

CLINICAL VIGNETTE

Clinical Review: Acute Aortic Regurgitation

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Case Report

A 66-year-old man with history of hypertension, aortic stenosis, and aortic valve replacement 7 years earlier with a Freestyle prosthetic heart valve (PHV), presented with sudden onset of chest pressure worse with deep breathing. This was associated with progressively worsening shortness of breath.

Upon arrival in the emergency department, his pulse was 66 beats per minute and blood pressure was 126/62 mmHg. On exam, he was found to have bounding carotid pulses. He had regular rhythm. There was an III/VI early diastolic decrescendo murmur over the precordium, loudest at the right upper sternal border.

Chest x-ray on admission showed mild cardiomegaly with pulmonary artery cephalization, consistent with pulmonary hypertension. ECG showed normal sinus rhythm with non-specific ST-T wave changes. Bedside echocardiography demonstrated torrential aortic regurgitation due to a flail aortic valve leaflet, as well as moderate mitral regurgitation (Figure 1, 2). The left ventricle was hyperdynamic with ejection fraction of 70-75%. Right ventricular systolic pressure was estimated at 70-75 mmHg. The patient reported an echocardiogram several months prior showing mild regurgitation of his prosthetic heart valve.

The patient was started on nitroglycerin and dobutamine and blood cultures drawn, and he was taken to the catheterization lab for further evaluation. He was found to have elevated left-ventricular end-diastolic pressure 21 mmHg with pulmonary artery pressure 44/25 mmHg. Aortogram (Figure 3) demonstrated severe aortic regurgitation.

Cardiothoracic surgery was consulted, and he underwent surgery on the third hospital day. He was found to have degenerative changes of the Freestyle PHV with a tear of the aortic valve leaflet. He underwent aortic valve replacement with a pericardial bioprosthesis. He was also found to have severe mitral regurgitation due to A2 prolapse and underwent mitral valve repair. His course was complicated by atrial fibrillation which resolved with use of amiodarone.

Discussion of Acute Aortic Regurgitation (AAR)

Mechanism

AAR of native aortic valves is most commonly caused by infective endocarditis, aortic dissection, or trauma. With PHV, AAR may be caused by these, as well as structural valve deterioration, valve dehiscence, or valve thrombosis (Table 1). Structural valve deterioration refers to degenerative changes in prosthetic heart valves. This may lead to valve stenosis or regurgitation. The Freestyle PHV, used in this patient, is a porcine aortic root with anti-calcification treatment that can be used for aortic valve replacement. These PHVs are subject to structural valve deterioration. The most common mode of presentation is AAR due to leaflet tear, occurring in 77.8% of patients with structural valve deterioration.¹ Valve regurgitation is also common after transcatheter aortic valve replacement, with moderate to severe regurgitation occurring in 12.2% of patients.²

Pathophysiology of AAR

AAR presents with severe hemodynamic changes. Although preload is increased, leading to increased stroke volume, the regurgitation directly decreases forward cardiac output. In chronic aortic regurgitation, left ventricular hypertrophy and dilation allow the left ventricle to adapt to increased left ventricular diastolic volume. In acute regurgitation, there is no time for these changes and increased left ventricular diastolic volume leads to increased left ventricular diastolic pressure. This has several important consequences. First, the decrease in diastolic transmural gradient leads to decreased coronary perfusion, causing global left ventricular ischemia and decreased myocardial performance. ECG may show ischemic ST-segment changes.³ Anginal chest pain may be present. Second, increased left ventricular end-diastolic pressure may lead to left atrial hypertension and subsequent pulmonary edema. Ensuing hypoxemia may further contribute to coronary ischemia and worsening myocardial function.

Presentation of AAR

The most common presenting symptoms of AAR include shortness of breath and weakness. In patients with endocarditis, fever may be present. In patients with chest pain, particularly radiating to the back, aortic dissection should be suspected. Because of the hemodynamic changes outlined above, patients may present with cardiogenic shock.

