CASE STUDY

CONTENTS

Duodenal wall hematoma imitating a pancreatic tumor as a rare cause of obstructive jaundice – a case report

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ABSTRACT

Obstructive jaundice is mostly caused by malignant lesions such as pancreatic head cancer and cholangiocarcinoma. Atypical benign conditions such as intramural duodenal hematoma (IDH), can imitate malignant processes in both clinical and radiological presentation. The diagnostic workup included computed tomography (CT), magnetic resonance cholangiopancreatography (MRCP), endoscopic retrograde cholangiopancreatography (ERCP), and endoscopic ultrasound (EUS) with biopsy. A 65-year-old patient was referred for assessment of obstructive jaundice and endoscopic retrograde cholangiopancreatography (ERCP). Initial presentation included hematemesis, anemia, cholestasis, and coagulopathy. Imaging (MRCP, CT) showed dilation of intra- and extrahepatic bile ducts and pancreatic duct, suggesting obstruction at the pancreatic head. CT also revealed a heterogeneous mass in the duodenum. ERCP demonstrated duodenal stenosis due to external compression and distal bile duct narrowing, and a self-expandable metal stent (SEMS) was inserted. Endoscopic ultrasound with biopsy revealed an intramural duodenal hematoma without malignant features. The patient's medical history was significant for multiple comorbidities and previous anticoagulation therapy. Follow-up imaging demonstrated complete regression of the hematoma, confirming its benign nature. The observed clinical course was consistent with spontaneous resolution; consequently, there was no need for surgical intervention. Intramural duodenal hematoma is a rare but important condition that may mimic pancreatic head tumors causing obstructive jaundice. Early detection using imaging techniques is critical to accurately establish the diagnosis. Early recognition facilitates conservative treatment and helps avoid unnecessary highly invasive surgical procedures.

KEY WORDS: hematoma, duodenum, jaundice, pancreatic neoplasm, endosonography

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INTRODUCTION

Obstructive jaundice is a frequent clinical challenge with multiple possible causes ranging from benign to malignant. Typical malignant etiologies include pancreatic head adenocarcinoma and cholangiocarcinoma with primary neoplastic infiltration of the biliary tree [1]. In contrast, benign disorders frequently involve gallstone disease, chronic pancreatitis, and post-surgical strictures [2]. Among the exceptionally rare and atypical benign conditions of extrahepatic biliary obstruction is the intramural duodenal hematoma [3]. In contrast to neoplastic diseases, this pathology provokes obstruction through an entirely different pathophysiological mechanism, requiring careful consideration in diagnosis [1].

Duodenal wall hematomas are traditionally classified into traumatic and non-traumatic categories. The majority are secondary to blunt abdominal trauma, predominantly in pediatric and young adult populations [4]. Conversely, non-traumatic or spontaneous intramural duodenal hematomas typically occur in patients with specific predisposing conditions [5]. Major non-traumatic risk factors include systemic anticoagulant therapy, primary coagulopathies, and iatrogenic injuries resulting from endoscopic procedures such as biopsies [6;7]. In susceptible patients, spontaneous bleeding originates from the dense submucosal vascular plexus, and progressively accumulates within the relatively confined layers of the intestinal wall [8].

The anatomical features of the duodenum, especially its fixed retroperitoneal location, significantly influence their clinical manifestations. Rapid expansion of a hematoma within the descending part of the duodenum can lead to significant extrinsic mechanical compression of the ampulla of Vater or the distal common bile duct [1]. This external mass effect fundamentally differs

from intrinsic neoplastic duodenal obstruction but can radiologically and clinically mimic a pancreatic head tumor or a cystic neoplasm [9]. Consequently, patients may present with atypical abdominal pain, gastric outlet obstruction, and obstructive jaundice, severely complicating the initial diagnostic workup [10].

Due to the similarities in clinical presentation between this benign hemorrhagic condition and malignant neoplasms, clinicians should maintain a high index of suspicion. Appropriate differential diagnosis is critical to distinguish extrinsic compression caused by a hematoma from a duodenal neoplasm [9]. Most intramural duodenal hematomas resolve completely with conservative medical management [11]. Recognizing that this condition can mimic more serious disease is essential to avoid unnecessary, high-risk surgeries, such as a pancreaticoduodenectomy (Whipple procedure), which carry significant risks and offer no benefit for a resolving hematoma [12].

AIM

The objective of the case is to describe how an intramural hematoma can mimic a pancreatic head tumor and to highlight the importance of multimodal diagnosis in patients with obstructive jaundice.

CASE REPORT

A 65-year-old patient was transferred to the Department of Internal Medicine and Gastroenterology in February 2025 in order to carry out endoscopic retrograde cholangiopancreatography (ERCP) procedure.

