Orlistat-induced acute pancreatitis

Orlistat tedavisine bağlı akut pankreatit

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INTRODUCTION

Obesity is a problem of increasing concern worldwide, resulting in a significant rise in the number of prescriptions for anti-obesity drugs. Orlistat is a pancreatic lipase inhibitor licensed for the treatment of obesity (1). Drug-induced pancreatitis is a rare but important cause of pancreatic injury. We present a case of drug-induced mild acute pancreatitis that developed 8 days after starting orlistat.

CASE REPORT

A 43-year-old woman presented to the emergency department with a 12-hour history of upper abdominal pain radiating to the back, with nausea and vomiting. She had no history of any illness, abdominal trauma or traffic accident. She denied using alcohol and her family history was unremarkable. She was super-morbid obese with a body mass index of 52.8, and orlistat had been commenced just 8 days previously for obesity. Her vital signs were normal; the physical examination revealed only obesity and epigastric tenderness without signs of peritoneal irritation. In the laboratory analysis, white cell count was 14800 d/L, hemoglobin 12.4 g/dl, platelets 212000 /mm³, amylase level 467 IU/L (0-100), lipase 311 IU/L (13-60 IU/L), lactate dehydrogenase 362 IU/L, aspartate aminotransferase 52 IU/L (0-40), alanine aminotransferase 69 IU/L (0-50), and calcium 2.15 IU/L. Renal function tests, bilirubin, alkaline phosphatase, gamma-glutamyl transpeptidase, serum electrolyte levels, and lipid profile were in normal limits. Abdominal ultrasound revealed a normal-walled gallbladder containing a 3x5 cm calculus with no evidence of hepatobiliary duct dilatation. Her abdominal computed tomography and magnetic resonance imaging (MRI) demonstrated peri-pancreatic edema and inflammation (Figure 1a, b) and the gallstone (Figure 1c, d). Magnetic resonance cholangiopancreatography (MRCP) showed a large single gallstone without intra- or extrahepatic bile duct dilatation (Figure 1d). She was diagnosed as having mild pancreatitis using the modified Glasgow score 1984, and supportive medical therapy with intravenous fluids and analgesia was applied. Serum amylase normalized within 3 days of admission. The patient was discharged home and advised to try alternative weight-reducing strategies and to avoid orlistat. In the follow-up period of 4 months, the patient had no abdominal pain. She underwent elective cholecystectomy at the fifth month of follow-up.

CONCLUSION

Drug-induced pancreatitis accounts for 2% of all cases with pancreatitis (2). According to our patient and the others mentioned in the literature, it can be suggested that the clinical course of orlistat-induced pancreatitis is mild (3). Previously, a few cases were presented with acute pancreatitis attributed to short-term orlistat therapy (at the 2nd, 4th and 10th days of orlistat; similarly, our case presented on the 8th day of therapy).
Coexisting diseases rather than obesity could be important in orlistat-induced acute pancreatitis. In the literature, one of the patients had a history of a previous episode of alcohol-induced acute pancreatitis, and the other had used fluoxetine for an extended period (4). Moreover, our patient had a single large gallstone but without any signs of hepatobiliary obstruction pattern. Another patient presenting with acute pancreatitis with normal level of serum amylase had diabetes mellitus, hypertension, asthma, and obstructive sleep apnea (3).

Although orlistat is generally believed to be safe, clinicians should remain vigilant for the clinical features of pancreatic injury, especially in patients with pre-existing risk factors for pancreatitis.

REFERENCES