In chronic aortic regurgitation, a wide pulse pressure is caused by an enlarged left ventricle that produces a large stroke volume and allows for substantial regurgitation. This leads to the well-known physical examination findings seen in chronic aortic regurgitation (eg, Corrigan’s sign, deMusset’s sign, etc.). In AAR, physical examination findings may be subtle. Tachycardia is the most common finding. If cardiogenic shock is present, the extremities may be cool and cyanotic. S1 may be soft due to premature mitral valve closure. Because of the rapid increase in left ventricular diastolic pressures, the difference between the aortic and ventricular pressure may narrow quickly, leading to a brief and low-pitched murmur. The diastolic rumble of the Austin-Flint murmur may be present if the aortic regurgitation jet impinges on the anterior valve leaflet, causing decreased leaflet excursion. Pulmonary edema may lead to pulmonary rales, and hypoxemia may be present.

Evaluation and Treatment

Patients with suspected acute aortic regurgitation should undergo echocardiographic evaluation. This can accurately diagnose acute aortic regurgitation, as well as provide evaluation of left ventricular systolic function, concomitant valvular lesions, and estimation of pulmonary artery pressure. In patients where aortic dissection is suspected, imaging with CT, MRI, or TEE is necessary. There should be a low index of suspicion for bacterial endocarditis. Blood cultures should be drawn prior to administration of antibiotics and should be repeated every 12 hours for 3 sets.

Acute aortic insufficiency is a surgical emergency. Timely surgical consultation is necessary. Treatment aimed at stabilizing the patient can be initiated pending definitive surgical treatment. Treatment with inotropic agents is useful in maintaining stroke volume and cardiac output. Furthermore, dobutamine and milrinone increase chronotropic response. At higher heart rates, diastolic time is decreased, potentially leading to less aortic regurgitant volume. For this reason, negative chronotropic agents such as beta-blockers and non-dihydropyridine calcium channel blockers should be avoided. Inotropes and vasopressors should be used with caution in the setting of aortic dissection.

In chronic aortic regurgitation, afterload reduction has been demonstrated to improve cardiac output, decrease left ventricular end-diastolic pressure, and regurgitant volume.^{4,5} This can be extrapolated to AAR. In this case, nitroglycerin was used and pulmonary artery pressures significantly improved in the brief period of time between echocardiogram and cardiac catheterization.

Treatment of AAR in the setting of infective endocarditis presents particular challenges. Early surgery presents the risk of implantation of a PHV into an aortic root with active

infection, increasing the risk of PHV endocarditis. However, delays in surgery may lead to further hemodynamic compromise. Furthermore, patients may have embolic lesions and sepsis, causing multi-system organ damage.

Conclusions

Acute aortic regurgitation often presents with cardiogenic shock and should be regarded as a surgical emergency. A high index of suspicion is needed because the signs and symptoms may be subtle. Echocardiography is a readily available, non-invasive imaging modality, which should be used early in any patient presenting with the signs and symptoms of cardiogenic shock. Aortic dissection and IE are important causes of AAR. PHVs put patients at unique risk for AAR. Medical management of AAR is limited and should be used as a temporizing measure prior to definitive surgical treatment.

Table 1: Causes of Acute Aortic Regurgitation

<i>Native Aortic Valve</i>	-Infective Endocarditis -Aortic Dissection -Trauma -Rupture of congenitally fenestrated valve leaflet -Iatrogenic (coronary angiography, balloon aortic valvuloplasty)
<i>PHV</i>	-Iatrogenic (TAVR) -Structural valve deterioration -Valve Dehiscence -Post-transcatheter aortic valve replacement (TAVR)

Figure 1: Parasternal long-axis view of the aortic valve showing a torn aortic valve cusp (arrow).

