# Duodenal hematoma – A case report of duodenal atresia following conservative management of a duodenal hematoma

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JL PASIEKA, H MACHIDA, A WONG. Duodenal hematoma – A case report of duodenal atresia following conservative management of a duodenal hematoma. Can J Gastroenterol 1991;5(1):15-17. In children, duodenal hematomas following blunt abdominal trauma are routinely treated conservatively. A case of a two-and-a-half-year-old female in whom conservative management was unsuccessful is presented. At surgery she was found to have an atretic duodenum secondary to the duodenal hematoma. Although uncommon, fibrotic stenosis must be considered in a patient who fails to show resolution of duodenal obstruction following conservative treatment for a duodenal hematoma.

Key Words: Atretic fibrotic segment, Duodenal hematoma, Stenosis

## Atrésie duodénale consécutive au traitement conservateur d'un hématome duodénal

RESUME: Chez l'enfant, l'hématome duodénal résultant d'une contusion de l'abdomen fait couramment l'objet d'un traitement conservateur. On présente le cas d'une fillette de deux ans et demi chez qui ce traitement a échoué. L'intervention chirurgicale a révélé une atrésie duodénale secondaire à un hématome duodénal. Bien que rare, une sténose fibreuse est à envisager lorsque le traitement conservateur ne semble pas corriger l'obstruction duodénale résultant d'un hématome.

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BLUNT ABDOMINAL TRAUMA CAN lead to an intramural hematoma of the duodenal wall resulting in varying degrees of duodenal obstruction and intestinal ileus. The accepted treatment of a duodenal hematoma is nasogastric suction and, when indicated, total parenteral nutrition. Most duodenal hematomas resolve spontaneously within the first seven to 10 days; however, there are isolated cases of duodenal obstruction taking up to 40 days to resolve (1).

A recent review reported that 73% of 62 pediatric cases of duodenal hematomas were successfully treated with conservative management (2). Several of the remaining children required operative intervention because of other intra-abdominal injuries. There were no missed perforations or duodenal strictures in either the surgical or conservative treatment groups.

To the authors' knowledge there are no reported cases of duodenal stenosis or atresia secondary to duodenal hematoma in the English literature. The following case is that of a young girl who presented with a duodenal hematoma secondary to child abuse and went on to develop stenosis and atresia of the duodenum over the course of conservative management.

#### CASE PRESENTATION

A previously well, two-and-a-halfyear-old female was seen in the emergency department with hair loss and bruising of the chest and abdomen. She was observed in the emergency department and sent home the same day. There was a suspicion of child abuse at that time but no evidence was gathered. Two weeks later she re-presented to the emergency department with abdominal pain and bilious vomiting. On physical examination the patient was approximately 5% dehydrated and febrile. There was bruising on her back and over the epigastrium. The abdomen was diffusely tender with moderate rebound tenderness. Bowel sounds were absent. The remainder of the examination was unremarkable. Laboratory data revealed hemoglobin 127 g/L, white blood cell count 23.8 g/L with 85% neutrophils, lipase 473 iu/L and amylase 289 iu/L. Electrolytes, prothrombin time and partial prothrombin time were all within normal limits.

Abdominal x-rays demonstrated a nonspecific gas pattern. Abdominal ultrasound revealed duodenal and pelvic hematomas. The pancreas appeared normal.

The patient was admitted to hospital with a diagnosis of duodenal hematoma and traumatic pancreatitis, thought to be secondary to nonaccidental trauma. She was treated with nothing by mouth and nasogastric suction, and resuscitated with intravenous fluids. Two days later she spiked a temperature of 39°C, hemoglobin had dropped to 89 g/L, and there was occult blood in her stools. At that time lipase was 1134 iu/L. The patient was started on total parenteral nutrition, ranitidine and triple antibiotic therapy (ampicillin, gentamicin and metronidazole). Two days later, when the blood cultures were negative, the antibiotics were discontinued. A computed tomography of the



Figure 1) Initial computed tomography scan demonstrating a duodenal hematoma with a normal appearing pancreas



Figure 2) Upper gastrointestinal series on day 10, showing a duodenal hematoma at the third part of the duodenum with flow distally

abdomen demonstrated duodenal and pelvic hematomas, but was otherwise negative (Figure 1). A negative gallium scan ruled out an intra-abdominal abscess. Over the next few days the

patient's abdomen remained diffusely tender; however, there was no evidence of peritoneal irritation. Conservative management was continued.

