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CASE REPORT

A 75-year-old woman with chest pain and transient severe left ventricular systolic dysfunction

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Abstract
Introduction: Coronary spasm can cause myocardial ischemia and angina in both patients with and without obstructive coronary artery disease. However, provocation tests using intracoronary acetylcholine (ACh) have been rarely performed in the Western world.
Case report: We report a case of a 75-year-old woman with a history of hypertension and a mechanical aortic prosthesis who presented in the emergency room with acute-onset chest pain, widespread ST-segment depression and severe left ventricular systolic dysfunction, with no signs of prosthesis dysfunction. Emergent coronary angiography excluded obstructive coronary artery disease. Pain relief and normalization of ST segment and systolic function occurred within six hours. The patient was treated for a possible thromboembolic myocardial infarction and was discharged home asymptomatic. Two weeks later, cardiac magnetic resonance was performed showing inferoseptal transmural infarct scar, inferior and inferolateral subendocardial infarct and mid-basal ischemia in the anterior and anterolateral walls. She was readmitted with recurrence of chest pain and it was decided to perform a provocation test with ACh. After injection of ACh into the left anterior descending artery, chest pain, ST-segment depression, blood flow impairment (TIMI 1) and transient grade 3 atrioventricular (AV) block occurred. Intracoronary administration of nitrates reversed the coronary spasm and AV conduction disturbances. Twenty minutes later, chest pain and ischemic ST changes recurred; there was no response to vasodilators and the patient developed cardiac arrest with pulseless electrical activity. Advanced life support was maintained for 32 minutes without return of spontaneous circulation.
Conclusions: Provocation tests have a high sensitivity and specificity for the diagnosis of vasospastic angina. Although it is rare, these tests have the potential risk of irreversible spasm leading to arrhythmia and death.

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PALAVRAS-CHAVE
Dor torácica;
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Angina vasospástica;
Testes de provocação com acetilcolina

Mulher de 75 anos com dor torácica e disfunção sistólica ventricular esquerda grave transitória

Resumo
Introdução: O espasmo coronário pode causar isquemia miocárdica e angina em doentes com e sem doença coronária obstrutiva. Contudo, os testes de provocação com administração intracoronária de acetilcolina (ACh) têm sido raramente usados no mundo ocidental.
Descrição do caso: Mulher de 75 anos com hipertensão arterial e prótese mecânica aórtica admitida no serviço de urgência por dor torácica aguda, depressão generalizada do segmento ST e disfunção sistólica grave do ventrículo esquerdo sem evidência de disfunção protésica. Realizámos angiografia coronária emergente que excluiu doença coronária obstrutiva. Seis horas após admissão, a dor, as alterações de ST e a disfunção ventricular normalizaram. Foi tratado como possível enfarte do miocárdio tromboembólico e teve alta assintomática. Duas semanas após, realizámos ressonância magnética cardíaca que mostrou cicatriz de enfarte transmural inferoseptal e de enfarte subendocárdico inferior e inferolateral, e isquemia anterior e ânterolateral. A doente foi readmitida pela mesma sintomatologia e decidimos realizar estudo de provocação com ACh. Após a injeção de ACh na artéria descendente anterior documentámos dor torácica, depressão do segmento-ST, compromisso do fluxo (TIMI 1) e bloqueio atrioventricular (AV) completo. A administração intracoronária de nitratos reverteu o espasmo coronário e a perturbação da condução AV. No entanto, 20 minutos depois, houve recorrência de dor e alterações de ST sem resposta aos vasodilatadores evoluindo a doente para atividade elétrica sem pulso. Mantivemos suporte avançado de vida durante 32 minutos sem recuperação da circulação espontânea.
Conclusão: Os testes de provocação têm elevada sensibilidade e especificidade para o diagnóstico de angina vasospática. Apesar de raro, estes testes também podem levar ao espasmo coronário irreversível, arritmia e morte.

Case report

A 75-year-old woman with a history of hypertension and aortic valve replacement with a mechanical prosthesis ten years earlier presented to the emergency room (ER) with acute-onset chest pain and diaphoresis. She described a three-month history of chest pain episodes of short duration with no clear relation with effort with no peripheral edemas or orthopnea.

On initial evaluation, the patient reported intense chest pressure but was not in acute distress. She was afebrile, with blood pressure 72/44 mmHg, heart rate 110 beats/min, and respiratory rate 32 breaths/min; pulse oximetry revealed oxygen saturation of 94% in room air. Chest auscultation revealed regular prosthetic sounds with a systolic murmur; rales were heard in both lung fields. The abdominal examination was unremarkable. Her lower extremities were pale and cold but showed no edema. The remainder of the physical examination was normal.

The patient's hypotension and tachycardia suggested the early stages of shock and thus warranted urgent diagnostic testing and management, particularly in a patient with chest pressure. Her hypertension increased the likelihood of an acute coronary syndrome or aortic dissection. The systolic murmur may have been related to prosthetic valve dysfunction or to a mechanical complication of myocardial infarction, such as ventricular septal rupture or papillary muscle rupture, causing acute mitral regurgitation.

A 12-lead electrocardiogram (ECG), obtained approximately six hours after the onset of chest pressure, revealed normal sinus rhythm with widespread ST-segment depression (1–2 mm in leads V2 to V6, DI and aVl, DII–III and aVF) (Figure 1A).

The electrocardiographic findings were consistent with myocardial ischemia, but other conditions associated with ST-segment depression and mimicking myocardial ischemia also had to be considered. An echocardiogram can help with differential diagnosis by identifying regional wall motion abnormalities, a native or prosthetic valve disorder, pericardial effusion, mechanical complications of acute myocardial infarction (as noted above), or even proximal aortic disease.

Transthoracic echocardiography performed in the ER revealed global hypokinesis and impaired left ventricular function with severely depressed ejection fraction (EF); there were no signs of prosthesis dysfunction and the peak gradient between aortic and left ventricular systolic pressures was 26 mmHg.

The patient became progressively less responsive, her blood pressure decreased and metabolic acidosis (pH 7.33, $PaCO_2$ 23.3 mmHg, HCO_3 12.1 mmol/l and base excess −13.8) ensued. In the presence of signs of shock, most probably for cardiocirculatory causes, dobutamine and noradrenaline were initiated.

The patient was also given aspirin 250 mg orally, and clopidogrel 600 mg orally. Her INR was 3.6.