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ANTEROLATERAL PAPILLARY MUSCLE RUPTURE AS A RESULT OF SINGLE-VESSEL CORONARY ARTERY DISEASE WITHOUT MYOCARDIAL INFARCTION

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Keywords: coronary artery disease, chronic myocardial ischemia, mitral valve, mitral regurgitation, mitral valve repair.

Summary

The rupture of a papillary muscle is usually associated with the previous occurrence of myocardial infarction, but it may have another etiology. The authors of this article have found no reports about anterolateral papillary muscle rupture caused by a single-vessel coronary artery disease without myocardial infarction. In this paper, an extremely rare case of anterolateral papillary muscle rupture caused by chronic ischemia due to single-vessel coronary artery disease is presented. Since the patient of this case had remained hemodynamically stable and responded well to medical treatment, the mitral valve was successfully repaired 6 weeks after the onset of symptoms.

Introduction

The rupture of a papillary muscle usually causes an acute atrioventricular valve incompetence and a life-threatening cardiac failure, constituting a surgical emergency [1]. This condition is often associated with a previous occurrence of myocardial infarction but it may also arise due to such etiologies as blunt chest trauma, endocarditis, or chronic ischemia [2-5].

Ischemic rupture of the anterolateral papillary muscle is several times less frequent than rupture of the posteromedial papillary muscle [6,7] and in the absence of acute myocardial infarction, it is extremely rare [4,5,8].

The latter case which has occurred in the medical practice of the authors of this paper, is described further below.

Case Report

A 64-year-old female arrived at a local hospital experiencing the symptoms of heart failure. She was complaining of the shortness of breath and the discomfort in her chest. Her physical activity has been limited to the walking to the toilet and the kitchen in her small apartment. At that time, her electrocardiogram and troponin levels were normal. The patient was diagnosed with pulmonary edema. No further investigations were performed. The patient had been treated with diuretics for ten days and when her symptoms had improved she was discharged. A few days later the same symptoms reoccurred accompanied by an increasing peripheral edema. The patient was referred to the tertiary hospital.

While the patient was being admitted to the hospital, she was conscious and alert. Her heart rhythm was irregular, at the rate of 95 beats per minute, and her arterial blood pressure was 125/80 mmHg. Auscultation revealed rales bilaterally. Severe peripheral edema was observed above her knees. The patient was in New York Heart Association (NYHA) class III–IV.

The woman was a non-smoker and non-drinker, had a BMI of 52 kg/m^2, and had had essential hypertension and diabetes mellitus for years. No previous history of myocardial infarction or stroke was documented. Her arterial blood pressure was controlled by beta blockers and angiotensin-converting enzyme (ACE) inhibitors.

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The chest radiographic examination revealed a bilateral hydrothorax. Moreover, electrocardiogram showed atrial fibrillation with no suggestion of acute ischemia. Severe mitral regurgitation as well as moderate tricuspid regurgitation along with left ventricular ejection fraction (LVEF) > 60% and left ventricular end-diastolic cavity dimension (LVEDD) of 57 mm as well as the systolic pulmonary artery pressure of 75 mmHg were observed on transthoracic echocardiography. The blood results of the patient did not indicate any significant abnormalities. The level of troponin I was 0.023 mcg/L (normal value is < 0.03 mcg/L).

Since the patient remained hemodynamically stable, she was being treated medically and her clinical status improved significantly. Transesophageal echocardiography specified the cause of severe mitral regurgitation: chordae rupture at A1 and A2 segments (Fig. 1). Coronary angiography showed significant stenosis of a distal obtuse marginal branch (Fig. 2).

On the 44th day after the onset of symptoms, the patient underwent elective surgery during which the median sternotomy was performed. Cardiopulmonary bypass was established between the distal ascending aorta and both vena cava. Then, the patient was cooled down moderately. Her heart was arrested with antegrade blood cardioplegia. During the operation, it was noted that the obtuse marginal branch was very small and distal therefore it was left intact. The mitral valve was accessed through the atrial septum. Inspection revealed a rupture of the anterolateral papillary muscle, causing the prolapse of A1 and A2 segments. Afterwards, repair of the mitral valve was performed. The papillary muscle was reattached to its native position by using *U*-shaped stitch reinforced with the pledget. Three artificial chordae were implanted to support prolapsing segments, 4-0 polytetrafluoroethylene sutures were used. A 32-mm semi-rigid annuloplasty ring (SJM™ Séguin Semi-Rigid Ring, St. Jude Medical Inc., St. Paul, MN) was used to support mitral annulus. A tricuspid repair was performed applying the suture bicuspidalization technique. The patient was then weaned from cardiopulmonary bypass and her chest was closed. Her further recovery was rather uneventful and on the 16th day after the operation she was discharged.

After her return home, patient's nutritional habits improved. She lost 12 kilograms in 6 months. The woman retired and her life style became less stressful. At 4-, 14- and 30-month follow-up visits, she had no symptoms of heart failure. According to the findings of transthoracic echocardiography, trace mitral regurgitation and tricuspid regurgitation along with left ventricular ejection fraction (LVEF) >55% were found. Due to bilateral osteoarthritis of patient's knees, her physical activity subsequently decreased and 24 months later she regained her presurgical weight.

Discussion

The rupture of a papillary muscle is defined as an acute complication of myocardial infarction [6]. However, it can occur as a consequence of other etiologies such as blunt chest trauma, endocarditis, or chronic ischemia. Ischemia may develop due to various pathologies or even rare diseases [1-5].