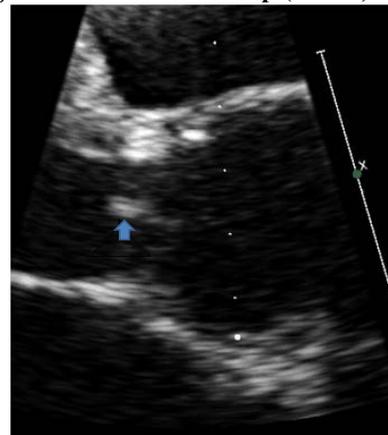


Figure 2: Parasternal long-axis view with color Doppler demonstrating severe aortic insufficiency.

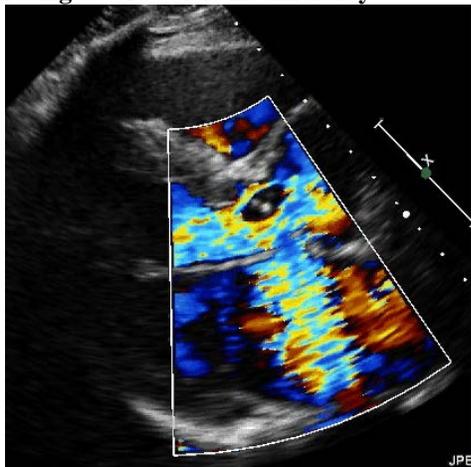
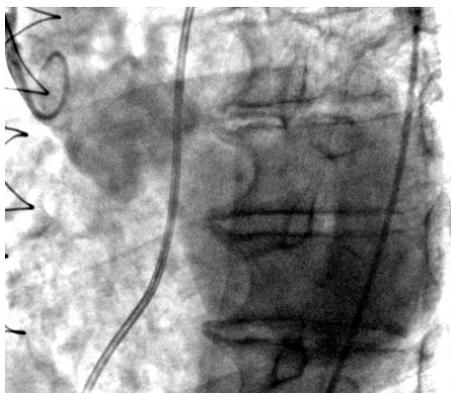


Figure 3: Aorogram in RAO projection showing opacification of the left ventricle equal to that of the aorta within one beat of injection. This is consistent with 4+ aortic regurgitation.



3. **Stout KK, Verrier ED.** Acute valvular regurgitation. *Circulation.* 2009 Jun 30;119(25):3232-41. doi: 10.1161/CIRCULATIONAHA.108.782292. Review. PubMed PMID:19564568.
4. **Bolen JL, Alderman EL.** Hemodynamic consequences of afterload reduction in patients with chronic aortic regurgitation. *Circulation.* 1976 May;53(5):879-83. PubMed PMID: 1260993.
5. **Miller RR, Vismara LA, DeMaria AN, Salel AF, Mason DT.** Afterload reduction therapy with nitroprusside in severe aortic regurgitation: improved cardiac performance and reduced regurgitant volume. *Am J Cardiol.* 1976 Nov 4;38(5):564-7. PubMed PMID: 983953.

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REFERENCES

1. **Mohammadi S, Tchana-Sato V, Kalavrouziotis D, Voisine P, Doyle D, Baillet R, Sponga S, Metras J, Perron J, Dagenais F.** Long-term clinical and echocardiographic follow-up of the Freestyle stentless aortic bioprosthesis. *Circulation.* 2012 Sep 11;126(11 Suppl 1):S198-204. PubMed PMID: 22965983.
2. **Smith CR, Leon MB, Mack MJ, Miller DC, Moses JW, Svensson LG, Tuzcu EM, Webb JG, Fontana GP, Makkar RR, Williams M, Dewey T, Kapadia S, Babaliaros V, Thourani VH, Corso P, Pichard AD, Bavaria JE, Herrmann HC, Akin JJ, Anderson WN, Wang D, Pocock SJ; PARTNER Trial Investigators.** Transcatheter versus surgical aortic-valve replacement in high-risk patients. *N Engl J Med.* 2011 Jun 9;364(23):2187-98. doi: 10.1056/NEJMoal103510. Epub 2011 Jun 5. PubMed PMID:21639811.