

Role of echocardiography in diagnosis and management of complete papillary muscle rupture caused by myocardial infarction

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ABSTRACT

Aim To evaluate the usefulness of echocardiography in the diagnosis of complete rupture of papillary muscle.

Methods Transthoracic (TTE) and transesophageal echocardiography (TEE) was performed with the ATL 3000 HDI Ultrasound Inc (Bothell, WA, USA) with a 2.5 MHz transducer and 5-7 MHz multiplane phased array transducer. We are reporting about two patients (a 45 and a 51-year old male) with complete ruptures of papillary muscle following acute myocardial infarction (AMI).

Results Both patients were previously treated with fibrinolysis in their local hospitals, 400 and 300 km, respectively, away from our hospital. Massive mitral regurgitation developed in both followed by rapid deterioration of hemodynamic state and severe heart failure, because of which both were transferred by helicopter to the Coronary Care Unit of our clinic. The diagnosis of complete papillary muscle rupture was confirmed in both patients by TTE and TEE. Due to the significant deterioration in their hemodynamic state, vasoactive drugs and intra-aortic balloon pump support were applied. Both patients then underwent mitral valve replacement, accompanied by concomitant coronary artery bypass grafting in one case.

Conclusion Transesophageal echocardiography is a more accurate and rapid diagnostic method in patients with mechanical complications of AMI than TTE.

Keywords: mitral valve, replacement, coronary artery, bypass grafting

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INTRODUCTION

Papillary muscle rupture is a rare, but often fatal mechanical complication of acute myocardial infarction (AMI); it occurs in 1% to 5% of patients with AMI (1). Echocardiography is the imaging technique of choice for detecting mechanical complications of AMI including myocardial free wall rupture, acute ventricular septal defect and mitral regurgitation secondary to papillary muscle rupture or ischemia (2,3). Transesophageal echocardiography is more sensitive than TTE, and useful for providing more detailed and/or unique anatomic information in patients with papillary muscle rupture than TTE (3,4).

Minami et al (5) have reported on six patients (from 1986 to 2002) with posterior (n=4) and anterior (n=2) papillary muscle rupture; all patients underwent mitral valve replacement, concomitant coronary artery bypass grafting (CABG) was performed in five of six patients, and the perioperative mortality rate was 33%. Tavakoli et al (6) reviewed 21 consecutive patients (from 1988 to 1998) with a perioperative mortality of 19%. Transthoracic (TTE) and/or transesophageal echocardiography (TEE) established the diagnosis of papillary muscle rupture in 14 patients, and in others the diagnosis was suspected on the basis of the presence of flail mitral leaflets (3).

Emergency surgery, even as a salvage procedure for acute postinfarction mitral papillary muscle rupture is justified (6).

The aim of this study was to evaluate the usefulness of echocardiography in the diagnosis of complete rupture of papillary muscle, based on a presentation of two male patients with massive mitral regurgitation and severe heart failure due to complete mitral papillary muscle rupture following acute myocardial infarction.

PATIENTS AND METHODS

Dubrava University Hospital provides acute care for about 220000 inhabitants from the east Zagreb area including Sesvete, DugoSelo, Vrbovec and Sv. Ivan Zelina. Our hospital is also one of the primary percutaneous coronary intervention(PCI) centers included in primary-PCI network of Croatia which provides care for patients with acute coronary syndrome for Međimurje County, Ko-

privnica - Križevci County, Bjelovar - Bilogora County and east part of the Zagreb County. About 750 patients with acute coronary syndrome are admitted to our hospital per year.

Diagnosis of AMI included clinical symptoms, electrocardiogram, cardiac biomarkers (troponin I, normal limit is 0.04 ug/mL), echocardiography and coronary angiography. Definite diagnosis of AMI requires cardiac biomarker troponin with at least one value above the 99th percentile of the upper reference limit and with at least one of the following: symptoms of ischemia, new or presumably new significant ST-T changes or new left bundle branch block (LBBB), development of pathologic Q waves in the ECG, imaging evidence of new loss of viable myocardium and identification of an intracoronary thrombus by angiography or autopsy (2). The diagnosis of heart failure was based on typical symptoms, typical signs and relevant structural heart disease. Patients' symptomatic limitation is graded using the New York Heart Association (NYHA) functional classification (class I - IV). This classification assigns patients to one of four class depending on the degree of effort needed to elicit symptoms of angina, fatigue, dyspnea or palpitation. Patients with NYHA IV class presenting with symptoms at rest and unable to carry out any physical activity without discomfort.

