A Double Whammy: Severe Aortic Stenosis and Cocaine Overwhelm the Mitral Valve

Rohit Maini, M.D.; *Jonathan Lim*, M.D.; *Jing Liu*, M.D.; *Itamar Birnbaum*, M.D.; *Farooq Mirza*, M.D.; *Nasser Lakkis*, M.D.; *Ihab Hamzeh*, M.D.

BAYLOR COLLEGE OF MEDICINE, HOUSTON, TEXAS

ABSTRACT: A 50-year-old man presented with acute onset dyspnea following cocaine use. He had severe aortic stenosis (AS), mild mitral regurgitation (MR) due to mitral valve prolapse, and no coronary artery disease on recent coronary angiography. He was in acute heart failure with signs of impending cardiogenic shock. Urgent bedside echocardiography revealed hyperdynamic left ventricular systolic function with acute severe MR from a ruptured chordae tendineae. The acute cocaine-induced spike of his already elevated left ventricular systolic pressure from severe AS likely precipitated chordal rupture of his vulnerable mitral valve. This patient underwent emergent mitral and aortic valve replacements. Although cocaine use has been associated with a myriad of cardiovascular complications, acute MR due to chordal rupture has not, to our knowledge, been previously reported in this setting. Prompt diagnosis with echocardiography and surgical intervention are of paramount importance in the management of acute MR.

INTRODUCTION

Common acute cardiac complications of cocaine abuse include coronary vasospasm and demand ischemia, with 0.7% to 6% of cocaine-related medical admissions involving chest pain and acute myocardial infarction.[1-3] However, acute mitral valve regurgitation with subsequent cardiogenic shock in the setting of cocaine use is an unusual complication.

CASE PRESENTATION

A 50-year-old man with previously asymptomatic severe bicuspid aortic stenosis (50 mm Hg mean gradient by left heart catheterization) presented to the emergency room with acute onset of chest pressure and dyspnea that woke him from sleep. Prior transthoracic echocardiogram had demonstrated mitral valve prolapse with mild mitral regurgitation, and prior coronary angiography showed no evidence of coronary artery disease. The patient reported cocaine use a few hours prior to the onset of symptoms. His initial vital signs were as follows: blood pressure 91/64 mm Hg, heart rate regular at 120 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation of 89% while on room air. The physical exam revealed jugular venous pressure of 7 cm H_20, a crescendo-decrescendo systolic murmur loudest at the right upper sternal border, a soft early systolic murmur at the apex with a third heart sound (S3), and diffuse bilateral crackles. He had cool extremities with trace edema and thready but equal peripheral pulses. An electrocardiogram showed sinus tachycardia with 0.5 mm ST depressions in the lateral leads. Laboratory tests were significant for elevated troponin of 3.63 ng/mL and brain natriuretic peptide of 1009 pg/mL. Bedside echocardiogram performed immediately after examination revealed a hyperdynamic left ventricle with a flail posterior leaflet (P2 segment) due to a ruptured chordae (Figure 1), resulting in severe anteriorly directed mitral regurgitation. He was taken to the operating room and successfully underwent mitral and aortic valve replacements. Pathologic evaluation of the mitral valve leaflets revealed a ruptured chord and myxomatous degeneration without evidence of endocarditis. The patient's remaining hospital course was uneventful.

DISCUSSION

This is a case of acute mitral regurgitation (MR) secondary to a ruptured mitral leaflet chord in the setting of cocaine use. A literature review did not reveal any similar reported cases. It is plausible that the acute chordal rupture occurred when the patient's preexisting elevated left ventricular systolic pressure from severe aortic stenosis was augmented by the cocaine-induced rise in afterload. The acute spike in left ventricular systolic pressure likely overwhelmed the mechanical limitations of the mitral valve apparatus—already vulnerable from myxomatous disease (MD)—and resulted in ruptured chorda tendineae and acute MR. Indeed, MD affects the mechanical properties of the mitral valve chordae so that they fail at much lower loads than normal chordae.[4] The most common cause of acute MR is acute chordae tendineae rupture, with the most common underlying pathologies being mitral valve prolapse, subacute bacterial endocarditis, rheumatic heart disease, MD, and ischemic heart disease, in order of decreasing incidence.[5]

Acute mitral insufficiency is a medical and surgical emergency, yet prompt recognition and accurate diagnosis of acute MR can

