Dear Editor,

We present a case of acute groove pancreatitis due to isoniazid (INH) occurring three months after treatment initiation. A 74-year-old female had tolerated three months of INH monotherapy (300 mg daily) for latent tuberculosis infection before presenting with severe epigastric pain and non-bloody, non-bilious emesis. Her other medications included mirtazapine, simvastatin, tramadol, ranitidine, colace, senna, fluticasone, vitamin D, and ferrous gluconate. She denied any history of alcohol, tobacco, or drug use. On examination, she was afebrile with epigastric tenderness and guarding, but no rebound tenderness, jaundice or organomegaly. Laboratory examination revealed elevated lipase (167 IU/l) and leukocytosis (17.7 K/Ul); amylase, urea, electrolytes, liver function, and IgG4 were normal. Abdominal ultrasound was normal with no gallstones, while abdominal CT revealed inflammatory changes consistent with groove pancreatitis, a rare segmental pancreatitis. INH was discontinued immediately, and with bowel rest, intravenous fluids, and analgesics, the patient’s symptoms resolved with lipase normalization by hospital day 3. At 219 days follow-up, she had remained off of INH and symptom-free.

INH-induced acute pancreatitis has been previously reported occurring within five weeks of treatment initiation and with non-specific radiographic findings, including by Chow et al. in this journal in 2004. Our report is the first case of groove pancreatitis due to INH, and the first occurring after five weeks of treatment initiation. INH was implicated as the cause based on the lack of more common risk factors, such as alcohol use or gallstone disease; simvastatin was deemed unlikely as the cause because our patient had tolerated it well for several years. Although we did not confirm the diagnosis via INH re-challenge, our patient’s rapid resolution of symptoms in response to discontinuing INH strongly suggests it as the culprit. The mechanism of INH-induced acute pancreatitis is poorly understood, possibly via toxic or immune-mediated effects. Groove pancreatitis is characterised by scarring of the head of the pancreas, the duodenum, and the common bile duct, and is thought due to pancreatic outflow obstruction, which may provide further insight into the mechanism of INH-induced pancreatitis.

Medications are a rare cause of pancreatitis that must be considered when more common causes are ruled out. Contrary to previous reports, our case occurred months after treatment initiation. Practitioners should therefore maintain a high degree of clinical suspicion in patients presenting with abdominal pain in the setting of INH therapy even months after starting therapy.

REFERENCES