Initially, the patient was admitted to a different hospital due to hematemesis, dehydration, and an episode of atrial fibrillation. Laboratory findings showed anaemia, increased inflammation markers and cholestasis.

The gastroscopy was performed. The bleeding resulting from the ruptured vessel in cardiac orifice of the stomach was treated with a hemostatic clip. The MRCP showed significant dilation of the bile ducts. The common bile duct measured 17 mm in width, and a significantly dilated pancreatic duct was noted in the pancreatic body and tail, measuring up to 9 mm. This suggested an obstruction at the level of the head of the pancreas. Due to recognized atrial fibrillation, electrical cardioversion was performed and the chronic anticoagulant therapy (rivaroxaban) was stopped.

Upon admission to the ward, the patient reported weakness, decreased exercise tolerance, insomnia, and stool discoloration. In three and a half months, he lost about 14 kg. The patient's medical history revealed chronic heart failure, persistent atrial fibrillation, type 2 diabetes, hyperlipidemia, nicotine addiction, an umbilical hernia, and colonic diverticula. The patient's treatment regimen included a PPI, metoprolol succinate, ramipril, torasemide, eplerenone, ezetimibe, atorvastatin, empagliflozin, metformin, and enoxaparin. Notably, rivaroxaban was discontinued and replaced with heparin (enoxaparin) to ensure more controlled anticoagulation.

CT scan revealed significant dilation of the intra- and extrahepatic biliary ducts - the right hepatic duct to about 10 mm, the left to about 13 mm, the common hepatic duct to about 12 mm, the common bile duct to about 17 mm. The gallbladder was enlarged to a width of approximately 56 mm. The gallbladder wall remained thin, and no occlusive deposits were visualized within the lumen. Imaging showed an atrophic pancreas. The head and uncinate process contained multiple calcifications (Fig.1), a pattern that is highly characteristic of chronic pancreatitis.

The pancreatic duct was dilated, measuring up to 14 mm. Significant widening of the distal section of the duodenum was observed, reaching a diameter of approximately 49 mm. A protruding heterogeneous solid-necrotic mass measures roughly 44 x 56 mm; this mass demonstrated slight contrast enhancement upon imaging (Fig.2). The ERCP showed a narrowing of the duodenum with external compression and stenosis of the distal part of the common bile duct. The SEMS was placed. EUS imaging and lesion biopsy findings were consistent with a duodenal wall hematoma, with no features of cancer in cytology.

During hospitalization, recurrence of the arrhythmia was observed and beta-blocker therapy was initiated. The patient was qualified for a percutaneous left atrial appendage closure. Following the procedure in March 2025, the patient was enrolled in the SAFE LAAC clinical trial, which assessed the duration and effectiveness of dual antiplatelet therapy after such interventions. In the control imaging studies, regression of the duodenal hematoma was observed. Gastroscopy confirmed inflammatory changes and signs of hemangioma in the medial wall of the duodenum. Laboratory findings revealed anaemia, cholestasis features, prolonged coagulation parameters, and the presence of a lupus anticoagulant (Table 1.) The cancer markers were negative. An increased tendency toward spontaneous haemorrhages was also observed.

Imaging tests identified chronic pancreatitis and duodenal hematoma as the causes of mechanical jaundice. Due to the presence of lupus anticoagulant, antiphospholipid syndrome (APS) was suspected; however, subsequent testing failed to confirm this diagnosis.

In September 2025, the patient was readmitted on schedule for stent revision. He reported abdominal

