Primary percutaneous coronary intervention in the presence of acute pancreatitis following rupture of Wirsung duct

Intervenção coronária percutânea primária na presença de pancreatite aguda após rotura de ducto de Wirsung

Paulo César Gobert Damasceno Campos¹, Luís Sergio Affonso de André Júnior², Breno de Oliveira Almeida³, Artur Berti Ricca⁴, Elias Knobel⁵

ABSTRACT
We describe the case of a patient who developed acute coronary syndrome with hemodynamic instability in the presence of acute pancreatitis complicated by rupture of Wirsung duct. Primary percutaneous coronary intervention of a totally occluded left anterior descending artery was performed. We considered a pathophysiology based on the occurrence of possible coronary vasoconstriction induced by somatostatin, which was prescribed to this patient, and probable coronary atherosclerotic plaque instability mediated by elevated levels of C-reactive protein. To our knowledge this is the first report of primary percutaneous coronary intervention in a case of acute pancreatitis with rupture of Wirsung duct.

Keywords: Coronary disease; Acute disease; Pancreatitis; Pancreatic ducts; Percutaneous coronary intervention

INTRODUCTION
Electrocardiographic changes and cardiovascular manifestations of acute pancreatitis (AP) have been described in the literature; however, the pathophysiology of such events remains to be completely elucidated (1). Acute pancreatitis may mimic acute coronary syndromes, and both conditions may occur simultaneously. In this case, we describe a patient with pancreatitis complicated by rupture of the Wirsung duct, who developed an acute coronary syndrome and required emergency cardiac catheterization and percutaneous revascularization. This is the first report of successful coronary angioplasty using a pharmacological stent in the presence of acute pancreatitis complicated by rupture of the Wirsung duct. We have to consider the possibility of cardiovascular events in the course of acute pancreatitis. Management is difficult and should be individualized.

CASE REPORT
A 51-year-old man was admitted to hospital due to recurrent episodes of epigastric pain, nausea and vomiting. He had no significant past medical history. The heart rate (HR) was 92 beats/min, blood pressure was 144/92 mm Hg and cardiovascular and pulmonary
physical examinations were normal. There was diffuse abdominal tenderness but no rebound or guarding. Laboratory tests showed a serum amylase level of 7453 U/L, serum lipase level of 12180 U/L, C-reactive protein (CRP) of 3470 mg/dL. Abdominal computed tomography (CT) with intravenous (IV) contrast demonstrated mild diffuse pancreatic edema, a small area of necrosis and inflammation over the pancreatic body-tail transition. An endoscopic retrograde cholangiopancreatography revealed a partial rupture of the Wirsung duct with formation of a small distal pseudocyst (figure 1). Upper GI endoscopy showed only mild gastritis. Proton pump inhibitors, IV fluids and somatostatin were introduced for initial medical management.

Eighteen hours later the patient suddenly became restless, pale and diaphoretic. His BP dropped to 70/39 mm Hg and his HR was 140 beats/min and regular. An electrocardiogram showed new ischemic T wave changes in the anterior leads. An emergency coronary angiography showed total occlusion at the mid-left anterior descending (LAD) artery (figure 2). A primary percutaneous transluminal coronary angioplasty (PTCA) was performed using two sequential 30 x 33 mm and 25 x 28 mm Cypher stents with post-stent TIMI grade III flow (figure 3). Clopidogrel and aspirin were started and a Doppler echocardiogram revealed minimal apical hypokinesis with left ventricular ejection fraction of 58%.

The patient evolved well, with no recurrence of abdominal symptoms. He started oral liquid diet with good tolerance, and was able to advance food intake to solid diet over the following week. Serum levels of lipase, amylase and CRP gradually normalized. A follow-up abdominal CT showed necrosis and inflammation corresponding to the pancreas pseudocyst (figure 4). Due to the occurrence of an acute
coronary syndrome (ACS) that required antiplatelet agents and to clinical gastrointestinal improvement, it was decided to maintain medical treatment for pancreatitis. The patient did well and was discharged 10 days after hospital admission.

C-reactive protein (CRP) was implicated in the inflammatory pathway of atherosclerosis, unstable angina and poor prognosis in coronary artery disease (CAD)\(^9\). Recent studies have suggested CRP may also be involved in plaque instability and acute cardiac events\(^{10}\).

We postulate the combined effects of very high levels of CRP and the use of somatostatin may have triggered the sudden development of ACS in this patient.

Our patient did not have any hemorrhagic or local pancreatic complication although receiving antiplatelet therapy with clopidogrel and aspirin.

In conclusion, we described the first report of ACS requiring primary percutaneous coronary intervention in the presence of AP complicated by rupture of the Wirsung duct.

ACKNOWLEDGMENT
We would like to thank Dr. Adriano Tachibana and Dr. Alexandre Parma from the Department of Radiology of Hospital Israelita Albert Einstein for their assistance and technical support regarding imaging data (CT, ERCP).

REFERENCES

DISCUSSION
Cardiovascular complications of AP were described in the literature\(^1\). Multiple pathophysiological mechanisms, including a myocardial depressant factor, shock state, cardiac toxic effect by pancreatic enzymes, metabolic derangements, coagulation abnormalities, exacerbation of underlying cardiac disease and coronary hypoperfusion were postulated\(^{1-3}\). Myocardial infarction in the setting of AP was reported, including a case of thrombolysis and performance of elective PTCA/stent\(^4\).

The rupture of the Wirsung duct is very rare and is usually due to abdominal trauma; it often requires surgical management\(^{5,6}\).

The use of somatostatin, a hormone that inhibits exocrine pancreatic secretion, demonstrated conflicting results in AP\(^7\). However it can cause vasoconstriction, elevation in vascular resistance and impairment in cardiac function\(^8\).