

## Intramural Duodenal Hematoma Secondary to Necrotizing Pancreatitis Leading to Gastric Outlet Obstruction

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### INTRODUCTION

Intramural duodenal hematoma (IDH) is a rare condition most associated with blunt abdominal trauma.<sup>1-10</sup> The first reported case was by McLaughlan in 1838.<sup>2,5,8,10-11</sup> Most reported cases have been associated with coagulopathy, iatrogenic procedures (i.e., endoscopic biopsy), and rarely, acute pancreatitis. The rare patient with IDH associated with acute pancreatitis develops a complication of pancreatic necrosis in 20 to 30% of cases. This can be complicated by the development of organized and unorganized peripancreatic edema leading to pseudocysts that become infected and lead to sepsis, increasing the overall mortality by 80%.<sup>4</sup>

Although IDH most commonly is due to trauma, about 30% of cases are attributable to nontraumatic causes.<sup>3</sup> Nontraumatic cases include coagulation abnormalities, anticoagulation therapy such as warfarin, malignancy, and blood disorders.<sup>2-5,8,10-11</sup> Although especially rare, it is possible to encounter nontraumatic IDH as a complication of acute necrotizing pancreatitis.<sup>4</sup> Therefore, if a coagulation profile is normal with no history of anticoagulation drug use or blood disorder, other etiologies such as pancreatic origin should be considered in nontraumatic IDH.<sup>11</sup> This report highlighted a case of acute necrotizing pancreatitis complicated by nontraumatic IDH leading to gastric outlet obstruction.

### CASE REPORT

A 41-year-old male with history of chronic pancreatitis secondary to alcohol abuse presented with acute onset abdominal pain, nausea, and vomiting. He reported daily, heavy alcohol use without any evidence of previous trauma. He denied any recent use of non-steroidal anti-inflammatory drugs or anti-coagulants. His medical history was significant for multiple hospital admissions for alcohol-induced pancreatitis, including three admissions within the prior 60 days. He was discharged most recently two weeks prior to the current admission.

On physical exam, the patient was febrile, tachycardic, tachypneic, and normotensive. He was in mild distress and diaphoretic. Respiratory exam revealed clear lung sounds, and tachypnea with a rate of 26 breaths per minute. However, he was not in any respiratory distress. Abdominal exam revealed epigastric tenderness with guarding and normal bowel sounds. The rest of the examination was unremarkable.

A complete blood count revealed macrocytosis with a hemoglobin of 15 g/dL as well as thrombocytopenia with platelets at 107 UL. Complete metabolic panel revealed bicarbonate of 7 mEq/L, creatinine of 1.33 mg/dL, and BUN of 30 mg/dL. Liver panel showed aspartate transaminase 334 U/L and alanine transaminase 90 U/L, alkaline phosphatase of 143 U/L, total bilirubin of 2.5 mg/dL, and lipase of 1015 U/L. Coagulation profile was within the normal range.

Urinalysis showed ketones with no signs of infections. A previous computed tomography (CT) of the abdomen with contrast done 14 days prior showed hepatic steatosis with no biliary dilation, status post cholecystectomy, peripancreatic fat stranding around the pancreatic head consistent with acute pancreatitis, as well as possible duodenal edema seen at the descending and transverse level (Figure 1). The patient met the criteria for admission based on laboratory and physical findings for acute pancreatitis and alcohol ketoacidosis, but no additional imaging was done on admission. Initial treatment consisted of nil per os, aggressive fluid replacement, pain control, and monitoring for alcohol withdrawal symptoms.



Figure 1. Computed Tomography of abdomen: Duodenum wall edema.

On hospital day two, the patient's respiratory status worsened with acute hypoxic respiratory failure. The patient subsequently was intubated. Chest x-ray showed diffuse bilateral alveolar opacities and broad-spectrum intravenous (IV) antibiotics were initiated for septic shock due to presumed aspiration pneumonia. Three days later, the patient had a 6 g/dL decrease in hemoglobin which warranted an urgent CT of the abdomen to evaluate for possible peritoneal bleed. CT angiography showed a large intramural hematoma of the third and fourth part of duodenum and pancreatic necrosis (Figure 2). The patient was transfused with two units of packed red blood cells and started on IV proton pump inhibitor therapy. Following these interventions, he remained stable during the hospitalization with no progression of his anemia.



