

The Difficult Diagnosis of Ischaemic Papillary Muscle Rupture

Case report from an urban emergency department

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تشخيص عسير لتمزق إقفاري في العضلة الحليمية تقرير حالة من قسم حضري لطب الطوارئ

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ABSTRACT: We present a rare case of severe ischaemic papillary muscle rupture in a 67-year-old male patient who was admitted to the Emergency Department of the University Hospital Bern, Switzerland, in November 2013 with acute chest pain. On admission, the patient's blood pressure was 60/40 mm/Hg, his pulse was 110 beats per minute and his respiratory rate was 20 breaths per minute. An electrocardiogram was normal and focused assessment with sonography in trauma was negative. Transthoracic echocardiography showed possible thickening of the mitral valve leaflet with no indications of severe mitral insufficiency or wall motion abnormalities. Triple-rule-out computed tomography angiography revealed no pulmonary emboli or aortic dissection, although coronary atherosclerosis was present. Finally, severe insufficiency of the mitral valve with rupture of the papillary muscle, likely due to *ischaemia*, was observed via transoesophageal echocardiography. The patient underwent a successful surgical intervention and was discharged 10 days later in stable condition.

Keywords: Emergency Medicine; Shock; Myocardial Infarction; Papillary Muscles; Heart Rupture; Echocardiography; Case Report; Switzerland.

المخلص: نعرض هنا حالة نادرة وحادة لتمزق العضلة الحليمية الإقفارية عند مريض عمره 67 عاما أدخل لقسم طب الطوارئ في المستشفى الجامعي بمدينة بيرن في سويسرا في نوفمبر 2013م بسبب ألم حاد في صدره. وكان ضغط دمه عند إدخاله للمستشفى 60/40 ملم زئبق، ومعدل نبضه 110 ضربة في الدقيقة، ومعدل تنفسه 20 مرة في الدقيقة. وكان رسم القلب لديه طبيعياً، وتقييم التخطيط التصواتي سلبياً. وأوضح تخطيط صدى القلب عبر الصدر إمكانية حدوث ثخن في وريقة الصمام المترالي من غير وجود إشارات إلى حدوث قصور مترالي وخيم، أو اعتلالات في حركة الجدار. وأوضح تصوير الأوعية بواسطة التصوير المقطعي المحوسب أنه لا توجد انصمامات رئوية أو تسلسل أبهري، رغم وجود تصلب في الشرايين التاجية. وأخيراً تم اكتشاف وجود قصور وخيم في الصمام المترالي مع تمزق إقفاري في العضلة الحليمية، غالباً ما تكون قد حدثت بسبب الإقفار (نقص التروية)، وذلك بواسطة تخطيط صدى القلب عبر المريء. وتم إجراء تدخل جراحي ناجح للمريض، وتم تخريجه من المستشفى عقب استقرار حالته بعد عشرة أيام من إجراء تلك العملية.

كلمات مفتاحية: طب الطوارئ؛ الصدمة؛ احتشاء عضلة القلب؛ العضلات الحليمية؛ تمزق القلب؛ تخطيط صدى القلب؛ تقرير حالة؛ سويسرا.

ALTHOUGH PATIENTS ADMITTED TO EMERGENCY departments often suffer from chest pain, the actual incidence of acute myocardial infarction is relatively low (approximately 2–3%).^{1,2} The rupture of a papillary muscle is a rare but generally fatal mechanical complication; it is found in 0.5–5% of patients with acute myocardial infarctions.^{3,4} Diagnosing an acute myocardial infarction becomes even more difficult if there is no evidence of acute *ischaemia* on an electrocardiogram and if echocardiography indicates no regional wall motion abnormalities. One of the current major challenges in emergency medicine is the rapid identification of these patients so as to treat them promptly with appropriate cardiac interventions. This report describes a rare case of severe ischaemic papillary muscle rupture diagnosed via transoesophageal echocardiography.

