Intramural duodenal hematomas (IDH) have been rarely associated with pancreatic diseases. Conservative treatment is recommended, but course of disease can be life threatening, serious complications may occur (i.e. duodenal perforation) with imperative surgery. The management of diagnostic and treatment in IDH has improved over the years. Computed tomography (CT) and endoscopic ultrasound are excellent tools for diagnosis and follow up of IDH. We report a case of a 31-year-old alcoholic who presented with vomiting, exsiccosis, hypochondriac pain and positive shock index. Esophagagastroduodenoscopy showed gastric outlet obstruction caused by obliterating tumor of bulbus duodeni. Initial suspicion was malign tumor of the duodenum confirmed by native CT and histology. Further diagnostic using EUS-guided aspiration resulted in IDH. By conservative management with nasogastric decompression and digestive rest the patient recovered. In course of disease the hematoma got smaller, but parts were still seen in CT 6 month later.

Case Report
A 31-year-old male presented with exsiccosis, hematemesis and belt-like abdominal pain in the interdisciplinary emergency unit (ED) of our hospital. He reported 3-5 daily episodes of watery, non-bloody diarrhea over a period of 5 days and daily alcohol intake of 1 bottle Vodka until 5 days before presentation. He lost about 5 kg weight in 2 weeks. The patient failed conservative management including medication intake like antibiotic therapy and denied recent travels or sick contact, prior medication intake like antibiotic therapy and denied recent travels or sick contact, prior exposure to health care environments. On presentation, the patient showed a positive shock index (>1/mmHg min) with orthostatic hypotension (systolic pressure: 90 mmHg and diastolic: 55 mmHg) and heart rate increased (heart rate: 100 beats per minute). Physical examination was unremarkable except for the finding of dehydration and tenderness in epigastric region. Laboratory data were pertinent for: leukocytes: 16.96/μL, hemoglobin: 11.5 g/dL; mean corpuscular volume (MCV): 104.3 fl, calcium: 2.11 mmol/L, uric acid: 10.8 mg/dL, creatinine: 2.0 mg/dL, glomerular filtration rate (GFR): <50 mL/min; glucose: 233 mg/dL, albumin: 3442 mg/dL; c-reactive protein: 28 mg/L, aspartate aminotransferase: 95 U/L, alanine aminotransferase: 57 U/L, gamma-glutamyltransferase: 1160 U/L, gamma-glutamyltransferase: 154 U/L, lipase: 1189 U/L, alcohol: <0.1 g/100 mL. Other initial workup including serum thyroid stimulating hormone, hepatitis A, B, C and E screening, electrophoresis, quantitative immunoglobulin’s, stool cultures, stool Clostridium difficile toxin assay, stool ova and parasites was unrevealing as well as the x-ray of the chest. After recovery from acute exsiccosis by application of sodium chloride liquid 0.9% via central vein catheter we undertake additional esophagogastro-duodenoscopy (EGD). The tissue of the esophagus was macerated, coated with hematin and fibrin corresponding reflux esophagitis D (Los Angeles Classification). These lesions were caused by overfilled stomach with nearly 1100 mL liquid aspirated by endoscope. By pushing forward the tissue of bulbus duodeni appeared necrotic and duodenal lumen was completely obliterated by tumor dubious origin with rough, vulnerable surface. We took tissue samples, and to specify the lesion native computed tomography (CT) of abdomen and pelvis was performed. Contrast-enhanced CT was impossible because of reduced GFR and elevated creatinine. The scan showed an extensive, homogenous mass of the duodenal wall from duodenal part I up to II (Figure 1).

Initial differential diagnosis was lymphoma or gastrointestinal stromal tumor (GIST). Secondary finding was an exudative pancreatitis of the pancreas head with little ascites matching to serological elevated lipase. Additional ultrasound of the abdomen with supplemental elastography (acoustic radiation force imaging) revealed fatty liver without sings of liver fibrosis or portal hypertension. As seen in the scan the duodenal wall was thickened up to 5 cm and pancreas head was swollen with little
Case Report

We presented a case of intramural duodenal hematoma in context with alcohol induced acute pancreatitis. The young patient was alcoholic and his condition at presentation was reduced with positive shock index because of exsiccosis. MCV was elevated corresponding to chronic alcoholism. Symptom of diarrhea could have been manifestation of exogenous pancreatic insufficiency (EPI), but determination of elastase as a stool marker for EPI was negative. Lipase was elevated and together with CT and ultrasound results alcohol induced pancreatitis was obvious.

