

Double papillary muscle infarction related to right coronary artery occlusion: a case report

Sağ koroner arter tıkanıklığına bağlı iki taraflı papiller kas enfarktüsü: Olgu sunumu

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Posterior papillary muscle is involved more frequently than the anterior papillary muscle during acute myocardial infarction. However, infarction of both papillary muscles is very rare. In this article, we report a 62-year-old male case with bilateral papillary muscle infarction which was likely related to occlusion of the right coronary artery. Following a successful coronary artery bypass grafting and mitral valve replacement, the patient was discharged without any complication.

Key words: Acute myocardial infarction; infarction of papillary muscle; occlusion of right coronary artery.

It is well known that the posterior papillary muscle is involved in myocardial infarction (MI) and that it ruptures more frequently than the anterior papillary muscle.^[1,2] The vulnerability of the posteromedial papillary muscle is linked to its single primary blood supply that arises from the dominant coronary artery, whereas the anterolateral papillary muscle has a dual blood supply emanating from the left anterior descending (LAD) artery as well as the obtuse marginal branch of the circumflex (Cx) arteries.^[1-3] However, infarction of both papillary muscles related to the occlusion of the right coronary artery (RCA) is extremely rare. Herein, we report a case with ischemic severe mitral regurgitation (MR) associated with the occlusion of the RCA which led to the infarction of both papillary muscles. The patient underwent successful

Akut miyokard enfarktüsü sırasında arka papiller kas ön papiller kasa göre daha fazla etkilenir. Ancak her iki papiller kasın enfarktüsü ise oldukça nadirdir. Bu yazıda, sağ koroner arter tıkanıklığına bağlı olabilen iki taraflı papiller kas enfarktüsünün gözlemlendiği 62 yaşında bir erkek olgu sunuldu. Başarılı bir koroner arter baypas greftleme ve mitral kapak replasmanı sonrasında, hasta sorunsuz bir şekilde taburcu edildi.

Anahtar sözcükler: Akut miyokard enfarktüsü; papiller kas enfarkti; sağ koroner arter tıkanıklığı.

concomitant coronary artery bypass grafting (CABG) and mitral valve surgery and has had an uneventful follow-up.

CASE REPORT

A 62-year-old man presented with progressively worsening dyspnea associated with a productive cough. He had been complaining of chest tightness, orthopnea, and decreased effort tolerance for over a month. The past medical history of the patient was unremarkable except for hypertension and active smoking, and he had no family history of coronary artery disease (CAD). At physical examination, he was in respiratory distress, his blood pressure was 140/80 mmHg, and he had a heart rate of 100 bpm with a regular respiratory rate of 26/minute. An examination of the cardiovascular



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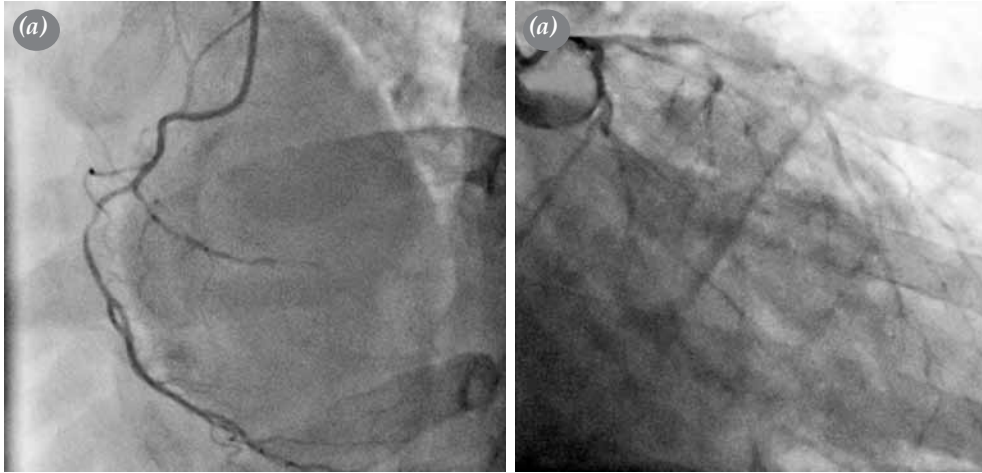


