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Conservative management of intestinal obstruction by isolated intramural duodenal hematoma. A case report and review of the literature

The duodenal injuries occur in the 3-5% of blunt abdominal traumas. The isolated intramural duodenal hematoma is a very rare lesion. An early diagnosis and an adequate therapy are crucial because a delay, beyond 24 hours, increases the mortality from the 11% to 40%. However, diagnosis is often hindered by a lack of specific symptoms.

We report a case of a 21 years-old man with an intestinal obstruction from isolated intramural duodenal hematoma occurred after a blunt abdominal trauma in a sport competition.

The patient was treated conservatively with total parenteral nutrition, gastric decompression and intravenous PPIs. The progressive spontaneous resolution of the hematoma was checked with periodical endoscopies.

The discharge occurred after three weeks with no early complications. No late complications occurred at one-year follow-up. The endoscopy is a good and safe tool in the management of this intestinal obstructions with the possibility of conservative or interventional treatment.

KEY WORDS: Abdominal injuries, Duodenal diseases, Duodenal obstruction, Trauma; Endoscopy

Introduction

The 70% of intramural duodenal hematomas (IDH) occurs as a complication of blunt abdominal trauma in children or young adults and causes a delayed intestinal obstruction. Spontaneous IDHs are commonly associat-

ed with oral anticoagulation or blood diseases. In the traumas, the incidence of duodenal injuries is the 3-5% and the isolated duodenal traumas are very rare occurrences ¹⁻⁴. The incidence of IDH is higher in men (80%). Pancreatitis is often associated or can complicate it. The management of these lesions is still debated and ranges from an operative management to a conservative one.

Case report

A 21 year-old man, after a blunt abdominal trauma that occurred during a sport competition, complaints colic abdominal pain arising in epi-mesogastrium and radiating to the remaining abdominal quadrants, spontaneously resolved.

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36 hours after trauma, for the recurrence of the pain and the occurrence of nausea and vomiting he was admitted in urgency. At the admittance he was hemodynamically stable; the laboratory tests showed normal blood count parameters (WBC 8.600, RBC 5.700.000; Hgb 15.4 g/dl; PLT 180.000); metabolic panel and liver function tests were within normal limits.

The CT scan showed the presence of an intestinal obstruction caused by an hematoma on the right lateral duodenal wall (TD 8 cm, APD 6 cm, LD 11cm), which compressed and displaced the duodenum, without signs of active bleeding. The pancreas head was dislodged to the left; effusion was present around the liver, right kidney and in the Douglas; the liver appeared healthy (Fig. 1).

It was done a gastric decompression and a total parenteral nutrition and intravenous pump inhibitors were administered.



Fig. 1

12 hours after, for the persistence of the abdominal pain, the appearance of a neutrophil leukocytosis (WBC 16.000, N 89.4%, RBC 5.270.000, Hgb 14.7 g/dl, PLT 191.000) and an increases of serum pancreatic enzymes (amylase 1.851 mg/dl; lipase 2.207 mg/dl) it was administered a broad-spectrum antibiotic therapy and octreotide.

At 48 hours from the diagnosis, an esophagogastroduodenoscopy was performed. It showed an extrinsic compression in the first part of the duodenum by a bluish swelling that not allowed the passage in the second duodenal portion and looked as an extra-parietal hematoma (Fig. 2).

It was suspected a peri-pancreatic hematoma that involved the duodenum. The patient followed in a hemodynamic stability, there was a gradual reduction of pancreatic enzymes and progressive blood counts normalization.

To evaluate the presence of coagulation disorders, they were studied the factor IX and VIII, the inhibitor of plasmin, the plasminogen and the VWF antigen, that were normal. Tests on platclet function with PFA 100 system were performed that showed an alteration of hemostatic capacity of platelets for both collagen/epinephrine (closing time of the biological membrane 228 sec; normal <160 seconds) and collagen/ADP (time closing of the biological membrane 131 sec; normal <120 sec). However, the thromboelastogram was essentially normal.

After 5 days, a new esophagogastroduodenoscopy revealed a massive parietal hematoma in the posterior wall of the duodenal bulb, extended up to the Treitz, prominent in the lumen and not hindering the transit. One week later it was performed an ultrasound examination that showed an unchanged extension of the lesion in a pattern of normal hematoma evolution.

Two weeks later, an endoscopic ultrasound was performed in order to evaluate the possibility of endoscopic drainage; it showed a healthy pancreas and confirmed the presence of a rounded lesion with a mixed echo-structure (8 cm







Fig. 3

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Fig. 4

DL; 5.6 cm DT; 6 cm DAP) which seemed localized below the layer 3 of the duodenal wall, surrounded by the remaining layers, and extended from the gastric antrum to the second duodenal portion (Fig. 3).

Due to the substantial delimitation of the lesion, the drainage wasn't performed.

Three weeks later we assisted to a clinical improvement of the patient and a new esophagogastroduodenoscopy showed a complete resorption of the hematoma, so a soft oral diet was started (Fig. 4).

During follow-up, the patient didn't report onset of symptoms worthy of note.

Esophagogastoduodenoscopy, performed one year later, showed no presence of duodenal stenosis and an ultrasound examination showed no signs of chronic pancreatitis.

Discussion

The sports injuries and traffic accidents are usually the etiological factors of the IDH; the possibility of child abuse should always be taken into account. Early diagnoses are often hindered by the lack of specific symptoms and the degree of urgency is frequently underestimated because duodenal lesions are late complications of a blunt trauma¹⁻³.

