Acute Aortic Regurgitation
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Aortic regurgitation (AR) is characterized by regurgitation of blood from the aorta to the left ventricle (LV) during diastole and is attributable to diverse congenital and acquired abnormalities of the aortic valve or the wall of the aortic root. AR can be either chronic or acute. The classic features of chronic AR have been known to clinicians for nearly 2 centuries. Corrigan described chronic AR in 1832 in his text “On Permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves.”1 Patients with chronic AR remain asymptomatic for many years as the LV becomes gradually enlarged; cardiac symptoms and clinical congestive heart failure then develop. On the other hand, acute severe AR, if untreated, leads to advanced heart failure and early death. Acute severe AR may be difficult to recognize clinically and is often erroneously diagnosed as another acute condition such as sepsis, pneumonia, or nonvalvular heart disease. Acute or subacute infective endocarditis, aortic dissection, and aortic valve damage caused by trauma are known causes of acute AR. We present 2 cases of acute AR (case 1, infective endocarditis; case 2, Stanford type A aortic dissection), and we propose management plans for each case (Figure 1A and 1B).

Case 1
A 23-year-old man admitted to an intensive care unit with Staphylococcus bacteremia presented with soft heart sounds and a to-and-fro murmur, which progressed to a silent precordium within 24 hours. Bedside transthoracic 2-dimensional and M-mode echocardiogram (TTE) and transesophageal echocardiogram (TEE) revealed aortic valve vegetations, severe AR, premature mitral valve closure, and diastolic and early systolic mitral regurgitation, indicative of acute left heart failure necessitating ventilatory support and urgent replacement of the aortic valve and aortic root with a homograft (Figure 2).

Acute AR results in the development of a distinct syndrome, the pathophysiological features of which differ from those of chronic AR (Figure 3A and 3B).2 Acute severe AR imposes a sudden excessive volume load on an unprepared LV that is normal in size, resulting in a dramatic rise in LV diastolic pressure (LVDP), which may approach or indeed equal the aortic diastolic pressure. Because LV pressure exceeds the left atrial pressure during diastole, the resulting rapid ventriculoatrial gradient causes the mitral valve to close prematurely before the onset of the next systole (Figures 2G and 3A). The premature mitral valve closure is beneficial in the sense that the high LVDP is not transmitted to the pulmonary venous system, thus preventing pulmonary edema and clinical left heart failure. However, the protection afforded by premature mitral valve closure is lost when a further rise in the ventriculoatrial gradient opens the mitral valve in late diastole, leading to diastolic mitral regurgitation. Mitral regurgitation in acute AR may occur either in diastole or in systole (when the LVDP exceeds the left atrial pressure) (Figure 2E, 2F, and 2H). It is likely that persistence of the ventriculoatrial gradient, as a result of extension of the high LVDP level to the isovolumic contraction period and the early systole, causes the mitral valve to open during this period, resulting in early systolic mitral regurgitation. Mitral regurgitation is usually effective to lower LVDP; the left atrium serves as a reservoir for blood regurgitated from the aorta to the LV. However, left atrial pressure may rise further, leading to pulmonary edema and circulatory failure, as in our patient.

Premature mitral valve closure is present when AR is both acute and...
severe, and it is best demonstrated by M-mode echocardiography with simultaneous electrocardiography. Premature mitral valve closure is a specific and sensitive noninvasive indicator of acute severe AR, and the extent of premature mitral valve closure has been correlated with the degree of rise in LVDP. Normally, the mitral valve does not close until shortly after the onset of LV contraction; leaflet closure occurs 40 ms after the onset of the QRS complex. Premature mitral valve closure is mild (grade I) when coaptation of the anterior and posterior mitral leaflets occurs at or before the initial inscription of the QRS (ie, up to 50 ms before the Q wave but after the P wave); premature mitral valve closure is very marked (grade II) when the mitral valve closes very prematurely, up to 200 ms before the Q wave. Such patients, in comparison with those with grade I premature mitral valve closure, exhibit extreme elevations in LVDP and volume, and may be only marginally compensated (Figure 1A).

A prompt and accurate diagnosis of acute AR is of great importance, because urgent or emergent aortic valve surgery is life-saving. Such patients

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**Figure 1.** Proposed management plan for acute aortic regurgitation (AR) due to infective endocarditis (A) and aortic dissection (B). CHF indicates congestive heart failure; TTE, transthoracic echocardiogram; TEE, transesophageal echocardiogram; IE, infective endocarditis; AR, aortic regurgitation; PMVC, premature mitral valve closure; DMR, diastolic mitral regurgitation; ESMR, early systolic mitral regurgitation; ICU, intensive care unit; EKG, electrocardiogram; LVOT, left ventricular outflow tract; and AV, aortic valve.
are critically ill but show minimal or no clinical signs of AR. Thus, the classic decrescendo diastolic murmur and the eponymous classical peripheral arterial signs may be lacking. An early diastolic murmur is often heard, but, compared with that of patients with chronic AR, the murmur is softer and shorter. This, together with the presence of a short and soft systolic murmur, often results in development of a soft to-and-fro murmur. Occasionally, such murmurs are absent. These findings, combined with a soft or absent first heart sound (S1) or A2 (aortic component of second heart sound), sometimes create a silent precordium. S4 and the presystolic component of the Austin Flint murmur are absent, but a short mid-diastolic component of the Austin Flint murmur is often present. Auscultation signs can be confusing, because it is difficult to distinguish diastole from systole. This is because the heart sounds are soft or absent, and diastole becomes shorter than systole. The mitral valve closes prematurely and opens late, because of prolongation of the systolic ejection time caused by volume and pressure loading of the LV. This leads to a reversal of the normal relationship between systole and diastole.

