

Acute myocardial infarction after myocardial scintigraphy with dipyridamole

Infarto Agudo do Miocárdio após Cintilografia Miocárdica com Dipiridamol

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Introduction

Dipyridamole is a vasodilator that is widely used for detecting myocardial ischemia. Dipyridamole stress test is as sensitive and specific as those used with other agents, including dobutamine and exercise. Besides, dipyridamole stress testing is considered a safe and feasible alternative to other stress modalities. The most common abnormality found on electrocardiography during dipyridamole stress is ST-segment depression. ST-segment elevation is uncommon.¹ Acute myocardial infarction (AMI) after administration of dipyridamole is an extremely rare event.²

Objectives

To describe a case of myocardial infarction after induction of pharmacological stress with dipyridamole.

Case report

Female 62-year-old patient with metabolic syndrome and previous CAD with a history of coronary angioplasty with conventional stent to descending coronary artery in 2007. She was hospitalized with cholestatic jaundice and underwent endoscopic retrograde cholangiopancreatography three months before. The day after the endoscopic retrograde cholangiopancreatography test, she had episodes of burning epigastric discomfort irradiating to the precordial region, associated with nausea. On two occasions, she was taken to the emergency department, which interpreted the symptom as secondary to biliary disease, and was prescribed an antispasmodic drug, as her cardiological tests were normal (Electrocardiography — ECG and Troponin). As the patient would undergo cholecystectomy, a cardiological opinion was requested. Due to a history of CAD, exercise stress testing was requested. However, the test was ineffective due to chronotropic incompetence secondary to chronic beta-blocker use. Myocardial scintigraphy (MS) with pharmacological stress was then performed.

Keywords

Myocardial infarction; Radionuclide Imaging; Dipyridamole.

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After infusion of dipyridamole for 4 minutes, the patient presented severe and long-lasting epigastric pain with diffuse ST-segment elevation. Aminophylline and nitrate were infused, with improvement of ischemic symptoms and ST-segment elevation. However, the patient completed the test under atrial fibrillation and the scintigraphy images revealed an extensive area of transient hypoperfusion in the anterior, lateral and inferior walls (Figure 1). The patient was referred to the cardiac emergency department, where she presented, once again, epigastric pain and ventricular tachycardia with a pulse, and was immediately submitted to electrical cardioversion. ECG after cardioversion showed ST-segment elevation in the anterior wall (Figure 2). Then, she was transferred to hemodynamics, where cardiac catheterization showed total occlusion in the middle segment of the anterior descending artery. Drug-eluting coronary stent was uneventfully implanted at the occlusion site (Figure 2). She was discharged after 7 days of hospitalization.

Discussion

Dipyridamole acts indirectly by inhibiting adenosine reuptake, increasing its endogenous levels and promoting a potent vasodilating effect. Its adverse effects include hypotension, bradycardia, bronchospasm, chest pain, headache and dizziness.²

In the presence of dipyridamole, there is a “theft” or redirection of flow from the stenosed vessels, which has a high persistent resistance to coronary arteries with preserved vasodilation capacity. This “theft” phenomenon can be intracoronary (when it involves a single coronary segment) or intercoronary (when it involves multiple coronary segments). In addition, dipyridamole-induced vasodilation also decreases perfusion pressure through collateral vessels, so they may produce ischemia in myocardial segments dependent on these vessels.³

The incidence rate of ST-segment depression is reported in 6–25% of studies with dipyridamole. Transient ST-segment elevation is very uncommon and is more frequently observed in patients with severe ischemic heart disease, variant angina, and ventricular aneurysm.^{1,4} AMI following dipyridamole infusion is extremely rare, with an estimated incidence of 1 case in 10,000 tests.⁵

In addition to ST-segment disorder, the patient also had atrial fibrillation and ventricular tachycardia (VT) with hemodynamic instability. VT following dipyridamole infusion is an even rarer event. In a study of 73,000 more patients