Transthoracic (TTE) and transesophageal echocardiography (TEE) was performed with the ATL 3000 HDI Ultrasound Inc (Bothell, WA, USA) with a 2.5 MHz transducer and 5-7 MHz multiplane phased array transducer. The presence and grade of mitral regurgitation (MR) were screened by color-flow imaging with the MR jet area to left atrium area ratio; a ratio of >20%, 20-40% and >40% represents mild, moderate and severe MR, respectively. In patients with more than trace regurgitation, the regurgitant orifice area was calculated by the proximal isovelocity surface area (PISA) method and the degree of MR was graded as mild (regurgitant orifice area <0.2cm²), moderate (regurgitant orifice area 0.2 to 0.4 cm²), or severe (regurgitant orifice area >0.4cm²). Classically a vena contracta width <3mm indicates mild MR, whereas a vena contracta width ≥7 mm indicates severe MR (7).

RESULTS

Case 1

A 45-year-old man was first admitted to a local hospital for chest pain, faintness, dyspnea and waning exercise tolerance. This hospital is approximately 400 km away from our clinic. Treatment consisted of fibrinolytic therapy with streptokinase, along with other standard therapeutic regimens. Transthoracic echocardiography showed mitral regurgitation (MR) grade 3, and in spite of aggressive therapy, the patient began developing severe hemodynamic instability. After a week-long clinical deterioration he was transferred to the coronary care unit (CCU) of our clinic. He presented with clinical signs of severe heart failure, NYHA class IV, while physical examination revealed bilateral basal pulmonary rales without jugular venous distension. On careful auscultation soft heart sounds and a holosystolic heart murmur with a point of maximum intensity at the apex grade 4/6 were detected. Blood pressure was 140/80 mm Hg, accompanied by tachycardia 133 beats/min. Electrocardiogram (ECG) revealed sinus tachycardia of 133 beats/min and Q-waves of the left ventricular inferoposterior wall. Chest radiography disclosed cardiac enlargement on the left side and pulmonary plethora. In laboratory findings there was increased aspartate aminotransferase 55 U/L, alanine aminotransferase 51 U/L, lactate dehydrogenase 765 U/L, uric acid 698 U/L, total plasma cholesterol 6.09 mmol/L, LDL-cholesterol 4.36 mmol/L, and fibrinogen 7.8 g/L. Transthoracic echocardiography and TEE discovered mitral regurgitation grade 4. There was a mobile solid mass of 1.6x0.6 cm in size on the posterior mitral leaflet, of the same echo-dense structure as the myocardium and suggestive of complete rupture of the head of the posteromedial papillary muscle. The ruptured head of the papillary muscle was detected in the left ventricle during diastole accompanied by its systolic displacement displaced to the left atrium during systole (Figure 1). Echocardiography showed hypokinesis of the left ventricular inferoposterior wall with normal ejection fraction (LVEF=65%). Ventriculography confirmed the diagnosis of severe MR with normal left ventricular function. Coronary angiography detected significant stenosis of the circumflex, the first obtuse marginal and of the right coronary artery, as well as occlusion

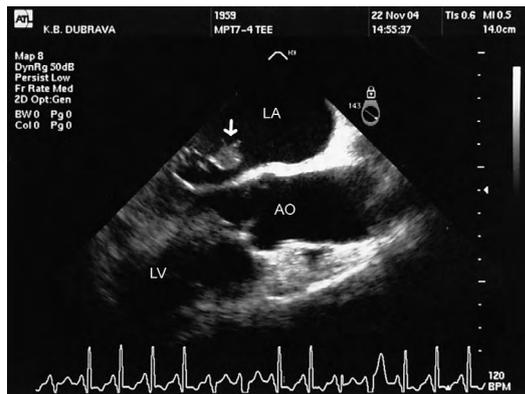


Figure 1. Transesophageal echocardiogram showing complete rupture of the head of posteromedial papillary muscle prolapsing into the left atrium (arrow) (Vincelj J, 2004)

LA, left atrium; LV, left ventricle; AO, aorta.

of the second obtuse marginal artery. Due to the significant fluctuations in the hemodynamic state of the patient, vasoactive drugs and the intra-aortic balloon pump (IABP) were applied. Ten days after AMI, the patient underwent mitral valve replacement with a Carbomedics mechanical valve and concomitant coronary artery bypass grafting of the circumflex artery. During surgery an intraoperative TEE also confirmed the diagnosis of complete posteromedial papillary muscle rupture (Figure 2). The patient was weaned from cardiopulmonary bypass without hemodynamic disturbances and 24 hours later he was also successfully weaned from both mechanical ventilation and IABP. Transthoracic echocardiography performed in the early postoperative course disclosed normal function of mechanical mitral valve and normal left ventricular function. Histopathological analysis found ischemic necrosis of the posteromedial papillary muscle and fibroelastic thickness of the endocardium. A part of the mitral leaflet showed fibroelastic thickness to some extent. Microbiology results showed a sterile papillary muscle and leaflet.