By day 10 the abdominal examina-

tion was essentially unchanged. Ultrasound revealed a normal pancreas despite an increase in lipase to 1441 iu/L that morning. An upper gastro-intestinal series revealed a hematoma of the third and fourth parts of the duodenum, with flow beyond the hematoma into the proximal jejunum (Figure 2).

The patient continued to improve clinically, and on day 12 the abdominal examination was unremarkable except for some minor tenderness. During the next three weeks the patient continued to have high volume gastric aspirates and could not tolerate clamping or removal of the tube. After 22 days amvlase and lipase returned to normal. At this time the patient became jaundiced with an elevated bilirubin, aspartate aminotransferase, alkaline phosphatase, and gammaglutamyltransferase, suggesting total parenteral nutrition-induced cholestasis. A repeat upper gastrointestinal series on day 34 revealed a complete duodenal obstruction (Figure 3), and a computed tomography scan demonstrated a normal pancreas with resolution of the pelvic hematoma. Because of the failure of conservative management and the development of total parenteral nutrition-induced cholestasis, the patient was surgically explored.

### SURGICAL FINDINGS

At laparotomy, there was yellow staining of the peritoneal cavity and a green tinge to the liver. The duodenum was dilated from the pylorus to the third portion, with a complete atretic segment between the third and fourth parts of the duodenum. Just beyond the ligament of Trietz there was a serosal tear in the jejunum, and the proximal jejunum was dilated. Further examination revealed a membrane on the anterior wall of the proximal jejunum about 3 cm from the ligament of Trietz. Other than mild fat necrosis around the head of the pancreas, there were no other intra-abdominal abnormalities. A duodenotomy was performed which

confirmed an atretic segment at the junction of the third and fourth portions of the duodenum. An enterostomy was made in the proximal jejunum about 3 cm from the ligament of Trietz. The membrane causing stenosis of the jejunum was biopsied to rule out a neoplasm. This biopsy revealed hemorrhage consistent with ieiunal hematoma. The patient underwent a side-to-side duodenojejunostomy and closure of the proximal jejunal enterostomy. Postoperative course was essentially unremarkable. On discharge the bilirubin, liver function tests, and pancreatic enzymes had all returned to normal.

#### DISCUSSION

Since the early 1970s, the accepted treatment of a diagnosed duodenal hematoma has been conservative, with nasogastric suction and total parenteral nutrition. Prior to the advent of conservative management, many authors advocated early operative intervention to prevent duodenal stenosis; however, there are no reported cases in the literature of a duodenal hematoma causing stenosis or atresia. It has been postulated that the rich blood supply to the duodenum protects it from late stenosis (3). The present case demonstrates that stenosis following a duodenal hematoma can occur and should be considered in the differential diagnosis of a patient who demonstrates continuing or increasing duodenal obstruction with conservative management.

In the present case the duodenal injury was apparently severe enough to cause significant vascular compromise, resulting in the development of a fibrotic atretic segment of bowel. Although there was evidence early in the course of management that the duodenal obstruction was not resolving, the persistence of symptoms was thought to be related to ongoing pancreatitis and secondary inflammatory duodenal obstruction. It became apparent by the fifth week, when clinical and biochemical evidence of



Figure 3) Upper gastrointestinal series on day 34 showing a distended stomach and dilated duodenum with complete obstruction of the third portion of the duodenum

pancreatitis had resolved, that the patient had persistent duodenal obstruction.

In summary, duodenal stenosis and atresia may occur secondary to a duodenal hematoma. Conservative management is the treatment of choice for a duodenal hematoma. However, failure of response or evidence of increasing bowel obstruction after several weeks of conservative management, may necessitate laparotomy.

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