CASE REPORT

Anterolateral papillary muscle rupture after intervention of the right coronary artery

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Anterolateral papillary muscle rupture;
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Abstract Rupture of the anterolateral papillary muscle following a right coronary artery occlusion is extremely rare, and when complicated by a right ventricular infarction, can be fatal. The literature on optimal management of this complication is limited. We present an unusual case of anterolateral papillary muscle rupture following intervention of the right coronary artery. Published by Elsevier España, S.L.U. on behalf of Sociedade Portuguesa de Cardiologia.

PALAVRAS-CHAVE
Rotura do músculo papilar anterolateral;
Regurgitação mitral;
Enfarte do ventrículo direito;
Enfarte do miocárdio periprocedimento

Rotura do músculo papilar anterolateral após intervenção na artéria coronária direita

Resumo A rotura do músculo papilar ântero-lateral, na sequência da oclusão da artéria coronária direita é extremamente rara e, quando complicada por um enfarte do ventrículo direito, pode ser fatal. Presentemente, a literatura do tratamento otimizado da mesma é escassa. Apresentamos um caso muito particular da rotura do músculo papilar ântero-lateral, na sequência de uma intervenção à artéria coronária direita.
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Papillary muscle rupture is a life-threatening complication of acute myocardial infarction (MI) that accounts for 5% of deaths in these patients.1,2 Rupture of the posteromedial papillary muscle is more frequent than of the anterolateral papillary muscle due to the dual blood supply to the latter (left anterior descending and left circumflex arteries).2,3 However, a right coronary artery (RCA) lesion causing an anterolateral papillary muscle rupture is extremely rare, with only two reported cases in the literature.4,5 An associated right ventricular infarction further complicates management and the literature on this complication is limited. Herein, we present a rare case

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of an isolated right coronary artery (RCA) lesion resulting in anterolateral papillary muscle rupture and concomitant right ventricular (RV) infarction. A 78-year-old white male with a history of hypertension, hyperlipidemia, cerebrovascular accident, right carotid endarterectomy (three years previously) and chronic stage 5 kidney disease, presented to the emergency department with retrosternal chest pressure starting two days prior to admission. The patient described the pain as dull in nature, radiating to the left arm. His history was positive for dyspnea on minimal exertion since the onset of symptoms; however there was no associated orthopnea or paroxysmal nocturnal dyspnea. His social history was negative for smoking, alcohol or illicit drug use. His home medications included aspirin, lisinopril, pravastatin, ferrous sulfate, calcium acetate, ergocalciferol and calcium carbonate.

On physical exam, heart rate was 102 beats/min and blood pressure was 103/87 mmHg. The rest of the clinical exam was unremarkable with normal S1, S2 and no murmur. The ECG revealed sinus rhythm, left axis deviation, non-specific intraventricular conduction block and ST-T wave abnormalities suggestive of inferior ischemia. Laboratory tests revealed an elevated troponin I level of 11.8 ng/ml. A two-dimensional (2D) echocardiogram showed severe inferior wall hypokinesis, with moderately reduced left ventricular systolic function and estimated ejection fraction of 35–40%, with no significant mitral regurgitation (MR). The patient was urgently taken to the cardiac catheterization laboratory and was found to have a 99% RCA lesion (Figure 1), with no significant lesions in the other vessels. Successful percutaneous coronary intervention with stenting of the RCA was performed (Figure 1); the patient was admitted to the telemetry floor for close monitoring, and continued on intravenous normal saline to prevent contrast-induced nephropathy.

The next morning, the patient complained of diffuse weakness and lethargy. He was found to be hypotensive (systolic blood pressure 60 mmHg), tachycardic (136 beats/min) and tachypneic (24 cycles/min). Physical examination revealed elevated jugular venous pressure, a new 2/6 holosystolic murmur heard best over the apex, radiating to the axilla, and scattered bibasilar crackles on pulmonary auscultation. The ECG showed sinus tachycardia, with inferior ST elevations suggestive of injury. Postprocedural troponin was also elevated at 114.0 ng/ml. The chest X-ray revealed changes consistent with pulmonary edema. Repeat 2D echocardiography revealed anterolateral papillary muscle rupture with severe eccentric mitral regurgitation (Figure 2), and evidence of right ventricular hypokinesis. He was started on norepinephrine and taken urgently to the catheterization laboratory, where the previously placed stent was found to be patent. Right heart catheterization revealed elevated right-sided pressures with pulmonary artery pressure of 55/25 mmHg, RV pressure of 47/21 mmHg and right atrial pressure of 20 mmHg, and an intra-aortic balloon pump was placed. He was started on a low-dose milrinone infusion due to RV dysfunction and low cardiac output, and the cardiothoracic surgery department was consulted for emergent surgery. Due to RV infarction and improved hemodynamic status with vasopressors and inotropes the surgery was delayed for 2–3 days by the surgical team. However the patient suffered cardiorespiratory failure on day 2, requiring intubation with mechanical ventilation, and expired after cardiac arrest on day 3.

We present an unusual case of anterolateral papillary muscle rupture due to an isolated RCA lesion, complicated by a right ventricular infarction. Papillary muscle rupture usually occurs two to seven days after the infarction, and in our patient occurred on day 3 (he complained of symptoms lasting two days prior to admission). Furthermore, coronary angiography revealed a large RCA with an overwhelming right predominance. The left circumflex artery was small in comparison, and showed only mild disease, which could explain the anterolateral papillary muscle rupture, as its major blood supply was from the RCA. We believe a periprocedural MI also contributed to this catastrophe, as shown by...
Anterolateral papillary muscle rupture

Figure 2 Subcostal transthoracic echocardiographic images showing anterolateral papillary muscle rupture and severe mitral regurgitation (white arrow).

a significant elevation in cardiac biomarkers post-procedure, coupled with findings of RV infarction on imaging studies. Although an early conservative approach was adopted, due to the presence of an RV infarction and a favorable response to vasopressors and inotropic agents, the severity of the MR and the acute nature of its onset contributed to the patient’s demise.

In conclusion, anterolateral papillary muscle rupture from an isolated right coronary artery lesion is extremely rare, and when associated with right ventricular infarction, can be fatal. Echocardiography remains the key to diagnosing this devastating mechanical complication of an acute MI. Early surgery still remains the best possible management option, however data on right ventricular infarctions complicating the above is limited.

Ethical disclosure

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors declare that no patient data appear in this article.

Conflicts of interest

The authors have no conflicts of interest to declare.

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