Clinical vignette
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Biventricular rupture with extracardiac left-to-right shunt in the setting of an acute myocardial infarction

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An 80-year-old man with a history of coronary artery bypass surgery 6 months earlier due to symptomatic three- vessel disease presented with acute chest pain of 9 h duration. The ECG showed ST-segment elevation in the inferior leads, and fibrinolysis was immediately carried out. Nevertheless, no signs of reperfusion were noted, so he was referred to our institution for rescue angioplasty.

At arrival, blood pressure was normal, but the patient exhibited signs of low peripheral perfusion, and a loud pansystolic murmur was heard. Coronary angiography showed three- vessel disease, patent bypasses, and a new thrombotic occlusion of a small posterolateral branch of the right coronary artery, which was considered to be the cause of the myocardial infarction. Left ventriculography (Panels A, right anterior oblique view, and B, left anterior oblique view) revealed a left ventricular wall rupture at the inferior basal segment which connected the left ventricle (LV) to a pseudoaneurysm (P) containing the cardiac rupture. It also disclosed the appearance of contrast within the right ventricle (RV), but the mechanism of this finding was unclear.

To further clarify the problem, a transesophageal echocardiogram was performed. Transgastric basal short-axis view showed the rupture of the basal inferior left ventricular wall (Panel C, black arrow), and colour Doppler demonstrated the presence of turbulent, bidirectional flow between the LV and the pseudoaneurysm through a narrow neck (Panel D, arrow). Transesophageal echocardiography also revealed a defect in the anterolateral right ventricular free wall communicating the pseudoaneurysm with the RV (Panel C, white arrow). After advancing and anteflexing the tip of the probe to avoid the turbulent flow from the LV, this communication between the pseudoaneurysm and the RV could be selectively demonstrated by colour Doppler (arrows in Panels E, diastole, and F, systole). No ventricular septal defects were seen.

Unfortunately, the patient’s haemodynamic status deteriorated rapidly and he died shortly after admission.