How can the characteristic cardiac auscultations be explained? First, premature mitral valve closure is responsible for softness or absence of the S1 because the mitral valve is closed at the beginning of LV systole. A short early diastolic murmur and a short mid-diastolic murmur (terminating with a soft premature mitral valve closure sound) produce a palpable summation gallop. The mid-diastolic portion of the Austin Flint murmur is not affected by premature mitral valve closure; the short diastolic filling time and the high left atrial pressure are accompanied by turbulent blood flow through the mitral valve. The flow dynamics are similar to those associated with mitral stenosis.

Second, A2 is soft because of destruction of valve tissue and impairment of the valve closure mechanism. Third, the soft systolic murmur is attributable to increased flow through the aortic valve and/or development of diastolic mitral regurgitation and early systolic mitral regurgitation.

Patients with infective endocarditis are at risk of developing acute and severe AR that can be detected by TTE and TEE. Aortic valve surgery may be timed with reference to whether the premature mitral valve closure is mild or severe. The use of echocardiogra-
phy permits AR severity to be graded and facilitates medical and surgical management of such patients. We propose that AR patients exhibiting grade II premature mitral valve closure require urgent aortic valve replacement. Patients with grade II premature mitral valve closure and mitral regurgitation should undergo emergent aortic valve replacement. Surgery in these 2 groups of patients should proceed regardless of infection status and without waiting for completion of antibiotic therapy. Patients with grade I premature mitral valve closure and who do not show clinical heart failure can be managed by medical therapy; the decision on early aortic valve replacement should include consideration of both relative hemodynamic severity (as judged by physical examination and echo-Doppler findings) and the severity and extent of infection (including intra- and extracardiac complications (Figure 1A).

Case 2
A 59-year-old man presented to the emergency department with chest and back pain and syncope. A chest x-ray revealed a widened mediastinum, and a diagnosis of Stanford type A dissection was confirmed by computed tomography of the chest. The dissection flap originated at the level of the aortic valve and involved the entire thoracic aorta, including the ascending aorta, the aortic arch, and the origins of the innominate, left common carotid, and left subclavian arteries. TTE and intraoperative TEE revealed severe AR, a dissection flap at the level of the non-coronary cusp of the aortic valve, and premature mitral valve closure (Figure 4). Emergent surgery was performed with the aortic root and aortic valve replacement by use of a St Jude composite valve conduit, with coronary reimplantation (Bentall procedure), followed by an uneventful recovery.
Acute type A aortic dissection is an uncommon but catastrophic acute event with an annual incidence of 3 to 4 cases per 100,000 of the general population in the United Kingdom and the United States; preadmission mortality is 20%, and inpatient mortality is 30%. Early-stage mortality is as high as 1% to 2% per hour over the first several hours, but survival levels have improved in recent years. Clinical recognition is now prompt; definitive diagnostic testing with the use of TEE and/or chest computed tomography is available; and emergent surgery has become established. AR is an important complication of proximal aortic dissection; an AR murmur is detected in 16% to 67% of all such patients. Intraoperative TEE is indispensable to demonstrate the mechanism of AR and to facilitate the choice among aortic valve surgical procedures, resuspension, or replacement. These mechanisms include dilatation of the aortic root and annulus (Figure 5A), pressure of a false lumen on 1 cusp causing asymmetrical cusp coap-

Figure 3. Schematic representations contrasting the hemodynamic, echocardiographic, and phonocardiographic manifestations of acute severe (A) and chronic severe (B) AR. Ao indicates aorta; AR, aortic regurgitation; EDP, end-diastolic pressure; LV, left ventricle; LA, left atrium; AML, anterior mitral leaflet; PMC, posterior mitral leaflet; ECHO, echocardiogram; PCG, phonocardiogram; C, mitral valve closure; S1, first heart sound; S2, second heart sound; SM, systolic murmur; and DM, diastolic murmur.

Figure 4. Severe AR (A) and dissection flap at the level of non coronary cusp of the AV (B). AR indicates aortic regurgitation; AV, aortic valve; and TTE, transthoracic echocardiogram.
tation (Figure 5B), flail of an aortic cusp attributable to annular support disruption (Figure 5C), and prolapse of a mobile intimal flap through the aortic valve (Figure 5D).4

In summary, patients with infective endocarditis and aortic dissection are at risk of developing acute severe AR that can be detected with the aid of TTE and/or TEE. These techniques permit AR severity to be graded and facilitate appropriate medical and surgical management of patients with acute AR (Figure 1A and 1B).

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None.

References

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