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Severe tricuspid regurgitation due to papillary muscle rupture: a rare complication of anterior myocardial infarction and ventricular septal perforation

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An 85-year-old woman presented with anorexia, general fatigue, and cold extremities. She had no history of chest pain. Her blood pressure was 74/62 mmHg, and SpO_2 was 86%. V-wave was observed in her jugular vein. A pan-systolic murmur with Levine grade 3/6 at the left sternal border of the fourth intercostal space. Electrocardiography revealed ST elevation in V_2 to V_4 and QS pattern in V_1 to V_3. Her creatinine level was 1.58 mg/dL (range: 0.37–0.84 mg/dL), and urea nitrogen was 87 mg/dL (range: 10–24 mg/dL).

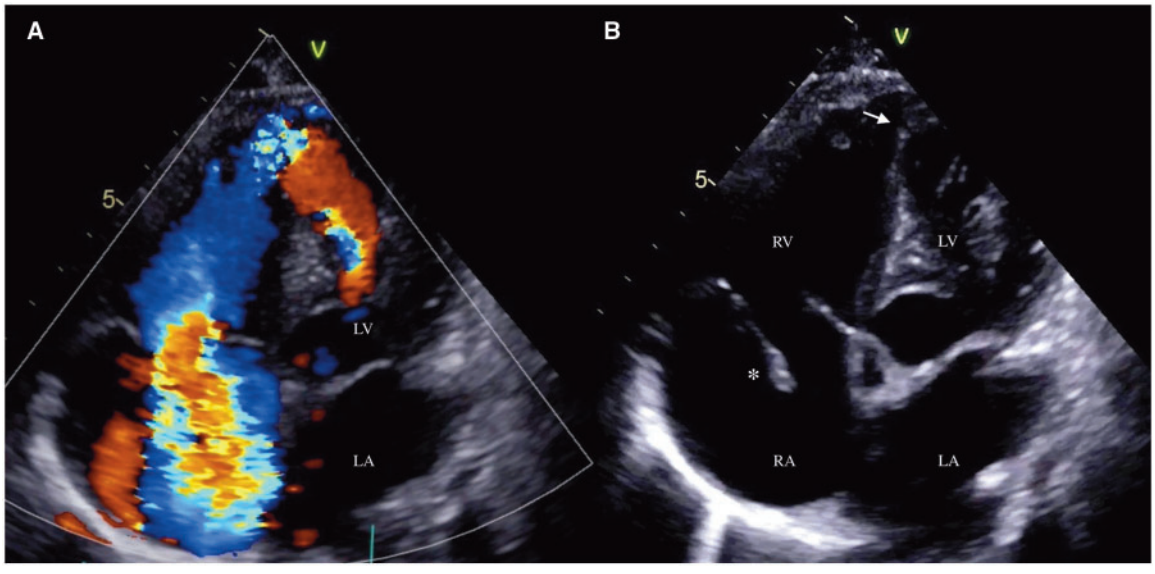

Figure 1 (*A*) Colour-Doppler showing severe tricuspid regurgitation and bidirectional shunt flow via the ventricular septal perforation (white arrow). (*B*) Four-chamber view showing the anterior tricuspid leaflet (*) is flail due to papillary muscle rupture as a complication of acute anteroseptal infarction. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

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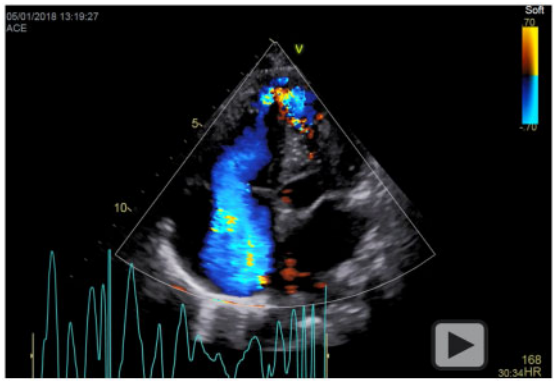
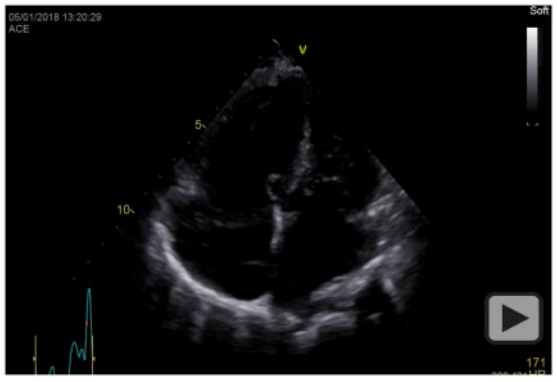


Video 1 Colour-Doppler showing severe tricuspid regurgitation and bidirectional shunt flow via the ventricular septal perforation. Shunt flow through ventricular septal perforation is seen mainly from the left ventricle to the right ventricle in systole, and a small right ventricle to left ventricle shunt is seen in diastole.