Figure 2. Computed tomography of abdomen: Showing duodenal hematoma.

On hospital day seven, the patient was extubated. He complained of persistent nausea and was not able to tolerate anything orally. An upper gastrointestinal series revealed severe constriction of duodenum consistent with gastric outlet syndrome secondary to the IDH (Figure 3). The patient was treated conservatively with resolution of symptoms and was discharged on hospital day 26 on a liquid diet with close outpatient follow up.



Figure 3. Upper Gastrointestinal series: gastric duodenal obstruction is confirmed as no contrast is seen pass duodenum.

## DISCUSSION

Few cases of nontraumatic IDH associated with acute pancreatitis have been reported. Its exact mechanism remains unknown.<sup>2-5,7-12</sup> However, two important mechanisms have been proposed that work simultaneously in the formation of duodenal edema and nontraumatic IDH. The first includes leakage of pancreatic enzyme during an episode of acute pancreatitis causing inflammation, leading to local necrosis, and, subsequently, duodenal wall hematoma formation. The second mechanism is from pressure necrosis caused by pancreatitis, especially in the setting of pseudocyst or walled-off pancreatic necrosis. This leads to the leakage of pancreatic enzymes causing autodigestion that increases permeability of local tissues and blood vessels. Both mechanisms contribute to duodenal wall thinning that causes damage to duodenal blood vessels, eventually leading to duodenal edema and hematoma formation.<sup>2-7,9-10</sup> A periampullary IDH in the second portion of the duodenum may cause obstruction of the pancreatic duct and may be a cause of acute pancreatitis in this setting, as this is a common complication of nontraumatic IDH. This acute episode activates a massive inflammatory response that contributes to formation of organized

and unorganized peripancreatic fluid that could lead to peripancreatic edema and the development of pseudocysts and pancreatic necrosis.

Other causes of hematoma formation are iatrogenic and due to duodenal biopsy via esophagogastroduodenoscopy.<sup>2-5,8,10-11</sup> Ultimately, the distinction between nontraumatic IDH leading to acute pancreatitis or pancreatitis leading to nontraumatic IDH is difficult to assess solely based on imaging, therefore correlating with the patient's history becomes an important factor to speculate on the specific mechanism. Shiozawa et al.<sup>2</sup> included 33 cases from 1981-2010; 11 cases of IDH were secondary to pancreatic disease. Furthermore, about one-third of reported IDH cases had concomitant heavy alcohol use, which raised questions of the impact alcohol may have on the development of IDH. However, the exact mechanism was unclear and poorly reported in literature.

The diagnosis of IDH is best made with CT with contrast.<sup>2-3,12</sup> However, abdominal ultrasound or endoscopy also can be used. A major complication of nontraumatic IDH seen in the reported case was leading to severe gastric outlet obstruction, which is a common complication of IDH.<sup>1,7,10-11</sup> Previous episodes of pancreatitis may have contributed to an early formation of IDH by the mechanism discussed above, however, this was not consistent with the initial CT. Furthermore, stable hemoglobin on admission as well as the late presentation of the gastric outlet obstruction led to a low suspicion of IDH initially. In this case, the sequence of events may have presented inversely of what is seen typically. It was speculated that the late presentation of IDH likely resulted from the release of pancreatic enzymes causing vascular disruption, subsequently leading to pressure necrosis and autodigestion, leading to the formation of the duodenal hematoma which ultimately caused the gastric outlet obstruction. In this case, acute pancreatitis was likely a contributing factor to nontraumatic IDH rather than a result of IDH, especially given the location of the hematoma in the third and fourth portion of the duodenum and the time course of the presentation.