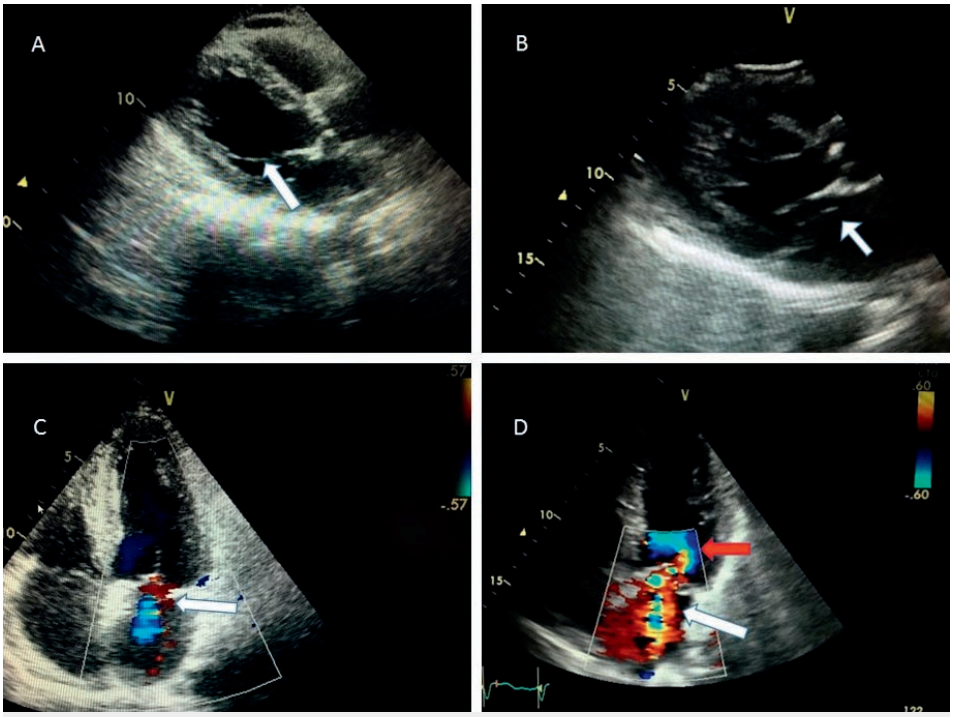

Figure 1.
(A) Transthoracic echocardiogram with color Doppler from 1 year prior to presentation shows mitral valve prolapse (arrow points to preserved chord attached to posteromedial papillary muscle). (B) Transthoracic echocardiogram on presentation shows flail leaflet with ruptured chord (arrow to ruptured chord). (C) Transthoracic echocardiogram from 1 year prior to presentation with color shows mild mitral regurgitation (white arrow). (D) Transthoracic echocardiogram on presentation prior to intervention with large area flow convergence (red arrow) consistent with severe mitral regurgitation (white arrow).

be difficult.[6-10] Although cardiovascular collapse is a common presentation, exam findings suggesting acute MR are often subtle. The classic findings of severe chronic MR include a high-pitched holosystolic murmur accompanied by an S3, best heard over the cardiac apex with the diaphragm of the stethoscope. The physical exam findings of acute MR, however, are dramatically different from those of chronic MR. An acute MR murmur is usually early systolic, soft, and at times not audible due to rapid equalization of pressure between the left ventricle and atrium. Cardiomegaly is usually absent. These dissimilarities make sound clinical judgment and a high index of suspicion vital to making this diagnosis. Acute MR can lead to severe heart failure, flash pulmonary edema, and increased cardiovascular mortality. Patients with acute chordal rupture with hemodynamic instability should be treated emergently with mitral valve replacement or repair, if feasible.[11-14]

CONCLUSION

This case illustrates the pivotal role of echocardiography in evaluating acute cardiac decompensation and the importance of recognizing acute valvular regurgitation in the absence of typical physical exam clues. It also underscores the untoward cardiovascular effects of cocaine, which can be even more catastrophic with underlying cardiac disease. Although chordae tendineae rupture due to cocaine use is rare, prompt diagnosis is of paramount importance given the risk for significant morbidity and mortality, and early surgical intervention should be considered in all cases.

Conflict of Interest Disclosure:
The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

Keywords:
mitral regurgitation, papillary muscle, chordae tendineae, cardiogenic shock, aortic stenosis

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