Anterolateral papillary muscle rupture: an unusual complication of septic coronary embolism

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Received 10 August 2010; accepted after revision 3 September 2010; online publish-ahead-of-print 28 September 2010

In most cases, acute mitral valve regurgitation in the setting of infective endocarditis is caused by the destruction of either the mitral valve leaflets or the chordal apparatus. A 54-year-old woman had development of respiratory failure due to pulmonary oedema from severe acute mitral valve regurgitation in the setting of acute bacterial endocarditis. She was found to have a ruptured anterolateral papillary muscle from occlusion of the circumflex artery by embolic vegetations arising from the aortic valve. Although this occurrence is uncommon, an embolic phenomenon resulting in myocardial infarction and subsequent rupture of papillary muscle must be considered as a cause of acute severe mitral valve regurgitation.

Case report

A 54-year-old woman sought care for a 3 day history of fever, chills, and backache. Her medical history was significant for end-stage renal disease, systemic lupus erythematosus, hypertension, and diabetes mellitus. Three months earlier, she had received an unrelated living-donor renal transplant and was treated for vancomycin-resistant enterococcal (VRE) urinary tract infection (UTI).

At initial evaluation in the emergency department, the patient was afibrile, and physical examination revealed an ejection systolic murmur over the aortic area caused by aortic valve sclerosis, which had been noted 3 months previously by transthoracic echocardiography (TTE). Relevant laboratory findings included leucocytosis, increased liver enzyme values, and no evidence of UTI. Blood cultures were obtained and she was sent home. The following day she had development of gram-positive bacteraemia, and she was admitted to the hospital. Her condition deteriorated within 36 h because of severe sepsis. Subsequently, hypotension developed, followed by respiratory distress due to pulmonary oedema, which required mechanical ventilation and initiation of vasopressors. Electrocardiography showed inferolateral ST-segment elevation. A new systolic murmur over the apex was also noted. Because of technically difficult TTE, she underwent urgent transoesophageal echocardiography (TEE), which showed large, mobile, aortic valve vegetations (Figure 1) with minimal aortic valve regurgitation. More importantly, TEE also detected that the head of the anterolateral papillary muscle (AL-PM) was ruptured (Figure 2A and B; see Supplementary data online, Movie 1), along with a hypokinetic lateral wall of the left ventricle and severe mitral valve regurgitation (Figure 3; see Supplementary data online, Movie 2). Cardiac catheterization revealed abrupt occlusion of the inferior limb of the obtuse marginal branch of the codominant left circumflex artery (Figure 4), most likely due to embolic vegetations, without significant coronary artery stenosis in other arteries. The patient underwent aortic and mitral valve replacement and coronary artery bypass grafting. During valve replacement surgery, aortic valve vegetations (Figure 5) and rupture of the AL-PM (Figure 6) were confirmed. No gross vegetations were seen on the mitral valve. Ecchymosis of the lateral wall of the left ventricle was noted, as a result of infarction.

Postoperatively, the patient had development of severe VRE sepsis and multiorgan failure and then died. In this case, AL-PM rupture due to ischaemic necrosis was ultimately considered the
cause of the severe mitral valve regurgitation rather than the usual mechanism of destruction of the mitral valve by vegetations, as is commonly seen in patients with endocarditis.

**Discussion**

Complications of endocarditis include, in general, abscess formation and valvular destruction with or without significant regurgitation. In addition, systemic embolization is also a frequent manifestation (up to 65% of cases) of left-sided endocarditis. In most cases, the embolization leads to neurologic events or compromise of the blood supply to the peripheral organs. Rarely (0.62% incidence), septic embolism can invade coronary arteries and result in acute coronary syndrome. Most frequently, severe mitral valve regurgitation in the setting of endocarditis is due to either destruction of the mitral valve or, uncommonly, destruction
of the papillary muscles by direct invasion of the infection. Posterior papillary muscle rupture due to septic coronary embolism has been reported. However, to our knowledge, extensive infarction of the lateral wall and resultant AL-PM rupture from occlusion of a single obtuse marginal branch by septic coronary embolism has never been reported. Our case underscores that, although this occurrence is uncommon, embolic vegetations can cause rupture of the AL-PM leading to severe mitral valve regurgitation, as a result of infarction of the lateral wall despite the dual blood supply of the AL-PM.

**Supplementary data**

Supplementary data are available at European Journal of Echocardiography online.

**Conflict of interest:** none declared.

**References**