Figure 1. Coronary angiography revealed a dominant, occluded right coronary artery distal to the acute marginal branch and 80% stenosis at the mid-portion of the left anterior descending.

system revealed a late systolic murmur. In addition, the patient had bilateral coarse crepitation at the basal segments of both lungs. An initial electrocardiogram (ECG) showed ST depression at the inferior leads, and chest radiography showed cardiomegaly with congestive heart failure.

The patient was admitted to the coronary care unit with the diagnosis of decompensated heart failure secondary to acute coronary syndrome (ACS). A transthoracic echocardiogram (TTE) showed a

normal left ventricular ejection fraction (LVEF) with a hypokinetic inferobasal segment. The mitral valve morphology was normal with a flail anterior leaflet that was not coapting with the posterior leaflet. Color flow Doppler showed severe MR with a predicted pulmonary artery pressure (PAP) of 90 mmHg. Furthermore, coronary angiography revealed a dominant, occluded RCA distal to the acute marginal branch and 80% stenosis at the mid portion of the LAD (Figure 1). The patient was then diagnosed with CAD along with severe MR, and a surgical intervention was planned.

During the operation, distal anastomosis of the RCA with a saphenous graft was initially performed, the left atrium was subsequently opened. Both papillary muscles were a yellowish color and softened, which is consistent with necrosis (Figure 2). Next, the mitral valve was resected without any attempt to preserve



Figure 2. A macroscopic specimen showing the soft, yellowish papillary muscles.

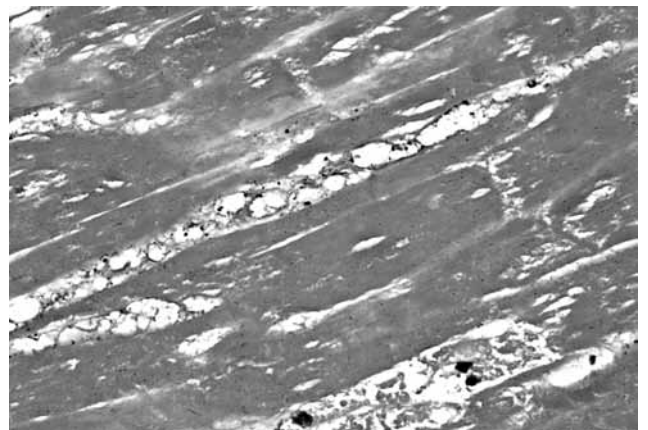


Figure 3. A microscopic examination revealed complete karyolysis and loss of striation in the myocardial cells (H-E x 400).

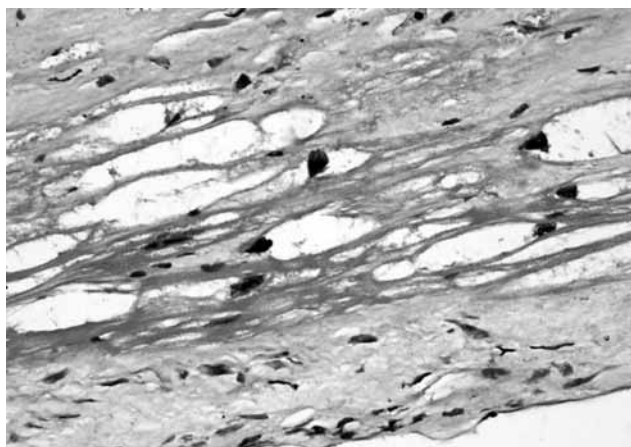


Figure 4. A microscopic examination showed necrotic myocardial tissue apposed by a thin layer of viable myocardial cells that disclosed severe vacuolar degeneration (H-E x 400).