The traumas are sometimes so trivial that often subjects don't report it, as in our case where the trauma occurred 36 hours before the admission and it was initially not reported by the patient.

The clinical presentation may range from a vague abdominal pain until an acute abdomen with an intestinal obstruction caused by the gradual grow of the hematoma $^{2.5}$.

As part of the duodenal traumas, the intramural duodenal hematoma is categorized according to AAST Organ Injury Scaling Committee in the I-II categories of the less disruptive traumas ⁶.

A prompt diagnosis and an adequate therapy prove to be crucial because a delay in diagnosis and treatment, beyond 24 hours, increases the mortality from the 11 to the 40%.

The laboratory is usually not specific and could show only a slight decrease in hemoglobin and, in case of involvement of the ampulla region, an increase of pancreatic and hepatic enzymes.

Our patient presented, at admission, an intestinal obstruction that was complicated, 48 hours later, by a pancreatitis.

The diagnostic gold standard of IDH is represented by the abdominal CT-scan with the contrast medium, although an ultrasound examination is useful and is often used in the monitoring of the evolution of the lesion. The endoscopic studies and magnetic resonance are performed in selected cases for uncertain diagnosis.

At the beginning of the 70s, the majority of patients that presented a duodenal hematoma were treated with surgical therapy. The surgical interventions ranged from the incision and drainage of the hematoma to the packaging of a gastrojejunostomy, followed or not by a bypass in cases of severely damaged duodenum ⁷.

Today, the literature agrees on the conservative management of the IDH, reserving the operative treatment for persistent occlusions or progressive growth of the hematomas⁸.

The timing for the intervention is still debated; it should be done in emergency in case of suspected perforation, while could be delayed and done after 7-14 days in case of lack of improvement 7 .

The drainage should be considered prior to any laparotomy, the evacuation of the hematoma can be achieved by the CT-guided or ultrasound-guided procedures. It can be performed even endoscopically, laparoscopically or by laparotomy ⁹⁻¹². The arterial embolization may be used to stop bleeding.

As an alternative, conservative treatment, comprising the gastric decompression, parenteral nutrition and antibiotic prophylaxis, can be choosing. Any coagulation disorders should be excluded or treated 7 .

We studied the coagulation profile of our patient that was normal.

If occurs, the pancreatitis must be properly treated ⁷. Medical therapy in our patient improved the inflammation. The use of EUS in combination with contrastenhanced CT has made possible, in our case, an assessment of the exact location of the lesion and the absence of visceral lesions or active bleeding.

The absorption of the hematoma should be monitored with imaging techniques, usually an ultrasound examination can be the choice; however an endoscopic follow-up to monitoring the grade of duodenal obstruction and the resolution of the lesion is, in our opinion, really useful.

We used, and we propose to perform, serial endoscopies to monitor the resolution of the occlusion and, at the same time, with the aid of endoscopic ultrasound, to choose, if there is the need, to drain it.

The outcome of conservative treatment is good with complete resolution of IDH within 2-3 weeks.

Late complications as the duodenal stenosis were never reported but cases of chronic pancreatitis have been described.

Authors' contribution

DF have made substantial contributions to conception, revising it critically for important intellectual content and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved; OG and BEO have made substantial contributions to conception and design, acquisition of data, analysis and interpretation of data; GS has made substantial contributions to acquisition, analysis and interpretation of data; OG and TR have been involved in drafting and writing the manuscript and revising it; GG have given final approval of the version to be published. All authors read and approved the final manuscript.

Riassunto

Il primo caso di ematoma duodenale fu pubblicato nel 1838 da McLaughlan che lo descrisse come una "fatale tumefazione pseudoaneurismatica". Nei traumi chiusi dell'addome l'incidenza delle lesioni duodenali è approssimativamente del 3-5%. Il trauma duodenale isolato risulta essere un'evenienza molto rara. L'ematoma duodenale intramurale (IDH), una delle possibili lesioni duodenali, è solitamente complicanza di un trauma addominale chiuso in bambini o giovani adulti e si presenta solitamente con un quadro "insidioso" di occlusione intestinale alta insorgente circa 48 ore dopo un trauma. L'incidenza è maggiore nel sesso maschile con un rapporto M:F di 5:1. Dato lo stretto rapporto anatomico duodeno-pancreatico, una pancreatite ad eziologia traumatica è spesso associata all'IDH o ne può rappresentare una complicanza.

Presentiamo un caso di occlusione intestinale alta da ematoma intramurale duodenale risoltosi con trattamento conservativo in assenza di complicanze tardive.

La diagnosi di occlusione intestinale da ematoma duodenale intramurale è stata basata sulla storia clinica e sull'evidenza della lesione all'esame TC, confermata dall' EGDS e dall'EUS. Il laboratorio more solito non era specifico. Le possibili strategie di trattamento includevano il trattamento conservativo, l'evacuazione chirurgica e il drenaggio percutaneo o endoscopico. Lo stretto controllo dell'evoluzione della lesione attraverso l'endoscopia tradizionale e l'ecoendoscopia ha permesso di non eseguire trattamenti operativi, che pur si era pensato di attuare. L'atteggiamento conservativo per IDH dovrebbe essere quello di scelta, riservando il trattamento operativo ai casi di persistenza del quadro occlusivo oltre le 3 settimane o ai casi di incremento volumetrico dell'ematoma.

Il risultato del trattamento conservativo è buono, in termini di completa risoluzione dell'IDH generalmente entro 2-3 settimane, soprattutto nei casi diagnosticati precocemente.

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