient, older patients with ischemic heart disease and significant secondary mitral regurgitation may benefit from chord-sparing valve replacement.

There is debate on when asymptomatic patients with severe mitral regurgitation and normal left ventricular systolic function should be referred for surgery. Asymptomatic patients with severe mitral regurgitation responded well with close follow-up and guideline-based surgical referral in one well referenced prospective trial. Others argue for asymptomatic patients to be referred early based on quantitative signs of severe mitral regurgitation, such as effective regurgitate orifice area and left atrial volume index. Early repair was linked to better 7-year survival in a retrospective, propensity-matched research. As a result, we prefer early treatment for patients with a high possibility of repair and low surgical risk. We employ exercise echocardiography in inactive patients to search for inducible pulmonary hypertension or the onset of symptoms at a low exertion. Quantitative echocardiograms should be performed every 6 months-12 months for truly asymptomatic patients to detect left ventricular dilatation, systolic dysfunction, or pulmonary hypertension. In individuals with severe mitral regurgitation, CMR can detect subclinical left ventricular dysfunction and predict ventricular dysfunction after surgery. In certain patients, it should be considered.

The use of trans catheter methods to treat mitral regurgitation is being studied. The best studied of them is the MitraClip (Abbott Laboratories, Abbott Park, Ill.), which is a clip that approximates opposing mitral leaflets, resulting in a narrower, double-orifice valve. There was a significant improvement in left ventricular volume, ejection fraction, and quality of life in an initial randomized trial for patients at high risk for conventional surgery. Nonetheless, the amount of improvement was smaller than that of surgery, and 20% of the participants required valve surgery within a year. In persistent severe mitral regurgitation, there is minimal evidence for effective medical therapy. Small trials using vasodilators failed to show that they improved hemodynamics or reduced the need for surgery. For concurrent heart failure, standard medication is appropriate, but surgery remains the cornerstone of treatment. Patients with functional mitral regurgitation may benefit from cardiac resynchronization.