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Management of Aortic Regurgitation: A Review Article

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1. Abstract

1.1. Introduction: Aortic regurgitation occurs due to incomplete closure of the aortic valve leaf which can result from abnormalities in the aortic valve, valve support structures (aorta and annulus), or both. Diseases that often affect the valve leaflets include aortic bicuspid, and other congenital disorders, degeneration of aortaclerosis, infective endocarditis, rheumatic heart disease, connective or inflammatory tissue disease, antiphospholipid syndrome. Based on the time of occurrence, chronic aortic regurgitation and chronic aortic regurgitation can be distinguished.

1.2. Discussion: The main goal of medical therapy is to reduce systolic hypertension associated with severe chronic AR, thereby reducing stress on the ventricular wall and improving left ventricular function. Treatment options include ACE inhibitors, ARBs, or Ca blockers. ACE inhibitors are also an option where there is continued left ventricular dysfunction after surgery, as well as in patients who have contraindications for surgery.

1.3. Conclusion: After symptoms appear, the mortality rate in patients without surgery treatment can reach 10-20% per year, whereas in patients with chronic severe regurgitation without symptoms and normal left ventricular function, the mortality is quite low. The management of aortic regurgitation is importance based on condition.

2. Introduction

Aortic regurgitation occurs due to incomplete closure of the aortic valve leaf which can result from abnormalities in the aortic valve, valve support structures (aorta and annulus), or both. Diseases that often affect the valve leaflets include aortic bicuspid, and other congenital disorders, degeneration of aortaclerosis, infective endocarditis, rheumatic heart disease, connective or inflammatory tissue disease, antiphospholipid syndrome. Based on the time of occurrence, chronic aortic regurgitation and chronic aortic regurgitation can be distinguished [1].

The prevalence of chronic aortic regurgitation is much higher than acute aortic regurgitation, and it has a different etiology. In a study conducted by Dujardin, it was found that 35% had no known cause, 26% idiopathic aortic root dilatation, 13% congenital abnormalities, 12% rheumatic heart disease, 10% infective endocarditis, and 7% degenerative valve disease. These figures are only rough estimates because demographic changes in population age, geographic location and dense socioeconomic status affect the prevalence of diseases, such as rheumatic heart disease [2].

Aortic regurgitation places a considerable volume load on the left ventricle. With each contraction, the ventricles must be able to excrete the same amount of blood as the normal stroke volume plus the regurgitant volume. The left ventricle was severely dilated and eventually hypertrophied, resulting in a spherical shape. This increased tensile strength of the ventricles will allow for an increase in diastolic volume without an increase in diastolic pressure [3].

The high compensatory ability of the left ventricle accompanied by a competent mitral valve can maintain left ventricular function for a long period of time. Symptoms rarely appear before decompensation of the heart, which can sometimes be accompanied by regurgitation of the functional mitral valve. Until one day there can be decompensation. It is difficult to determine when this phase began. Dypsnea on exertion is an early symptom that is often seen in conditions of chronic severe aortic regurgitation, usually due to increased ventricular pressure at the end of the diastolic end of activity. Often patients do not notice a gradual decrease in exercise capacity due to the slow progression of the disease. Patients with acute aortic regurgitation have a poor prognosis. Patients with severe chronic regurgitation with symptoms also have a poor long-term prognosis [3]. Purpose of this article to review management of aortic regurgitation. The management of aortic regurgitation is importance based on condition.

3. Discussion

3.1. Etiology

Diseases that often cause abnormalities in the aortic ring or base include idiopathic dilatation of the aortic root, artoannular ectation, Marfan's syndrome, Ehler Danlos syndrome, osteogenesis imperfecta, aortic dissection, syphilitic aortitis or various connective tissue diseases. The most common causes of aortic dilatation are arterosclerosis and medial necrosis. Acute aortic regurgitation is most often caused by bacterial endocarditis, aortic dissection, or blunt chest trauma. Other less common causes include nonbacterial endocarditis, aortic laceration and complications from invasive procedures such as aortic valvuloplasty and percutaneous balloon dilatation. Acute aortic regurgitation has a poor prognosis [5].