Case Report

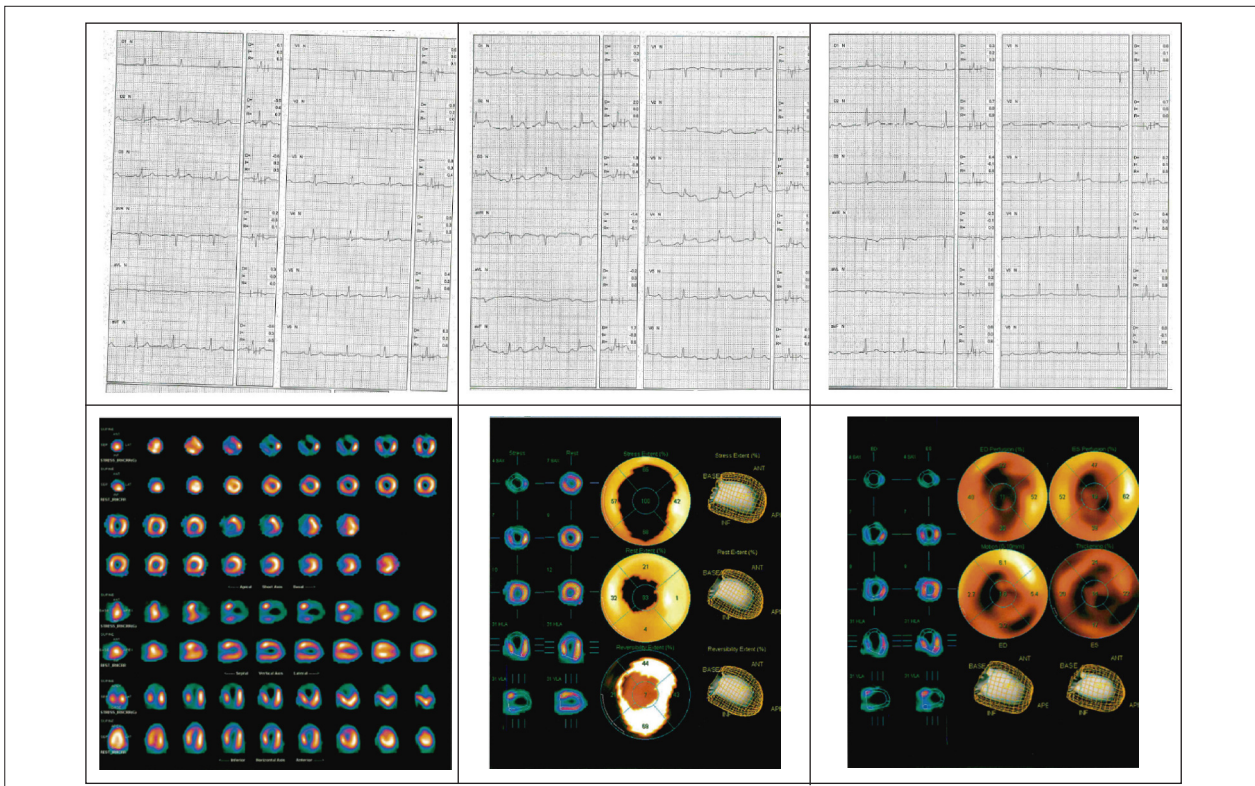


Figure 1 – Myocardial scintigraphy.