Case Report

A 67-year-old male was admitted to the Emergency Department of the University Hospital Bern in Switzerland in November 2013 with dyspnoea at rest and a one-hour history of chest pains radiating down both arms. When the ambulance arrived, his oxygen saturation in room air was 84%. During transport to the hospital, the patient became hypotensive (blood pressure: 70/50 mmHg). The patient's only known cardiovascular risk factor was a history of smoking cigarettes and he reported that he was not currently taking any regular medications. On arrival to the Emergency Department, the patient's blood pressure had reduced to 60/40 mmHg. He had a pulse of 110 beats per minute, a respiratory rate of 20 breaths per minute and his oxygen saturation was 96% after the

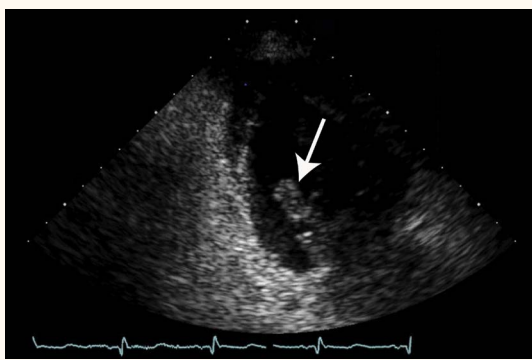


Figure 1: Transthoracic echocardiography showing a thickened posterolateral mitral leaflet (arrow) in a 67-year-old male patient with ischaemic papillary muscle rupture. Note the lack of wall motion abnormalities or pericardial effusion.

administration of 2 L of oxygen. His temperature was 36.8 °C and consciousness was measured at level 14 of the Glasgow Coma Scale.

A physical examination of the patient on arrival indicated regular heart sounds with a systolic murmur; the heart sounds were heard most clearly above Erb's point. Auscultation of the lung, examination of the abdomen and a preliminary neurological examination were all normal. An electrocardiogram was normal with no evidence of acute *ischaemia*. Focused assessment with sonography in trauma was negative. Transthoracic echocardiography performed by cardiologists revealed a thickened mitral valve leaflet with mild mitral insufficiency and, most importantly, no wall motion abnormalities or pericardial effusion [Figure 1]. The left ventricular ejection fraction was estimated to be 65%. Creatine kinase (1,319 U/L; normal value: <190 U/L), creatine phosphokinase-MB (119.2 µg/L; normal value: <4.9 µg/L) and high-sensitivity cardiac troponin T (2.150 µg/L; normal value: <0.014 µg/L) levels were all substantially elevated. Although the patient was becoming increasingly haemodynamically unstable, the diagnosis remained unclear. Consequently, triple-rule-out computed tomography was performed. No evidence of pulmonary emboli or aortic dissection was noted, although there were signs of general coronary atherosclerosis with plaques and without any luminal occlusion. The scan also revealed an infiltrate in the left upper lobe, perhaps due to aspiration pneumonia, with a small amount of left pleural effusion.

After diagnostic and laboratory testing, the patient was prescribed vasopressin before being intubated and receiving analgaesics. He was in a progressively unstable state and was re-evaluated by an interdisciplinary team, including intensive care physicians, cardiologists and emergency physicians. Although there were marked increases in heart enzymes, due to the lack of segmental wall motion abnormalities on transthoracic

Table 1: Comparison of transthoracic and transoesophageal echocardiographic findings for a 67-year-old male patient with ischaemic papillary muscle rupture

Finding	TTE	TOE
Left ventricular EF	Normal	Normal
Regional wall motion abnormalities	None	None
Pericardial effusion	None	None
Papillary muscle rupture	Thickened mitral valve leaflet	Thickened mitral valve leaflet and rupture of the papillary muscle
Mitral valve regurgitation*	Mild insufficiency†	Severe insufficiency‡
Left atrium	No dilatation	Dilatation and severe secondary pulmonary hypertension

TTE = transthoracic echocardiography; TOE = transoesophageal echocardiography; EF = ejection fraction.

*Using colour Doppler imaging. †Small jet of mitral regurgitation.

