

Can Roxithromycin and Betamethasone Induce Acute Pancreatitis? A Case Report

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ABSTRACT

Context Acute pancreatitis has been reported in a few cases treated with macrolides or glucocorticoids.

Case report We report the case of a 58 year old patient who, after 2 days of treatment with roxithromycin and betamethasone, manifested acute pancreatitis. Other causes of the disease were ruled out. No re-occurrence of pancreatitis was observed in a 16 month follow-up.

Conclusion Our case sheds new light on glucocorticoid pancreatotoxicity and confirms the role of macrolides as potential pancreatotoxic drugs.

INTRODUCTION

Among macrolides, erythromycin [1, 2, 3] and less frequently clarithromycin [4, 5], have been responsible for drug-induced pancreatitis. To our knowledge, roxithromycin has only been involved in two cases of pancreatic disorders [6, 7]. Regarding glucocorticoids, their pancreatotoxicity still remains controversial despite several reports [8, 9].

In the following, we report the case of a possible roxithromycin- and betamethasone-induced acute pancreatitis occurring during

the treatment of an upper respiratory tract infection.

CASE REPORT

A 58-year-old man was admitted to the hospital in November 2001 presenting severe epigastric pain with dorsal and lumbar irradiation. Two days before admission, he had started taking roxithromycin (300 mg per day) and betamethasone (4 mg per day) for an upper respiratory tract infection. He had no notable past medical history except for having had an operation for a herniated disk in 1982. There was no history of alcohol or tobacco use nor had he had cholecystectomy; he had not undergone any other drug therapy and had never suffered from pancreatic disorders before.

Serum amylase and lipase were significantly elevated at 631 UI/L (reference values: 10 to 55 UI/L) and 1,240 U/L (reference values: less than 190 U/L), respectively, and CRP was at 14 mg/L (reference values: less than 2 mg/L). Other causes of pancreatitis such as hyperlipidemia and hypercalcemia, were excluded, and hepatic, renal and thyroid functions were found normal. Viral serodiagnostics for Epstein Barr virus, cytomegalovirus, hepatitis B virus, hepatitis A virus, hepatitis C virus, HIV and coxackie were negative. Bacterial serodiagnostics for *Salmonella*, *Yersinia* and *Mycoplasma* were also negative. Abdominal ultrasonography

performed upon admission showed a hypoechogenic swollen pancreatic head (diameter: 35 mm) with a blurred contour but no abnormality of the biliary tracks. The computed tomography scan performed on the following day showed a harmonious moderate hypertrophy of the pancreatic head without any focal damage or collection. No irregularities existed regarding the main biliary track. Mild hepatic steatosis was noted after contrast medium injection. A nuclear magnetic resonance cholangiopancreatography was not performed since the clinical features were sufficient to confirm acute pancreatitis.

The patient's clinical state quickly and spontaneously improved in the 48 hours following cessation of roxithromycin and betamethasone. Serum amylase and lipase returned to normal within a few days. A delayed endoscopic ultrasonography did not show any evidence of chronic pancreatitis, congenital abnormality of the pancreatic duct system or intraductal papillary mucinous tumor of the pancreas.

DISCUSSION

In this case, the short delay between roxithromycin and betamethasone ingestion and the onset of the symptoms suggests an iatrogenic origin of the acute pancreatitis presented here. This is confirmed, first, by spontaneous clinical improvement and second, by the normalization of the biological pancreatic parameters upon cessation of both drugs. Finally, all the investigations performed (biological, ultrasonographic, endoscopic) as well as a rigorous questioning of the patient allowed us to exclude any other origin for this pancreatitis. Moreover, a 16 month follow up with no intake of these two drugs showed no reoccurrence.

Erythromycin pancreatic toxicity is described in many reports [1, 2, 3]; however, no specific study has been performed in animals. Roxithromycin has only been reported twice in pancreatic disorders. In the first report, Souweine *et al.* [6] described a case in which roxithromycin was administered immediately

following a 6-day course of erythromycin. In the second report, de La Fuente Aguado *et al.* [7] observed a slight elevation of serum amylase and lipase three days after the introduction of roxithromycin in an HIV positive patient who had been treated by zidovudine and antitubercular therapy.

As for betamethasone and glucocorticoids in general, their pancreatic toxicity remains controversial despite several reports [8, 9].

Our case suggests possible drug-induced acute pancreatitis. It sheds new light on glucocorticoid pancreatotoxicity and confirms the role of macrolides as potential pancreatotoxic drugs.

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Keywords Betamethasone; Case Report; Chemicals and Drugs Category; Pancreatitis; Risk Factors; Roxithromycin

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