Video 2 Four-chamber view showing the anterior tricuspid leaflet was flail due to papillary muscle rupture as a complication of acute anteroseptal infarction.

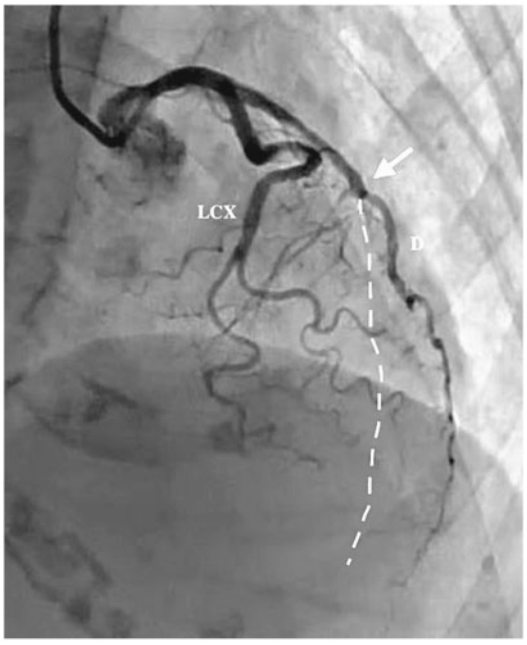
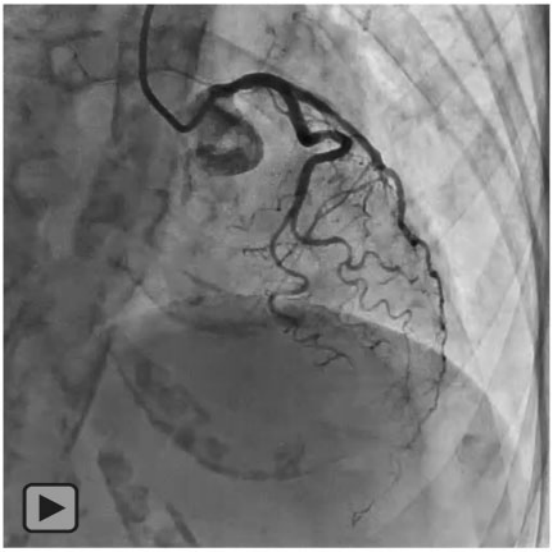


Figure 2 Coronary angiography in right anterior oblique view showing total occlusion (white arrow) at the mid-portion of the left anterior descending artery; the dotted line indicates the invisible distal left anterior descending artery. D, diagonal branch; LCX, left circumflex coronary artery.



Video 3 Coronary angiography in right anterior oblique view showing total occlusion at the mid-portion of the left anterior descending artery.

Transthoracic echocardiography revealed dyskinesis of the apex and ventricular septal perforation (VSP) with bidirectional shunt. Left ventricular (LV) ejection fraction was 58%. The anterior tricuspid leaflet was flail due to papillary muscle rupture (PMR), resulting in severe tricuspid regurgitation (*Figure 1* and *Videos 1 and 2*). These findings suggested acute myocardial infarction (MI) and cardiogenic shock with multiorgan failure.

We recommended surgical repair. Coronary angiography revealed total occlusion at the mid-portion of the left anterior descending artery (LAD; *Figure 2* and *Video 3*). Ventricular septal perforation closure was attempted using the modified Daggett's procedure and tricuspid annuloplasty using a 26-mm Physio Tricuspid ring; however, we were unable to perform coronary artery bypass grafting to the LAD because of extensive myocardial necrosis. The patient died of multiorgan failure postoperatively. The tricuspid valve (TV) papillary muscles (PM) are resistant to ischaemia because systolic pressure of the right ventricle (RV) is lower than that of the LV. The anterior PM are attached to the moderator band and supplied by the RV branches of the LAD.[1,2] Anteroseptal MI sometimes extends to the anterior

RV free wall along the LAD. In our patient, VSP increased RV systolic pressure, making the TV PM susceptible to ischaemia. To the best of our knowledge, TV PMR complicated with acute anterior MI are rare in the literature.[3]

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Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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