Figure 2. Specimen of the resected ruptured posteromedial papillary muscle (Vincelj J, 2004)

Case 2

A 51-year-old man was treated for acute ST-segment elevation myocardial infarction (STEMI) of inferior localization in his local hospital, 300 km away from our clinic. Treatment consisted of fibrinolytic therapy with streptokinase, along with other standard therapy. During the first week of hospitalization, acute heart failure emerged and the patient started exhibiting signs of rapid hemodynamic deterioration. On day 12, he was transferred to the CCU of our hospital. He presented with clinical signs of severe heart failure, NYHA class IV. Physical examination showed bilateral basal pulmonary rales without jugular venous distension. Careful auscultation revealed calm heart sounds and holosystolic heart murmur grade 3/6. Blood pressure was 105/65 mm Hg, accompanied by sinus tachycardia 120 beats/min. ECG revealed sinus tachycardia of 120 beats/min and Q-waves of the left ventricular inferoposterior wall. Chest radiography disclosed cardiac enlargement on the left side and pulmonary plethora. His routine laboratory test revealed increased creatine kinase 254 U/L, aspartate aminotransferase 36 U/L, alanine aminotransferase 74 U/L, lactate dehydrogenase 775 U/L, uric acid 452 U/L, LDL-cholesterol 3.74 mmol/L, and fibrinogen 10.4 g/L. Transesophageal echocardiography exposed a completely ruptured head of the anterolateral papillary muscle prolapsing into the left atrium with massive MR. There was a display of chaotic movement of mitral valve. Excessive amount of pericardial effusion (20 mm) was seen, along with a collapse of the right atrium in telediastole, as sign of impending tamponade (Figure 3). Coronary angiography detected

a subtotal stenosis of first obtuse marginal artery along with a non-significant ostial stenosis of hypoplastic right coronary artery. The patient was referred for urgent cardiac surgery. Being unsuitable for surgical revascularization, he only underwent mitral valve replacement with a Carbomedics mechanical valve 24 days after AMI. An intraoperative TEE was also performed. Postoperative course of the patient (with known prior medical history of obstructive lung disease) was complicated by a respiratory infection and two episodes of bronchospasm, which were both successfully treated. Rapid postoperative improvement of cardiac function and regression of congestion in pulmonary circulation ensued, and echocardiography performed during postoperative course revealed normal function of the mechanical mitral valve and no impairment of left ventricular systolic function. Histopathological analysis showed ischemic necrosis of the anterolateral papillary muscle. The patient was discharged, fully recovered, 14 days after surgery. Both patients fully recovered and were discharged from hospital 8 and 14 days, respectively, after surgery.

DISCUSSION

Papillary muscle rupture is a rare but often fatal mechanical complication of AMI with hemodynamic instability (4,8). The involvement of the posteromedial papillary muscle is 6-12 times more common than that of the anterolateral (4,8). Indeed, posteromedial papillary muscle vascularization is provided only by the inter-ventricular posterior coronary artery originating even from the right coronary artery or from the circumflex coronary artery, and it may aggravate infarction heralded by occlusion in such vessels (8). Echocardiography is the imaging technique of choice for detecting mechanical complications of AMI including myocardial free wall rupture, acute ventricular septal defect, and mitral regurgitation secondary to papillary muscle rupture or ischemia (2). Transthoracic echocardiography is able to identify a papillary muscle rupture with a diagnostic sensitivity of 65-85%. Transesophageal echocardiography is more sensitive than TTE (4,9). Recently, TEE has been reported as a valuable adjunct to conventional echocardiography, by providing more detailed and/or unique anatomic information in patients

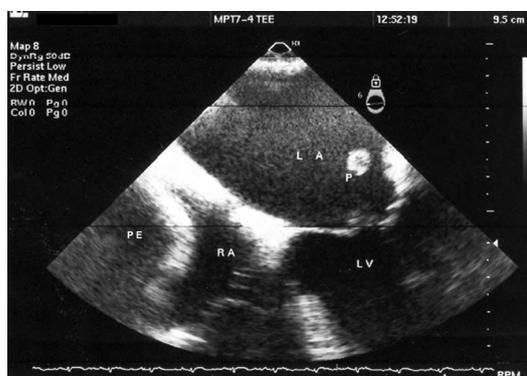


Figure 3. Transesophageal echocardiogram showing complete rupture of the head of anterolateral papillary muscle (P) prolapsing into the left atrium (Vincelj J, 2004)

LA, left atrium; LV, left ventricle; RA, right atrium; PE, pericardial effusion.

