

Intramural duodenal hematoma related to antivitamin K overdose, an unusual cause of acute pancreatitis: Three case presentations

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Dear Editor,

Intramural duodenum hematoma (IDH) is usually caused by abdominal contusion. Non traumatic IDH is rarely reported in adults. It is particularly associated with coagulation disorders, von Willebrand disease, and hemophilia. It can also be related to duodenal biopsy or endoscopic injection for a bleeding peptic ulcer (1).

Few cases of acute pancreatitis due to IDH are reported in literature, especially in a non traumatic setting. The physiopathology of IDH remains unclear.

Here, we report three patients treated by vitamin K antagonist (VKA) who presented with IDH complicated by an acute pancreatitis accident during VKA overdose and compared them other previously reported cases of IDH with acute pancreatitis. Our aim was to clarify the causal relationship between IDH and acute pancreatitis and discuss the management.

The first patient was a 68-year-old man with type 2 diabetes and atrial fibrillation. He complained of epigastric abdominal pain and vomiting. A physical examination showed epigastric tenderness and abdominal ecchymosis. Biological blood tests showed an international normalized ratio (INR) of 7, hemoglobin of 100 g/L, and an elevated serum lipase at 850 UI/L. A computed tomography (CT) scan showed parietal hematoma of D2, increased pancreatic volume without glandular necrosis, and a small amount of inflammatory stranding extending to left pre-renal fat. The patient was hospitalized in the surgical department and received human prothrombin complex (PPSB) infusion, vitamin k, and analgesic treatment. The clinical evolution was rapidly favorable. A CT

scan control 15 days later showed regression of the lesions.

The second patient was a 72-year-old woman with mitral valve prosthesis. She was admitted to our intensive care unit with hemorrhagic shock. Initial laboratory tests showed an INR of 8, hemoglobin of 60 g/L, serum lipase at 1050 UI/L, and C-reactive protein at 250 mg/L. A CT scan showed a parietal duodenal hematoma extended from the superior flexure of the duodenum to the duodenojejunal junction and severe acute pancreatitis with glandular necrosis of the pancreatic gland and retroperitoneal fat necrosis; the modified CT severity index was 10 points. The patient was hospitalized in the department of surgical resuscitation for severe acute pancreatitis (clinically and radiologically). The modified Marshal Score was four. The VKA overdose was quickly corrected by the administration of PPSB and vitamin K. Her hemoglobin level increased 6 hours after she received three units of packed red blood cells. The patient died 48 hours later due to multiple organ failure.

The third patient was a 54-year-old woman who had mitral valve prosthesis. She was admitted for epigastric abdominal pain, vomiting, and hematemesis. A physical examination showed epigastric abdominal tenderness. The biological blood tests showed an INR of 8, hemoglobin of 80 g/L, and serum lipase at 1400 UI/L. Endoscopy showed parietal D2 hematoma. An abdominal CT scan showed multiple small bowel hematomas, including one at the second portion of the duodenum; the pancreas size was increased with infiltration of peripancreatic fat without glandular necrosis. She received PPSB, vitamin k, and analgesic treatment. The evolution was rapidly favorable. The patient was transferred to the cardiology

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department for therapeutic adjustment. A CT scan control showed regression of lesions 7 days later and became normal within 3 weeks.

In our department, 52 patients with gastrointestinal bleeding related to an anticoagulant overdose were treated over 14 years. Seven patients had IDH and three had acute pancreatitis secondary to VKA overdose.

These cases of non-traumatic hematoma are rare (1) and are usually responsible for acute intestinal obstruction. They were first reported by Sutherland in 1904. The main causes of these non-traumatic IDH are anticoagulation or antiplatelet therapy, coagulation disorders, and endoscopic hemostasis (2). Although various pancreatic conditions, such as acute or chronic pancreatitis, are believed to be associated with IDH, the nature of the association remains unknown. Acute pancreatitis related to IDH is probably due to the compression of the pancreas and/or obstruction of the duodenal papilla (3).

A PubMed search by Shiozawak et al. (1) from 1980 onward using the key words IDH and acute pancreatitis identified 32 case reports (2). The association between IDH and acute pancreatitis for these cases was studied. As a result, IDH complicated with pancreatitis was classified into three categories:

(A) acute pancreatitis due to duodenal papilla obstruction by hematoma

(B) hematoma formation due to vascular disruption by pancreatic enzymes released during acute pancreatitis

(C) hematoma formation due to vascular disruption by pancreatic enzymes released during chronic pancreatitis or its acute exacerbation.

In type A, hematoma formation precedes the development of obstructive acute pancreatitis as a complication. Non-traumatic IDH often develops in the duodenal mucosa or sub-mucosa (4). The obstruction of the duodenal papilla is responsible for an impaired pancreatic enzyme secretion, which would lead to an acute pancreatitis.

In our patients, we have retained the VKA overdose induced IDH as an etiology of acute pancreatitis in light of the clinical context. Abdominal ultrasonography did not

reveal vesicular lithiasis. The clinical context and the biological explorations did not evoke alcoholic, iatrogenic, or metabolic pancreatitis. The relationship between the duodenal hematoma caused by VKA overdose and acute pancreatitis seemed certain.

Another major argument in favor of the involvement of IDH in the occurrence of acute pancreatitis is the existence of other digestive hematoma and other hemorrhagic sites. Indeed, if pancreatitis was at the origin of duodenal hematoma, the existence of other hemorrhagic lesions in other sites could not be explained.

Acute pancreatitis secondary to IDH can be very serious and can lead to death. Death was reported in one of our patients who presented with severe pancreatitis and died due to multiorgan failure.

Conservative treatment of IDH usually leads to the improvement of symptoms within 4–8 days (3,5).

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