IDH occurs mostly due to blunt abdominal trauma. Causes for non-traumatic hematomas such as the one described are related to the intake of anticoagulants (Warfarin, acetylsalicylic acid), coagulation disorders, malignant diseases, side effects of chemotherapy or inflammatory and autoimmune processes. There are rare reports of an association between IDH and acute pancreatitis.

The pathogenesis of and relationship between IDH and pancreatitis remain unknown. Further it is unclear if pancreatic induced intramural duodenal bleeding or duodenal hematoma is trigger for pancreatitis.

Especially in cases with mild pancreatitis development of duodenal hematoma is difficult to explain. The fixed position of duodenum, its nearness to vertebral column and rich vascularization are anatomic preconditions for IDH especially in context with blunt trauma. For non-traumatic IDH two hypotheses have been postulated. First, the presence of ectopic pancreatic tissue within the wall of the duodenum may develop acute inflammation and subsequent necrosis, and hematoma formation. Second, leakage of pancreatic enzymes in pancreatitis can injure duodenal blood vessels, namely anterior and posterior pancreatico-duodenal arcades. It may cause local necrosis and hematoma formation.

The most common presenting symptoms are abdominal pain, vomiting and successive dehydration or exsiccosis. Because of the intramural bleeding the lumen of the duodenum will be obliterated and gastrointestinal passage of liquid or chime is disturbed. Gastric outlet obstruction occurs. Occasionally, patients may present with jaundice due to bile duct compression by the hematoma. On the other hand the papilla Vateri can be swollen by hematoma and the secretion of the exogenous pancreas enzymes is not possible any more resulting in pancreatitis.

Laboratory tests are of limited value for clinical diagnosis IDH. In general, a high index of clinical suspicion should be contemplated.

As diagnostic abilities, using contrast-enhanced CT and magnetic resonance imaging (MRI) have improved over the last few decades. Because of its availability and cost effectiveness contrast-enhanced CT is recommended over MRI for detection IDH. In our case we performed native CT because of reduced GFR and elevated creatinine contrast-enhanced CT was impossible. But native CT scan was not able to diagnose IDH directly and could have underestimated a life threatening condition retrospectively.

The changes of paradigm in management of IDH were dependent with the advent of contrast-enhanced CT. Reviewed case reports by Sorbello et al. with small bowel hematoma in context with anticoagulant therapy showed that particularly diagnostic surgery intervention declined since CT scan is available as a sufficient diagnostic tool in the mid eighties. The percentage of cases requiring surgical intervention declined from 40% in the 1981-1986 periods, to 28% in the post 1986 period.
In events of enormous intramural bleeding perforation of duodenum occurs and life threatening condition follows. In these situations surgical intervention is necessary. Furthermore surgery is indicated after 2 weeks of treatment without improvement, or in doubt of malignancy.\textsuperscript{4,14}

In our case the results of native CT and EGD gave hints of malignant tumor confirmed by initial histological examination and finally we diagnosed IDH with EUS-guided aspirate of the duodenal wall. Additional EUS is helpful and should used especially for taking biopsies in further diagnostic of duodenal or pancreatic processes.\textsuperscript{2}

In the last decade awareness of non-traumatic IDH has increased. The literature search recommends that current treatment of IDH without complications is conservative by nasogastric decompression, blood transfusion and correction of coagulation abnormalities.\textsuperscript{1}

In our case IDH occurred in context with alcohol induced pancreatitis. Because of his symptoms with shock index over 1 initial intensive medical care was necessary. The gastric outlet obstruction conditioned nasogastric decompression by three luminal, jejunal tube and consisted digestive arrest. Under conservative treatment the patient recovered clinically after few days. But parts of the duodenal wall alteration were still seen in follow-up CTs 5 weeks and 6 month later while panendoscopy showed normal duodenal mucosa without luminal narrowing. Important fact was further exclusion of malignant duodenal tumor.

Conclusions

IDH is a rare pathology. Contrast-enhanced CT of the abdomen and additional EUS are the diagnostic modalities of choice. Non-traumatic IDH can usually be managed with a conservative approach, and surgery should be reserved for complications like abdominal bleeding. Absolutely important is the exclusion of malignant origin.

References