Ramipril-induced acute pancreatitis
A case report and literature review

It is estimated that 1.4–2% of cases of acute pancreatitis are drug induced. Drug-induced pancreatitis is usually mild to moderate in severity, but rare cases of fulminant drug-induced acute pancreatitis have been reported. The case is presented of a patient with acute pancreatitis due to ramipril, an angiotensin-converting enzyme inhibitor. Discontinuation of the drug usually leads to disappearance of symptoms and normalization of the biochemical and radiological parameters of acute pancreatitis, as in this case.

INTRODUCTION
Drug-induction is estimated to be the cause of acute pancreatitis in 1.4–2% of cases. Drug-induced pancreatitis is usually mild to moderate in severity and tends to disappear after drug discontinuation, but rare cases of fulminant drug-induced acute pancreatitis have been reported.1 The case is presented of a patient with acute pancreatitis caused by the use of ramipril, an angiotensin-converting enzyme inhibitor.

CASE PRESENTATION
A 37-year-old female with type 1 diabetes mellitus (DM) and recently diagnosed nephrotic syndrome presented at the emergency department with epigastric pain which radiated to her back, accompanied by nausea and vomiting. The symptoms had begun two weeks earlier, but had worsened over the last 4 days. The patient did not drink alcohol or use any illegal drugs.

The laboratory tests were normal, apart from raised serum amylase (697 U/L), and urine amylase (1,926 U/L). The serum levels of triglycerides and IgG4 were within the normal range. Abdominal magnetic resonance imaging (MRI) revealed enlargement in the body and the tail of the pancreas, along with opacification of the peri-pancreatic tissue, findings compatible with pancreatitis (fig. 1). Neither abdominal ultrasound (US) nor the MRI showed any gallstones. Drug-induced pancreatitis was suspected. The patient had been taking ramipril, an angiotensin-converting enzyme inhibitor, at a dose of 2.5 mg/day for 4 weeks, because of the occurrence of nephrotic syndrome. Ramipril was stopped and candesartan 16 mg/day was administered for the nephrotic syndrome. The symptoms disappeared, and the patient was discharged from the hospital with serum and urine amylase levels within the normal range and free of abdominal symptoms. She remains asymptomatic 3 months after her hospitalization.

COMMENTS
Ramipril-induced pancreatitis has been reported only...
three times, although other angiotensin-converting enzyme inhibitors have been associated with acute pancreatitis. Angiotensin II receptor blockers have been associated with pancreatitis very rarely; specifically, 3 cases with losartan, 2 with imbesartan and one with telmisartan have been recorded. Angiotensin-converting enzyme inhibitors are known to affect the kallikrein-kinin system, resulting in intrapancreatic accumulation of bradykinin, thought to be caused by pancreatitis. Angiotensin receptor blockers have no effect on the kallikrein-kinin system and thus the mechanism by which they cause pancreatitis remains unclear. In experimental animal models, the administration of angiotensin receptor blockers decreases pancreatic damage and improves the biochemical and histopathological parameters of pancreatitis.

In conclusion, in the absence of other apparent causes, angiotensin-converting enzyme inhibitors should be sought as a cause of acute pancreatitis. Discontinuation of the drug usually leads to disappearance of symptoms and normalization of the biochemical and radiological parameters of acute pancreatitis.

Figure 1. Abdominal magnetic resonance imaging (MRI) in a 37-year-old female with epigastric pain, depicting enlargement of the body and the tail of the pancreas, and opacification of the surrounding tissues.
References


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