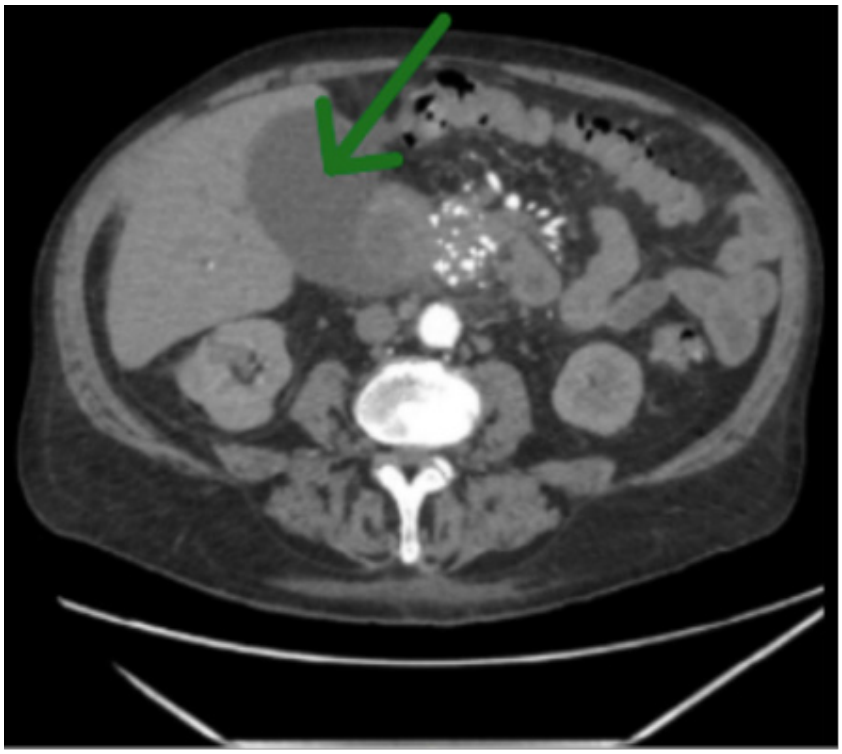

Fig. 1. Abdominal CT: pancreatic calcifications and enlarged gallbladder
Source: Own materials

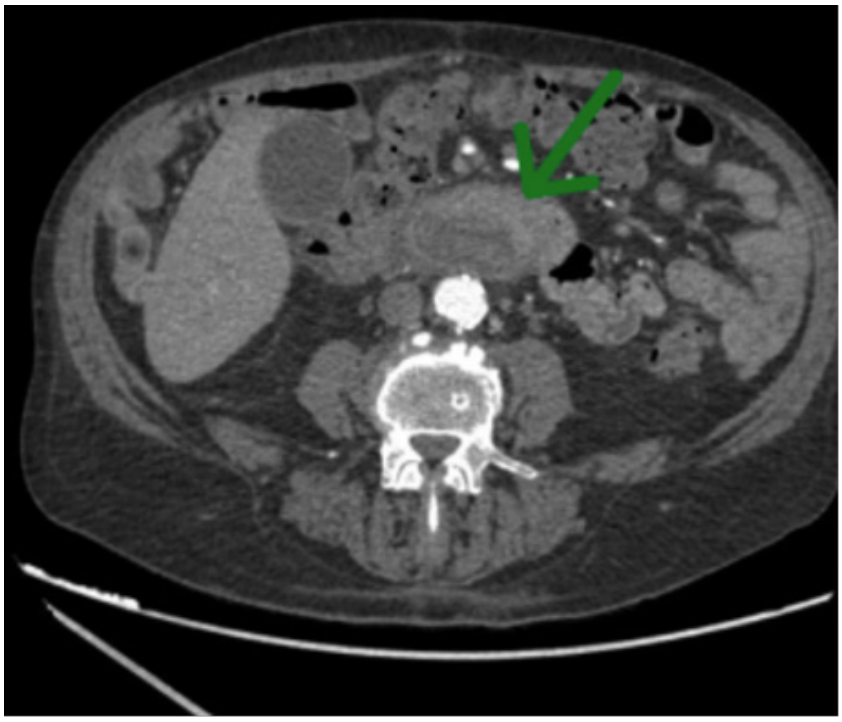

Fig. 2. Abdominal CT: solid-necrotic duodenal mass
Source: Own materials

pain, diarrhoea and further weight loss (a total of approximately 30 kg). CT showed complete regression of the hematoma. The ERCP was rescheduled as the patient had not suspended SGLT-2 inhibitor therapy as required. Gastroscopy revealed a shortening of the duodenal ampulla and deformation of the pylorus. In the prepyloric part of the stomach, the mucosa was focally congested with erosions. Colonoscopy showed intestinal diverticula and two colorectal polyps.

In October 2025, revision ERCP procedure was performed. The SEMS was removed, cholangiography revealed a 15-mm-long stricture of the intrapancreatic portion of the bile duct. Within the stenosis, the bile duct lumen was preserved. The bile ducts were inspected, with subsequent removal of some small food debris into the duodenum. They were irrigated in order to obtain a clear outflow. Due to the observed rapid, spontaneous contrast runoff, bile duct stenting was not recommended.

Table 1. Abnormalities in laboratory findings

Parameter [unit] [reference range]	Value
WBC [10^3/μl] [4,00-10,00]	11,37
HGB [g/dl] [13,00-17,50]	9,8
APTT [s] [20,00-40,00]	48,3
INR [-] [0,80-1,20]	1,43
PT [s] [9,4-13,8]	16,5
Lupus anticoagulant [-] [-]	Positive
ALP [U/l] [40-150]	225
GGTP [U/l] [12-64]	664

Note: ALP, alkaline phosphatase; APTT, activated partial thromboplastin time; GGTP, gamma-glutamyl transpeptidase; HGB, hemoglobin; INR, international normalized ratio; PT, prothrombin time; WBC, white blood cell count
Source: Compiled by the authors of this study

DISCUSSION

Among the many causes of obstructive jaundice, IDH requires careful consideration. Obstruction of the extrahepatic bile ducts is usually caused by more common conditions, such as pancreatic head cancer or choledocholithiasis. However, IDH should be recognized as a benign but potentially serious alternative [10]. In the literature, IDH is most often described as a rare cause of obstruction in the upper gastrointestinal tract. Its coexistence with biliary stricture is therefore unusual and may complicate the diagnostic process [1]. In our case, a patient receiving anticoagulant therapy developed a hematoma in the postbulbar part of the duodenum. This finding adds to the existing literature, where most non-traumatic cases are associated with anticoagulant therapy rather than antiplatelet drugs [13;6]. Similarly to a few previously reported cases, the hematoma in our patient led to secondary biliary stricture, which required appropriate and careful management [12].