Regarding rheumatic heart disease itself, valve abnormalities are more common in the mitral valve. About 60 percent of valve disease involvement in rheumatic heart disease is mitral stenosis. For aortic regurgitation alone, it is more often the result of endocarditis than rheumatic heart disease [5].

3.2. Pathophysiology and Symptoms

Aortic regurgitation causes reflux of blood from the aorta into the left ventricle during ventricular relaxation. In principle, the peripheral tissues and the left ventricle compete to get blood out of the ventricles during systolic. The amount of forward blood flow to the periphery of the retrograde flow to the ventricles depends on the degree of valve closure and the relative resistance to blood flow between the peripheral vessels and the ventricles. Peripheral vascular resistance is usually low in aortic insufficiency. This is a compensation to maximize blood flow to the future. However, at an advanced stage peripheral resistance will increase, thereby also increasing retrograde flow through the aortic valve and accelerating disease progression [1, 6].

Aortic regurgitation places a considerable volume load on the left ventricle. With each contraction, the ventricles must be able to excrete the same amount of blood as the normal stroke volume plus the regurgitant volume. The left ventricle was severely dilated and eventually hypertrophied, resulting in a spherical shape. This increased tensile strength of the ventricles will allow for an increase in diastolic volume without an increase in diastolic pressure. The high compensatory ability of the left ventricle accompanied by a competent mitral valve can maintain left ventricular function for a long period of time. Symptoms rarely appear before decompensation of the heart, which can sometimes be accompanied by regurgitation of the functional mitral valve. Until one day there can be decompensation. It is difficult to determine when this phase began [7].

Dypsnea on exertion is an early symptom that is often seen in conditions of chronic severe aortic regurgitation, usually due to increased ventricular pressure at the end of the diastolic end of activity. Often patients do not notice a gradual decrease in exercise capacity due to the slow progression of the disease. Palpitations happen in some patients due to increased pulse pressure are the initial complaints that lead to the diagnosis of aortic regurgitation. Chest pain may occur even though there is no coronary atherosclerosis, this is due to increased myocardial perfusion pressure, increased myocardial oxygen demand, and decreased ratio of coronary arteries to myocardial mass. Syncope or sudden death may occur with aortic regurgitation, although rarely [8].

Whereas in acute aortic regurgitation, the ventricle of a normal size will receive a large volume of regurgitation suddenly, so it does not have time to adjust to the volume excess load. This was also accompanied by a sudden decrease in stroke volume. Cardiac compensation in the form of tachycardia may mitigate a little of the problem of decreased stroke volume, but it is often insufficient to maintain cardiac output so that the patient can fall into cardiogenic shock. Pulmonary edema occurs as a result of increased filling pressure at the end of the left ventricular diastolic accompanied by an increase in pulmonary venous pressure. In addition, as the left ventricular end-diastolic pressure approaches the aortic and coronary artery diastolic pressures, the subendocardial myocardial perfusion pressure decreases, whereas oxygen demand increases due to the greater afterload effect and compensatory tachycardia itself. The blood flow needed to the coronary will suddenly decrease and cause subendocardial ischemia [9].

3.3. Clinical Signs

On auscultation of a patient with mild or moderate chronic regurgitation of the aorta, a high-frequency, de-crescendo diastolic murmur develops which is usually heard in the third or fourth rib space at the border of the left sternum. The loudness of the murmur is related to the severity of the disease to some extent. In severe regur-

gitation, there is also a systolic murmur due to an increase in stroke volume which is often heard more clearly than a diastolic murmur. In some patients, mid- and late-diastolic gurgling (Austin-flint murmur) may occur, this occurs due to vibrations of the anterior mitral valve leaflets as a result of impact with the posterior aortic regurgitation jet. On auscultation, a weak second heart sound and a third heart sound were also found. On another physical examination may reveal dilated blood pressure, and peripheral signs. What occurs due to the action of hyperdynamic mycocardium and low peripheral resistance. The manifestations that appear can be as [9]:

1. Waterhammer pulse or corrigan pulse which is characterized by a rapid filling and emptying pulse.

2. Pulses such as gunshot or Durozies noise heard in the femoral artery.

3. Quinke capillary pulsation, which is seen as a discoloration of the tiny blood vessels at the base of the nail that turn red and turn pale.