## CONCLUSIONS

IDH complicated with gastric outlet syndrome is treated conservatively by keeping the patient on nil per os and providing intravenous fluid hydration, nasogastric tube for decompression, and parenteral nutrition.<sup>1,7,10-11</sup> Surgical interventions and/or surgical decompression of the hematoma are reserved for complicated cases or following failure of conservative measures.<sup>1</sup> It is important to explore other uncommon causes of nontraumatic IDH such as pancreatic origin if initial imaging and laboratory results are within normal limits. Clinicians should monitor for a sudden decrease in hemoglobin as well as signs of gastric outlet obstruction in patients who present with recurrent alcoholic pancreatitis as this may be due to IDH. Therefore, establishing the diagnosis of nontraumatic IDH requires a good medical history, imaging, and complete laboratory work-up to narrow the differential diagnosis.

## REFERENCES

- <sup>1</sup> Lourenço LC, Horta DV, Reis J. Hepatobiliary and pancreatic: Gastric outlet obstruction from pancreatitis-induced intramural duodenal haematoma. *J Gastroenterol Hepatol* 2017; 32(4):739. PMID:28337787.
- <sup>2</sup> Shiozawa K, Watanabe M, Igarashi Y, Matsukiyo Y, Matsui T, Sumino Y. Acute pancreatitis secondary to intramural duodenal hematoma: Case report and literature review. *World J Radiol* 2010; 2(7):283-288. PMID: 21160669.
- <sup>3</sup> Fukunaga N, Ishikawa M, Yamamura Y, Ichimori T, Sakata A. Spontaneous intramural duodenal hematoma complicating acute pancreatitis. *Surgery* 2011; 149(1):143-144. PMID: 19744447.
- <sup>4</sup> Bodnár Z, Várvolgyi C, Tóth J, Sápy P, Kakuk G. Intramural duodenal hematoma complicating acute necrotizing pancreatitis. *Gastrointes Endosc* 2000; 52(6):791-793. PMID: 11115923.
- <sup>5</sup> Chang CM, Huang HH, How CK. Acute pancreatitis with an intramural duodenal hematoma. *Intern Med* 2015; 54(7):755-757. PMID: 25832937.
- <sup>6</sup> Munoz JC, Salyers WJ Jr. A 47-year-old man with a history of alcohol-induced chronic pancreatitis. 2016. <https://reference.medscape.com/viewarticle/863053.5>. Accessed September 13, 2021.
- <sup>7</sup> Fesenmyer ME, Nelson DB. Intramural duodenal hematoma due to pancreatitis. *J Clin Gastroenterol* 1998; 26(4):350-352. PMID: 9649031.
- <sup>8</sup> Veloso N, Amaro P, Ferreira M, Romãozinho JM, Sofia C. Acute pancreatitis associated with a nontraumatic, intramural duodenal hematoma. *Endoscopy* 2013; 45(Suppl 2 UCTN):E51-52. PMID: 23526514.
- <sup>9</sup> van Spreeuwel JP, van Gorp LH, Bast TJ, Nadorp JH. Intramural hematoma of the duodenum in a patient with chronic pancreatitis. *Endoscopy* 1981; 13(6):246-248. PMID: 7297515.
- <sup>10</sup> Ma JK, Ng KK, Poon RT, Fan ST. Pancreatic-induced intramural duodenal haematoma. *Asian J Surg* 2008; 31(2):83-86. PMID: 18490220.
- <sup>11</sup> Eurboonyanun C, Somsap K, Ruangwannaak S, Sripanaskul A. Spontaneous intramural duodenal hematoma: Pancreatitis, obstructive jaundice, and upper intestinal obstruction. *Case Rep Surg* 2016; 2016:5321081. PMID: 27891286.
- <sup>12</sup> Dubois J, Guy F, Porcheron J. A pancreatic-induced intramural duodenal hematoma: A case report and literature review. *Hepatogastroenterology* 2003; 50(53):1689-1692. PMID: 14571818.

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