with papillary muscle rupture involving either mitral or tricuspid valve apparatus, to either ischemic or traumatic damage. When the head of the papillary muscle is ruptured, it can often be imaged prolapsing into the left atrium. In up to 30% of patients, however, the ruptured head may not prolapse into the left atrium. In this case the diagnosis is made by noting chaotic movement of the ruptured head in the left ventricle (10). Complete papillary muscle rupture may cause acute MR with pulmonary edema or cardiogenic shock (8). These events depend on the infarction severity and papillary muscle morphology. There are morphology differences in papillary muscle attachment to the left ventricular wall. The main part of the posteromedial papillary muscle may be attached to the left ventricular wall with one, two or three heads, whereby the papillary muscle heads maybe attached separately or jointly (3). Furthermore, the *cordae tendineae* of one papillary muscle may be attached to both mitral leaflets. In papillary muscle rupture, the grade of mitral regurgitation depends on rupture severity (complete or incomplete) and number of papillary muscle heads. The regurgitation jet will be smaller if the papillary muscle has two or three heads, only one of them being involved by rupture. Severe mitral regurgitation develops upon complete rupture of a papillary muscle which has only one or two heads, or complete rupture of all three muscle heads, depending on differences in the muscle morphology (11). In our patients, the posteromedial and anterolateral papillary muscle was attached to the muscle wall with one head, while chordae tendineae were attached to the anterolateral leaflet, thus additionally contributing to MR. In this case mitral valve repair was impossible for two reasons. First, myocardial necrosis caused complete posteromedial papillary muscle rupture; and second, chordae tendineae of the posteromedial papillary muscle were in part attached to the anterolateral leaflet (A2 scallop) thus also participating in severe MR.

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Our patients have some distinctive features related to prior published data. Distinctive features of our cases are that both patients were being treated for AMI in hospitals 400 and 300 km away from our hospital. Initially, in both cases the diagnosis of papillary muscle rupture was suspected, and as rapid hemodynamic destabilization became evident, both patients were transported, as safely and as fast as possible. Optimal cooperation and coordination were crucial for successful outcome. This relates to interregional and regional cooperation, but also to collaborative work between cardiologists and cardiac surgeons, so called "hybrid strategy", which included early reperfusion, when possible, and subsequent mitral valve surgery (12).

Fibrinolytic therapy has positive effect on mechanical complications in AMI. The study of Gueret et al. demonstrated a dramatic reduction in the incidence of mechanical complications of AMI in the reperfusion era when compared with the non reperfusion era. Results of study documented beneficial effect of thrombolysis on some of these complications, whereas similar information after primary coronary angioplasty is scarce (13).

Transesophageal echocardiography as an accurate and rapid diagnostic method is highly important in patients with papillary muscle rupture in AMI. The role of coronary angiography is mandatory before making any therapeutic decision. Surgery, which should be ensured without delay, was performed after three days of treatment, with a satisfactory result. In our patients, urgent transportation, accurate and rapid diagnostics, hemodynamic stabilization and timely surgery were all crucial for successful outcome.

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TRANSPARENCY DECLARATION

Competing interests: None to declare.

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Uloga ehokardiografije u dijagnostici i zbrinjavanju kompletne ruptуре papilarnog mišića uzrokovane infarktom miokarda

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SAŽETAK

Cilj Procijeniti korisnost ehokardiografije u dijagnostici kompletne ruptуре papilarnog mišića.

Metode Za transtorakalnu (TTE) i transezofagusnu ehokardiografiju (TEE) korišten je aparat *ATL 3000 HDI Ultrasound Inc.* (Bothell, WA, USA), sondama od 2,5 MHz i od 5 do 7 MHz. Opisana su dva muška bolesnika u dobi 45 i 51 godine s potpunom rupturom papilarnog mišića u akutnom infarktu miokarda (AIM).

Rezultati Oba bolesnika liječena su trombolitičkom terapijom zbog AIM-a u dvije lokalne bolnice udaljene 400, odnosno 300 km. Zbog masivne mitralne regurgitacije koja je uzrokovala hemodinamsku nestabilnost i zatajivanje srca teškog stupnja bolesnici su helikopterom transportirani u koronarnu jedinicu naše Klinike. Dijagnozu kompletne ruptуре papilarnog mišića potvrdili smo u oba bolesnika pomoću TTE-a i TEE-a. Zbog teškog općeg stanja i hemodinamske nestabilnosti bolesnici su primali vazoaktivnu terapiju i priključeni su na intraaortalnu balon-crpku. U oba bolesnika implantirana je umjetna mitralna valvula, a aortokoronarno premoštenje učinjeno je u jednog bolesnika.

Zaključak Transezofagusna ehokardiografija u odnosu na TTE je točna i brza dijagnostička metoda u bolesnika s mehaničkim komplikacijama u akutnom infarktu miokarda.

Ključne riječi: mitralna valvula, implantacija, koronarne arterije, premoštenje