the subvalvular apparatus, and a 31 mm St. Jude mechanical mitral valve (St. Jude Medical Inc., St. Paul, MN, USA) was implanted. After completion of the distal anastomosis of the left internal artery to the LAD and the proximal anastomosis of the saphenous vein to the RCA, the patient was weaned from cardiopulmonary bypass (CPB) without any support. A microscopic examination revealed complete karyolysis and loss of striation in the myocardial cells. Additionally, the interstitial fibrovascular connective tissue was also destroyed, leaving behind only cellular debris (Figure 3), and necrotic myocardial tissue was apposed by a thin layer of viable myocardial cells, which disclosed severe vacuolar degeneration. Even though these cells underwent substantial injury, they had apparently escaped necrosis by receiving some nourishment through diffusion from the adjacent endocardium (Figure 4). The postoperative period was uneventful, and the patient was discharged from the hospital on the 10th postoperative day.

DISCUSSION

Although the frequency of papillary muscle rupture after MI has been researched in several studies, the frequency and clinical features of papillary muscle necrosis or infarction have been investigated in only a few reports because papillary muscle necrosis is difficult to visualize pre-mortem.^[4,5] In a recent study by Tanimoto et al.,^[5] the prevalence and clinical significance of papillary muscle infarction was detected via gadolinium-enhanced magnetic resonance imaging in patients with MI. In this study composed of 47 patients with papillary muscle infarction, the posterior papillary muscle was

involved in (77%) while the anterior papillary muscle was involved in only (23%). In addition, papillary muscle infarct was observed more frequently in the patients with Cx lesions (74%) and RCA lesions (48%) compared with those with LAD lesions (13%). In this randomized large study, only one patient (2%) with a proximal Cx lesion showed delayed enhancement in both the anterior and posterior papillary muscles.

In a clinical study, Calvo et al.^[6] assessed the differential clinical and angiographic characteristics of patients with severe MR with regard to whether they were related (n=31) or unrelated (n=16) to ruptured papillary muscle complicated by acute MI. The study also showed that in the patients with papillary muscle rupture, the involvement of the RCA was higher than that of the Cx, whereas in those with non-papillary muscle rupture, both arteries seemed to be equally responsible for the MR. In contrast, the rate of LAD involvement was remarkably low (6.6%) in both groups.

Reports of double papillary muscle infarction are very rare in the literature. In one study, Lobo et al.^[7] showed postinfarction double papillary muscle rupture in three surgically excised mitral valves. The RCA was dominant in all three cases and exhibited segmental occlusion in two of the patients. Surprisingly, in an analysis of 22 necropsy patients by Barbour and Roberts,^[4] a quantitative examination of the amounts of narrowing by atherosclerotic plaque in each of the four major epicardial coronary arteries (left main, RCA, LAD, and Cx) disclosed less narrowing in the patients with rupture than in the patients with fatal acute MI that was not associated with rupture. In our case, identifying the exact pathophysiological reason of the infarction in both papillary muscles without a post-mortem examination was not possible. However, we believe that the dominant and occluded RCA was the most likely culprit since it may have had an anatomic variation that was feeding both papillary muscles, and the occlusion could have caused the infarction. On the other hand, one of the other possible mechanisms may have been the narrowing of the LAD that was supplying the papillary muscle(s). As previously mentioned, although it was not occluded, it could have also caused the infarct.

The timing of surgery for these patients is critical, and postponing the operation to wait for optimal clinical conditions may worsen the cardiac and respiratory functions. Repair was our first surgical plan for the mitral valve since the diagnosis of papillary muscle infarction was not

suspected preoperatively. We initially thought that degenerative mitral valve disease was causing the leaflet prolapse and MR. The morphological features became apparent only upon careful examination during the operation. In our opinion, the operative diagnosis of papillary muscle infarction is crucial because the techniques for repair, for example neochordae replacement by placing double-armed mattress sutures at the fibrous tip of the infarcted papillary muscle, could be wasted.

In conclusion, our patient was suffering from a very rare case of acute MR caused by double papillary muscle infarction which was most probably related to RCA occlusion. He was treated via a successful surgical intervention and is currently in the first year of follow-up.

Declaration of conflicting interests

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