‡Large eccentric jet of mitral regurgitation.

echocardiography, a coronary angiogram was not immediately performed. The patient was transferred to the intensive care ward for further monitoring. While there, he rapidly developed cardiogenic shock and it was not possible to stabilise him. As a result, transoesophageal echocardiography was performed. This demonstrated severe mitral valve insufficiency with rupture of the papillary muscle, dilatation of the left atrium and severe secondary pulmonary hypertension. The left ventricular ejection fraction remained the same (65%) and there were still no signs of any regional wall motion abnormalities [Table 1].

A coronary angiogram was subsequently carried out which showed that the papillary muscle rupture was caused by a distally occluded right coronary artery (RCA). As the patient remained unstable, cardiac surgery was performed. The mitral valve was replaced with a biological prosthesis and a coronary artery bypass graft was undertaken. The postoperative course was uneventful and the patient was discharged in a stable condition 10 days later.

Discussion

This report presents an unusual case of ischaemic papillary muscle rupture. Firstly, it is rare for cardiogenic shock to develop only a few hours after the onset of symptoms; the highest risk of shock is normally two to seven days after the myocardial infarction, when healing has begun.⁵ In this particular

case, the short interval of time could potentially be explained by a silent ischaemic event occurring one or two days previously. Alternatively, the papillary muscle may have already been affected for some unrelated reason.⁶ Secondly, the lack of regional wall motion abnormalities is an uncommon finding, particularly in a hypotensive patient; the only possible explanation for this would be an acute papillary muscle rupture with severe acute mitral regurgitation. As in this case, the posteromedial papillary muscle is often affected during a myocardial infarction in the area perfused by the RCA; in comparison, the anterolateral papillary muscle has a dual blood supply and therefore very rarely exhibits ischaemic complications.¹ Thirdly, it is rare for cases of papillary muscle rupture with severe mitral regurgitation to have an uncomplicated postoperative course, as observed with the current patient. This condition usually carries a high mortality rate; Schroeter *et al.* determined a 30-day postoperative mortality rate of 39.3%, while another study reported rates of 4.2% and 25.0% for intraoperative and in-hospital mortality, respectively.^{7,8}

Echocardiography is the imaging modality of choice for the non-invasive assessment of mechanical cardiac complications such as acute mitral regurgitation in the setting of a myocardial infarction.⁶ In these cases, the appearance of mitral regurgitation, often in relation to papillary muscle rupture, is an important sign of poor prognosis.⁹ Consequently, echocardiography plays an essential role in the early diagnosis, estimation of severity and determination of the pathomechanisms of ischaemic mitral regurgitation in patients with acute myocardial infarctions.⁹ Transoesophageal echocardiography has been well established as the initial diagnostic tool for identifying papillary muscle rupture, with a diagnostic sensitivity of 65–85%.⁶ Due to the close proximity of the ultrasound transducer to the mitral apparatus, transoesophageal echocardiography is often used to improve reliability when diagnosing the cause of mitral regurgitation; it has been found to increase the diagnostic yield to approximately 95–100%.¹⁰

Christ *et al.* described a case of partial papillary muscle rupture diagnosed solely by transoesophageal echocardiography; previous transthoracic echocardiographs had shown no pathologies or only posterior mitral leaflet prolapse.³ Similar experiences have been reported for patients with acute septal ruptures in the acute stage of a myocardial infarction. Maillier *et al.* found that only eight out of 15 septal ruptures could be visualised directly by conventional transthoracic echocardiography; in contrast, transoesophageal echocardiography successfully diagnosed 14 of the same

patients.¹⁰ However, Monin *et al.* found that only 5% of patients required transoesophageal echocardiography to adequately quantify the severity of mitral insufficiency.¹¹ Transthoracic echocardiography is therefore an appropriate first-line imaging modality and transoesophageal echocardiography should be reserved for patients with poor-quality transthoracic images or those with continued deterioration and an unclear diagnosis.

Conclusion

Corresponding with previous findings in the literature, the current case report supports the view that transoesophageal echocardiography is a valuable tool to confirm the diagnosis of ischaemic papillary muscle rupture. In these cases, several diagnostic measures may often be needed. As such, the utilisation of transoesophageal, rather than transthoracic, echocardiography may lead to a life-saving diagnosis for patients who are cardiovascularly unstable.

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