4. A systolic protrusion in the head that occurs when a blood vessel collapses in the neck and fills rapidly. (de musset sign).

In accordance with the hemodynamic differences in acute and chronic aortic regurgitation, the signs seen in acute aortic regurgitation are different. Physical signs characteristic of chronic volume load often change and disappear in acute regurgitation, and this causes medical personnel to be unaware of the severity of regurgitation.Due to the acute hemodynamic disturbance, patients with acute aortic regurgitation often have tachycardia and tachypnea, diastolic murmurs are often less audible, can become slow and short due to the rapid increase in left ventricular diastolic pressure, which reduces the pressure gradient between the aorta and ventricles. The loss of a second heart sound may be a clue in clinical conditions of cardiogenic shock and pulmonary edema [1, 7].

The reduction in acute stroke volume, leading to a decrease in systolic pressure, and the adjustment of diastolic pressure by increasing the left ventricular diastolic pressure, consequently no increase in pulse pressure, is the reason for the absence of signs of perfier in acute aortic regurgitation [8].

3.4. Course of the Disease

In uncorrected aortic regurgitation, deterioration may occur. The American Heart Association stages the course of aortic regurgitation. Apart from being useful for assessing severity, this stage is also useful in determining the therapy to be performed, whether it is done medically or surgically [10].

Table 9. Stages of Chronic AR

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of AR	• Bicuspid aortic valve (or other congenital valve anomaly) • Aortic valve sclerosis • Diseases of the aortic sinuses or ascending aorta • History of rheumatic fever or known rheumatic heart disease • IE	• AR severity: none or trace	• None	• None
B	Progressive AR	• Mild-to-moderate calcification of a trileaflet valve bicuspid aortic valve (or other congenital valve anomaly) • Dilated aortic sinuses • Rheumatic valve changes • Previous IE	• Mild AR: ○ Jet width <25% of LVOT; ○ Vena contracta <0.3 cm; ○ RVol <30 mL/beat; ○ RF <30%; ○ ERO <0.10 cm²; ○ Angiography grade 1+ • Moderate AR: ○ Jet width 25%–64% of LVOT; ○ Vena contracta.03–0.6 cm; ○ RVol 30–59 mL/beat; ○ RF 30%–49%; ○ ERO.010–0.29 cm²; ○ Angiography grade 2+	• Normal LV systolic function • Normal LV volume or mild LV dilation	• None
C	Asymptomatic severe AR	• Calcific aortic valve disease • Bicuspid valve (or other congenital abnormality) • Dilated aortic sinuses or ascending aorta • Rheumatic valve changes • IE with abnormal leaflet closure or perforation	• Severe AR: ○ Jet width ≥65% of LVOT; ○ Vena contracta >0.6 cm; ○ Holodiastolic flow reversal in the proximal abdominal aorta ○ RVol ≥60 mL/beat; ○ RF ≥50%; ○ ERO ≥0.3 cm²; ○ Angiography grade 3+ to 4+; ○ In addition, diagnosis of chronic severe AR requires evidence of LV dilation	C1: Normal LVEF (≥50%) and mild-to-moderate LV dilation (LVESD ≤50 mm) C2: Abnormal LV systolic function with depressed LVEF (<50%) or severe LV dilatation (LVESD >50 mm or Indexed LVESD >25 mm/m²)	• None; exercise testing is reasonable to confirm symptom status
D	Symptomatic severe AR	• Calcific valve disease • Bicuspid valve (or other congenital abnormality) • Dilated aortic sinuses or ascending aorta • Rheumatic valve changes • Previous IE with abnormal leaflet closure or perforation	• Severe AR: ○ Doppler jet width ≥65% of LVOT; ○ Vena contracta >0.6 cm; ○ Holodiastolic flow reversal in the proximal abdominal aorta; ○ RVol ≥60 mL/beat; ○ RF ≥50%; ○ ERO ≥0.3 cm²; ○ Angiography grade 3+ to 4+; ○ In addition, diagnosis of chronic severe AR requires evidence of LV dilation	• Symptomatic severe AR may occur with normal systolic function (LVEF ≥50%), mild-to-moderate LV dysfunction (LVEF 40%–50%), or severe LV dysfunction (LVEF <40%); • Moderate-to-severe LV dilation is present	• Exertional dyspnea or angina or more severe HF symptoms

Figure 1: Stages of Chronic AR [10]

3.5. Electrocardiography (EKG)

Not really relevant, but left ventricular hypertrophy can usually be found. The strain pattern on the ECG correlates with the dimensions of the mass and pressure on the abnormal left ventricular wall [11].