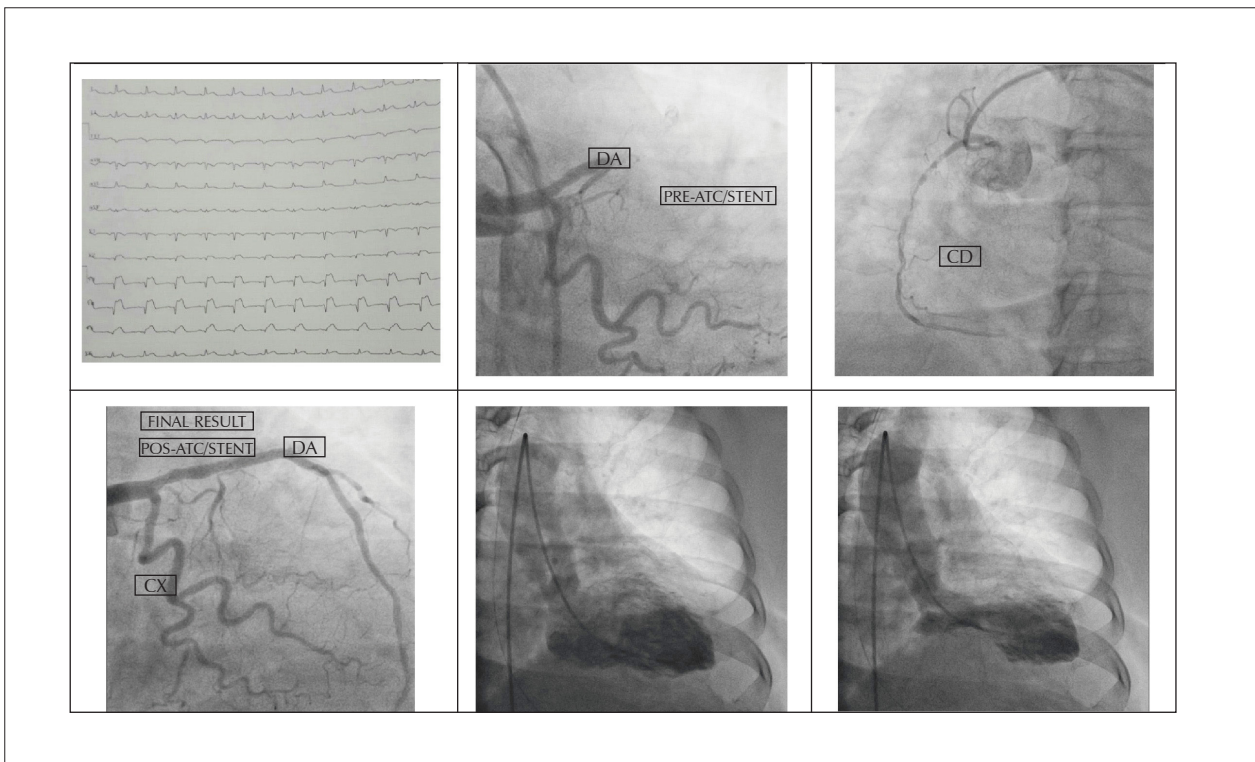


Figure 2 – ECG on admission and coronary angiography.

undergoing dipyridamole stress, the incidence of VT was 0.8 for every 10,000 tests.⁵

Some factors have been associated with ST-segment elevation, including coronary “theft,” viable peri-infarct myocardium, collateral circulation-dependent occluded vessel, and transient changes in heart rate and blood pressure, as well as microvascular dysfunction and diabetes.¹ The use of dipyridamole may increase heart rate by 20–40% and usually causes a slight decrease in systolic and diastolic blood pressure. However, this myocardial demand does not generally lead to transmural infarction.⁶

Dipyridamole-induced ST-segment elevation has been reported in the literature in patients without coronary lesions at the end of the procedure following administration of aminophylline. In this scenario, sudden cessation of vasodilation stimulation leading to coronary vasospasm and the flow-theft phenomenon have been implicated.³ Another possibility that could cause transmural ischemia during dipyridamole infusion would be severe hypotension caused by the drug.² In a study with 155 patients, of whom 67 underwent stress with dipyridamole alone, headache was identified in 40% of the patients, flushing in 30%, weakness in 22%, gastric discomfort in 10% and dizziness in 6%. A mean decrease in systolic blood pressure of 26 mmHg was observed in patients with no symptoms and 37 mmHg in those with dizziness.⁷

In this case, the mechanism leading to transmural ischemia could not be precisely determined. There were no reports of severe hypotension during the test or worsening of symptoms

after aminophylline infusion. Besides, no vasospasm was found on coronary angiography, which does not rule out, but makes this hypothesis less likely. The event is likely to have occurred due to the phenomenon of intracoronary flow “theft” in a vessel with previous atheromatosis and impaired perfusion pressure. A fact that may corroborate this hypothesis would be occlusion in a single vessel in a non-proximal third, with no description of the presence of collateral vessels or significant atherosclerotic disease in other vessels.

Conclusion

A rare case of AMI associated with significant arrhythmia after dipyridamole stress test has been reported. This finding further underscores the importance of proper monitoring of symptoms and ECG in patients undergoing pharmacological stress testing.

Authors' contribution

Research creation and design; data analysis and interpretation; manuscript writing: Gomes RAF. Data acquisition: Macêdo Junior ARA. Manuscript writing: Moreira ICAM, Macêdo Junior ARA. Critical revision of the manuscript for important intellectual content: Cardoso OGM, Melo AGS.

Conflict of interest

The authors declare that there is no conflict of interest regarding this manuscript.

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