Georgian Scientists
ქართველი მეცნიერები
Vol. 8 Issue 4(1), 2026
https://doi.org/10.52340/gs.2026.08.04(1).17

TOXIC PANCREATITIS AND HEPATOTOXICITY: PATHOGENETIC LINKS BETWEEN ORGAN INJURIES

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Background. Ethanol can injure the pancreas and liver through overlapping mechanisms, including oxidative and non-oxidative metabolism and cytokine-mediated inflammation, yet the organs are often assessed separately. We describe a case in which pancreatitis and hepatic involvement after heavy alcohol intake were detected by routine testing before typical pancreatic complaints developed.

Case. A 46-year-old man with hypertension, coronary artery disease after bypass grafting, peptic ulcer disease and reported hepatitis C (anti-HCV rapid test negative), drinking alcohol daily for one year, presented after two days of severe weakness, dizziness, vomiting, black stools, epistaxis and intermittent disorientation following excessive alcohol intake, mainly spirit; he reported hallucinations. Hemodynamic and respiratory parameters were stable, with hepatomegaly and a soft, painless abdomen. Endoscopy excluded gastroduodenal bleeding despite tarry stool and a blood-stained nasogastric aspirate. Because of the high risk of seizures, encephalopathy and hepatorenal syndrome, he was admitted to toxicology intensive care, adynamic and drowsy, with mild right hypochondrial and epigastric tenderness and fever (38.5 °C). Pancreatic amylase 391 U/L (reference 13–53) and lipase 2287 U/L (13–60) were measured routinely in view of these risks, with CRP 100 mg/L, AST 100 U/L, GGT 132 U/L and platelets 97 $\times 10^9$/L. Ultrasound showed hepatomegaly with increased echogenicity and biliary sludge; CT showed hepatic steatosis and no pancreatic lesions or peripancreatic changes.

Course. Alcohol-induced acute pancreatitis (K85.2) was diagnosed from lipase about 38 times the upper reference limit, before typical pancreatic complaints appeared. Subsequently, severe girdle-like abdominal pain with distension and radiation to the back developed, and he was transferred to the surgical department. Infusion, analgesic, gastroprotective and symptomatic therapy was continued. Fever resolved, lipase fell to 682 and then 155 U/L, CRP to 38.4 mg/L, and platelets

rose to 234 $\times 10^9$/L. He was discharged after seven days in satisfactory condition with advice on abstinence, diet and gastroenterology follow-up. Additional diagnoses: T51.9 and toxic encephalopathy (G92).

Conclusions. Marked lipase elevation with AST-predominant transaminase rise, raised GGT, hepatomegaly and steatosis after heavy alcohol intake is compatible with combined pancreatic and hepatic toxic injury through shared mechanisms, although a single case cannot establish causation. Pancreatitis was detected by routine enzyme testing before girdle-like pain and distension appeared, in a patient with altered perception; pancreatic enzymes and liver tests should therefore be routine on the basis of the alcohol history, alongside neurological assessment and abstinence counseling.

Keywords: pancreatitis; hepatotoxicity; ethanol; lipase; toxicology.