• Chest X-ray

Shows enlarged cardiac silhouette due to left ventricular dilation

• Echocardiography

Is the most important diagnostic tool.

TEE is indicated in patients with signs or symptoms of AR for accurate diagnosis of the cause of regurgitation, severity, size and function of systolic ventricular envy, as well as determining clinical outcome and timing of intervention.

• Exercise testing

May help identify patients with left ventricular early systolic dysfunction

• Cardiac catheterization

Assess hemodynamics, coronary artery anatomy, and the severity of AR if echocardiography is still in doubt

3.6. Management

1. Medical

The main goal of medical therapy is to reduce systolic hypertension associated with severe chronic AR, thereby reducing stress on the ventricular wall and improving left ventricular function. Treatment options include ACE inhibitors, ARBs, or Ca blockers. ACE inhibitors are also an option where there is continued left ventricular dysfunction after surgery, as well as in patients who have contraindications for surgery [12, 13].

Based on the 2014 American Heart Association guidelines on prevention of infective endocarditis, prophylactic antibiotics are only recommended in patients who are at high risk of developing IE [10].

2. Surgery

On the basis of the American Heart Associaton the indications for aortic valve replacement are as follows [10, 14]:

1. Patients with severe AR symptoms are relieved of left ventricular systolic function (stage D)

2. Asymptomatic patient with chronic severe AR and left ventricular systolic dysfunction (LVEF <50%) at rest (stage C2), if no other cause of systolic dysfunction has been identified

3. Patients with severe AR (stage C or D) undergoing cardiac surgery for other indications

Aortic valve replacement is reasonable for:

1. Asymptomatic patient with severe AR and normal left ventricular systolic function (LVEF> 50%), but with severe left ventricular dilatation (LVESD> 50% mm or LVESD index> 25 mm / m2)

2. Patients with AR are currently undergoing surgery on ascending aorta, CABG or mitral valve surgery.

The following is a flowchart of the treatment selection along with the recommended levels of the AHA.

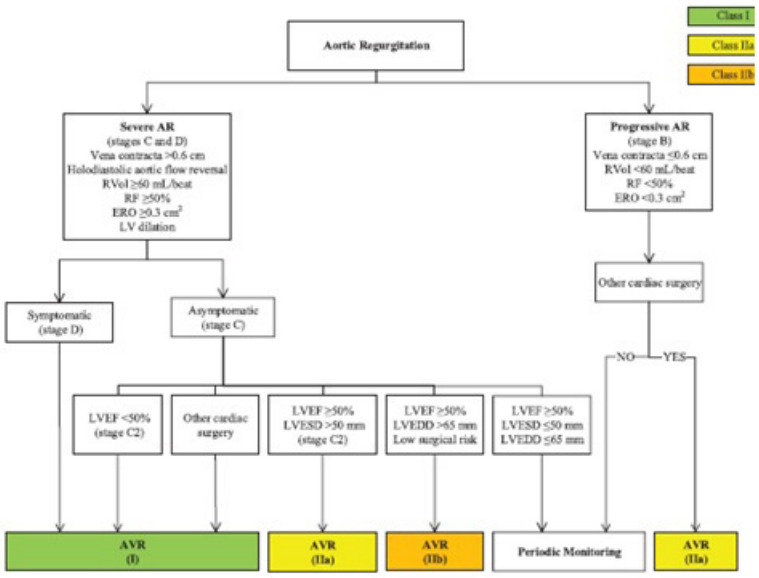

Figure 2: Management of AR [10]

4. Conclusion

Patients with acute aortic regurgitation have a poor prognosis. Patients with severe chronic regurgitation with symptoms also have a poor long-term prognosis. After symptoms appear, the mortality rate in patients without surgery treatment can reach 10-20% per year, whereas in patients with chronic severe regurgitation without symptoms and normal left ventricular function, the mortality is quite low. The management of aortic regurgitation is importance based on condition.

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