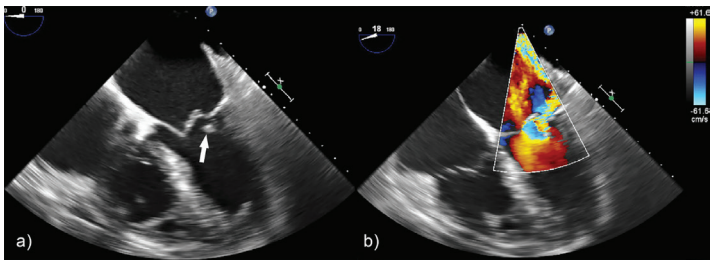

Figure 1. 2D transesophageal long-axis view: a) arrow points to the flailing mass attached to the leaflet of the mitral valve, b) Doppler color flow mapping shows the jet of severe eccentric mitral regurgitation.

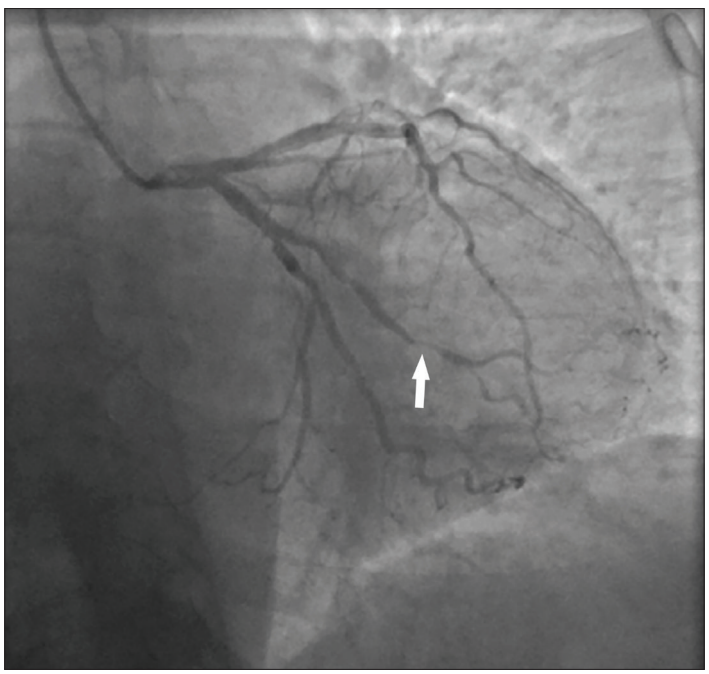

Figure 2. Coronary angiogram showing the severe narrowing of obtuse marginal branch of the circumflex artery.

The case of anterolateral papillary muscle rupture due to coronary artery disease with no evidence of acute coronary syndrome is presented in this article. A review of literature of the past 15 years in the PubMed database was conducted and only 2 cases of papillary muscle rupture due to coronary artery disease without myocardial infarction were found [4,5]. Rupture of the anterolateral papillary muscle is several times less frequent than rupture of the posteromedial papillary muscle [6,7].

In 71% of the population, the anterolateral papillary muscle has a double-vessel blood supply, while in 29%, it has a single-vessel blood supply [8]. Coronary angiography of the patient described in this article showed the pathology limited to the single branch of the circumflex artery. The authors of this paper presume that in this case, the rupture of the anterolateral papillary muscle had occurred because of the diseased single-vessel blood supply.

Rupture of the papillary muscle most often causes acute severe mitral regurgitation and pulmonary edema with hemodynamic compromise. This condition requires surgical emergency. Since the patient of the presented case remained hemodynamically stable and responded well to medical treatment, the surgery was performed 6 weeks after onset of the symptoms.

Transesophageal echocardiography did not reveal a rupture of anterolateral papillary muscle. Rather a chordae rupture of A1 and A2 segments was seen. Surgery revealed the detailed and true cause of mitral regurgitation. Transesophageal echocardiography is a sensitive tool which provides the detailed anatomy of the mitral valve. Nevertheless, the presented case demonstrates that it may be difficult to define the cause of mitral regurgitation using the diagnostic method of transesophageal echocardiography.

Conclusions

Chronic ischemia related to single-vessel coronary artery disease may cause a rupture of anterolateral papillary muscle. In the cases of severe mitral regurgitation due to papillary muscle rupture, elective surgery may be performed in hemodynamically stable patients.

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SPENINIO RAUMENS PLYŠIMAS DĖL KORONARINĖS ŠIRDIES LIGOS, NEĮVYKUS MIOKARDO INFARKTUI

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Raktažodžiai: išeminė širdies liga, lėtinė miokardo išemija, dviburio vožtuvo nesandarumas, dviburio vožtuvo taisymas.

Santrauka

Speninio raumens plyšimas dažniausiai yra miokardo infarkto komplikacija. Kartais plyšimas gali įvykti ir dėl kitų priežasčių. Mokslinėje literatūroje trūksta publikacijų, kuriose būtų aprašomas speninio raumens plyšimas, nulemtas koronarinės širdies ligos, neįvykus miokardo infarktui. Todėl šio straipsnio autoriai nusprendė aprašyti ypač retai pasitaikantį priekinio šoninio speninio raumens plyšimą, įvykusį dėl lėtinės koronarinės širdies ligos, kuri savo ruožtu pasireiškė susiaurėjus vienos vainikinės arterijos spindžiui. Kadangi pacientės būklė išliko hemodinamiškai stabili ir ji gerai reagavo į medikamentinį gydymą, autoriai aprašė sėkmingą dviburio vožtuvo taisymo operaciją, praėjus 6 savaitėms po simptomų atsiradimo.

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