An 86-year-old woman without any history of heart disease or trauma to the chest experienced an acute onset of dyspnea associated with a productive cough for 2 hours. Her medical history revealed that she was hypertensive for the past 20 years. Her blood pressure was 90 mm Hg in systole and 50 mm Hg in diastole. Immediately after admission, mechanical ventilation was initiated because there was progressive respiratory depression. A chest x-ray showed marked pulmonary congestion (Figure 1A). On physical examination, a grade III systolic murmur was noted at the apex, and a coarse breathing sound was detected within the whole lung field. ECG showed sinus tachycardia without any evidence of myocardial infarction (Figure 1B). Creatine kinase and its MB isoenzyme were not elevated, but high-sensitivity troponin T was 0.035 ng/mL (normal, 0–0.014 ng/mL). Transthoracic echocardiography revealed left ventricular hypertrophy without regional wall motion abnormalities. The left ventricular ejection fraction was >70%, and E/E′ was 34, suggesting highly elevated left ventricular filling pressure. There was a posteriorly directed eccentric jet of severe mitral regurgitation in the color-flow Doppler imaging (Figure 2A and 2B and Movie I in the online-only Data Supplement). The posterior mitral annulus showed severe calcification, and the mean transvalvular pressure gradient was 6.8 mm Hg, suggestive of functional mitral stenosis (Figure 2C). A large finger-like hypermobile material attached to the anterior mitral leaflet and an unstable postero medial papillary muscle were seen (Figure 2D–2F and Movies II and III in the online-only Data Supplement). Two-dimensional and 3-dimensional transesophageal echocardiography clearly visualized the ruptured head of the postero medial papillary muscle, the freely mobile stump within the left ventricle, and severe eccentric mitral regurgitation (Figure 3A–3C and Movies IV–VI in the online-only Data Supplement). In terms of treatment, we strongly recommended an emergency mitral valve surgery, but her family refused cardiac surgery because of her old age. After 2 weeks of intensive medical treatment, her breathing became tolerable, and she maintained a good level of oxygen saturation. Hence, mechanical ventilation was removed on hospital day 14. A coronary angiogram revealed a significantly calcified stenotic lesion on the mid left anterior descending artery (Figure 4A), but there was no critical stenosis on the right coronary artery that could explain the infarct-related rupture of the postero medial papillary muscle (Figure 4B). Two-dimensional (Figure 5A and 5B) and 3-dimensional (Figure 5C and 5D) images on computed tomography visualized severe mitral annular calcification extended to the posterior mitral leaflet and mitral valve apparatus.

A papillary muscle rupture is a rare cardiac emergency and is most frequently associated with acute myocardial infarction. It is usually fatal and refractory to medical treatment. Once a papillary muscle rupture has been diagnosed, urgent mitral valve replacement is required to correct the hemodynamic
deterioration of the patient. Spontaneous papillary muscle rupture rarely develops without acute myocardial infarction. Here, we detail a case of an elderly woman with acute mitral regurgitation caused by a spontaneous rupture of a papillary muscle. This rupture was likely related to severe mitral annular calcification with myocardial and valvular extension. This patient has functional and structural risk factors for a spontaneous papillary muscle rupture. She had long-standing hypertension that could have increased myocardial wall tension. The patient had severe mitral annular calcification and degenerative changes of the mitral apparatus.

Disclosures
None.

Figure 2. Color-flow Doppler imaging in a transthoracic echocardiogram (A and B) shows a posteriorly directed eccentric jet of severe mitral regurgitation. The continuous-wave Doppler image across the mitral valve (C) is compatible with functional mitral stenosis resulting from severe mitral annular calcification. In the parasternal short-axis view (D), a hyperdynamic left ventricle is shown without regional wall motion abnormalities. In the apical 4-chamber (E) and 2-chamber (F) views, severe mitral annular calcification and the unstable posteromedial papillary muscle (arrow) are visible.

Figure 3. Two-dimensional transesophageal echocardiography (A and B) visualizes the ruptured head of the posteromedial papillary muscle (arrow) and posteriorly directed severe eccentric mitral regurgitation. The real-time, 3-dimensional transesophageal echocardiography (C) shows the prolapsed anterior mitral leaflet with a ruptured papillary muscle from the surgeon’s view (arrow).
Figure 4. Coronary angiography of the left coronary artery (A) shows a significantly calcified stenotic lesion on the mid left anterior descending artery. Coronary angiography of the right coronary artery (B) reveals no critical stenosis.

Figure 5. Two-dimensional and 3-dimensional images on computed tomography show severe mitral annular calcification (A and C) extended to the posterior mitral leaflet and mitral valve apparatus (B and D). The arrow indicates a stump of a ruptured posteromedial papillary muscle.
Spontaneous Rupture of a Papillary Muscle
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_Circulation_. 2013;127:e586-e588
doi: 10.1161/CIRCULATIONAHA.112.142448

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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