The pathophysiology of IDH formation is closely related to the anatomical features of the duodenum [5]. The relatively fixed, retroperitoneal position of this organ and the rich vascular plexus located in the submucosal layer make this area highly susceptible to injury and accumulation of extravasated blood [14]. Historically, blunt abdominal trauma was considered the primary precipitating factor for this condition, especially in the pediatric population [19]. In adults, however, iatrogenic or spontaneous IDH is being recognized more frequently and may result from factors such as systemic anticoagulant or antiplatelet therapy, severe primary coagulopathies, or direct endoscopic interventions [7]. In the presented case, it was the anticoagulant drugs that predisposed the patient to massive intramural bleeding and a secondary mass effect.

The main diagnostic challenge in these cases results from the significant clinical and radiological similarities between an enlarging IDH and neoplastic tumors [9]. With progressive submucosal bleeding, the growing hematoma causes major mechanical compression of surrounding structures [1]. When this compression involves the ampulla of Vater or the distal common bile duct, as in our patient, it may closely mimic a pancreatic head tumor and lead to jaundice and biliary obstruction [10]. In clinical practice, it is important to understand that the following obstruction results strictly from the extrinsic mechanical compression of organs, and not from the actual neoplastic infiltration [12].

The accurate differentiation between a benign hematoma and malignancy depends on the proper use of advanced imaging modalities [9]. Non-contrast CT is the first-line tool, most often depicting an intramural mass that promotes bowel lumen narrowing [13;5]. MRI facilitates the identification of specific blood degradation products, occasionally presenting the pathognomonic "concentric ring" sign [16]. Furthermore, EUS is of pivotal diagnostic value. Its high spatial resolution enables precise visualization of the duodenal wall layers and facilitates the accurate differentiation of an avascular submucosal hemorrhage from a pancreatic parenchymal tumor [17].

An accurate diagnosis determines the management strategy, in which the general clinical approach strongly favors conservative treatment [18]. Management based on bowel rest strategy, correction of coagulation parameters, and temporary drainage allows for the spontaneous resorption of the hematoma [5;7]. In critical situations, the use of a temporary stent allows for the control of obstructive jaundice [3;8]. This intervention was effective in our patient, who had a SEMS successfully removed once free flow of contents was restored. High-burden surgical procedures, particularly the highly morbid pancreaticoduodenectomy (Whipple procedure), are strongly discouraged and should be reserved exclusively for severe complications such as ischemia, bowel perforation, or a persistent inability to rule out a malignant neoplasm [19].

CONCLUSIONS

Intramural duodenal hematoma (IDH) is a rare cause of obstructive jaundice. The ability of the following condition to mimic a pancreatic head tumour clinically and radiologically poses a significant diagnostic challenge. Diagnostic alertness is particularly important in patients with pre-existing coagulopathies or undergoing systemic anticoagulant therapy. These pharmacological and systemic factors can predispose patients to spontaneous submucosal bleeding, as it's clearly demonstrated by the clinical picture of our patient.

Effective differentiation of this mild, external mechanical compression from actual tumor infiltration relies heavily on advanced multi-modal imaging, which provides the necessary tissue resolution to exclude invasive growth. Initial assessment of a bile duct mass and occlusion is possible with CT and MRI, but it is EUS that plays the most decisive diagnostic role in these complex cases. EUS, in conjunction with a biopsy, allows the final identification of the layers of the duodenal wall, which effectively confirms the non-neoplastic nature of the haemorrhagic lesion and prevents serious diagnostic errors.

The main clinical implication of cases like this is that early and accurate IDH detection requires a highly effective conservative treatment strategy based on spontaneous resorption of the hematoma. By recognising the pattern of this condition, clinicians can save patients from the significant morbidity and mortality associated with unnecessary, extensive surgical interventions such as Whipple procedure. Therefore, it is strongly recommended that the priority is given to a thorough review of the patient's pharmacological history and coagulation status whenever they encounter an atypical pancreatic tumour. Ensuring that mild haemorrhagic etiologies have been definitively ruled out before considering invasive options remains the foundation of safe patient care.

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CONFLICT OF INTEREST
The Authors declare no conflict of interest

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