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Intramural Duodenal Haematoma after Endoscopic Biopsy: Case Report and Review of the Literature

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Key Words
Intramural duodenal haematoma · Endoscopic biopsy · Children · Bone marrow transplantation · Leukaemia

Abstract
The development of intramural duodenal haematoma (IDH) after small bowel biopsy is an unusual lesion and has only been reported in 18 children. Coagulopathy, thrombocytopenia and some special features of duodenal anatomy, e.g. relatively fixed position in the retroperitoneum and numerous submucosal blood vessels, have been suggested as a cause for IDH. The typical clinical presentation of IDH is severe abdominal pain and vomiting due to duodenal obstruction. In addition, it is often associated with pancreatitis and cholestasis. Diagnosis is confirmed using imaging techniques such as ultrasound, magnetic resonance imaging or computed tomography and upper intestinal series. Once diagnosis is confirmed and intestinal perforation excluded, conservative treatment with nasogastric tube and parenteral nutrition is sufficient. We present a case of massive IDH following endoscopic grasp forceps biopsy in a 5-year-old girl without bleeding disorder or other risk for IDH, which caused duodenal obstruction and mild pancreatitis and resolved within 2 weeks of conservative management. Since duodenal biopsies have become the common way to evaluate children or adults for suspected enteropathy, the occurrence of this complication is likely to increase. In conclusion, the review of the literature points out the risk for IDH especially in children with a history of bone marrow transplantation or leukaemia.

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Introduction

Intramural duodenal haematoma (IDH) is an uncommon lesion, usually after blunt abdominal trauma in children and young adults. Upper gastrointestinal endoscopy and the diagnostic role of small bowel biopsy in intestinal diseases in children is well established. Complications from this procedure are extremely rare and include perforation and bleeding. In the last years, IDH has been increasingly recognised as a complication of endoscopic biopsy [1]. Especially patients with bleeding disorders [2] and anticoagulation therapy [3], but also malnourished or growth-retarded children seem to be at risk for IDH [4].

The typical clinical symptoms of duodenal haematoma are due to duodenal obstruction. They include abdominal pain and bile-stained vomiting. The haematoma may also lead to obstruction of the papilla duodeni, and pancreatitis and cholestasis may follow. The clinical presentation and imaging techniques, e.g. ultrasound, upper gastrointestinal series, magnetic resonance imaging (MRI) or computed tomography (CT), confirm the diagnosis. Once IDH is confirmed and perforation excluded, conservative management with fasting, total parenteral nutrition and nasogastric suction is possible. Resolution of symptoms generally occurs within 2 weeks. However, the complications, treatment and natural history of duodenal haematomas secondary to biopsies have not been well characterized yet.

We report a case of duodenal haematoma in a child after endoscopic biopsy of the duodenum and review the literature regarding the occurrence and management of IDH after endoscopic biopsies.

Case Report

A 5-year-old girl suffering from chronic graft versus host disease (GvHD) of the skin and gut after stem cell transplantation of severe combined immunodeficiency disease was presented because of failure to thrive and growth retardation. She was growing below the first height and weight percentile for German girls and her BMI was 13.3. She was treated with tacrolimus and topical steroids because of GvHD of the skin. Percutaneous endoscopic gastrostomy (PEG) had been performed 2 years earlier to ensure sufficient enteral nutrition. During the last year there had been no weight gain observable and several weeks before she had begun to suffer from abdominal pain and to vomit frequently. To further clarify her eating disorder and failure to thrive, upper gastrointestinal endoscopy was performed with a nasogastric videoendoscope (Olympus GIF-N180, diameter 4.9 mm). Stenosis of the upper gastrointestinal tract was excluded and small bowel biopsies were obtained from the duodenum and stomach using endoscopic grasp forceps (Olympus SwingJaw FB-231K). No excessive bleeding after biopsy occurred and there was no history of bleeding disorder; laboratory tests performed prior to the procedure showed normal platelet count, prothrombin time, and activated partial thromboplastin time. In addition there was also no family history of bleeding disorder. The gastroduodenoscopy revealed only mild gastroduodenitis with lymphangiectasia of the duodenum (**fig. 1**). Histological examination showed only unspecific mild inflammation of the stomach and duodenum, with isolated lymphangiectasia and normal structure of the villi. In addition also focally increased apoptosis indicating GvHD of the gut was detected in the small intestine.

About 10 h later the patient presented with acute abdominal pain, frequent vomiting and mild haematemesis. Physical examination revealed diffuse abdominal tenderness. The haemoglobin level had decreased by about 3 to 10.4 g/dl; blood transfusion was not necessary. Her platelet count was still over 300,000/μl. During the next hours the level for leucocytes, lipase and gamma glutamyl transferase increased.

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Abdominal ultrasound showed a solid abdominal mass and gastrointestinal series demonstrated a radiopaque material stop in the second part of the duodenum (**fig. 2**). MRI confirmed the presence of a 7-cm-long asymmetrical mass with a maximum diameter of 28 mm located within the second and third duodenal portions consistent with an intramural haematoma (**fig. 3**).

Conservative treatment consisting of total parenteral nutrition, continuous outflow via PEG, and systemic antibiotics was started. Complete resolution of the haematoma was confirmed by ultrasound examination on day 10. At this time enteral nutrition via PEG was restarted with cold liquids, 2 days later hydrolysed food was added first and then normal enteral nutrition was administered via PEG and well tolerated. Control abdominal MRI in Sellink technique on day 22 revealed complete resolution of the haematoma and excluded stricture or disturbed passage.

Discussion

The first case of IDH was published in 1838 by McLaughlan as a 'fatal false aneurysmal tumour' [5]. Since then several cases of this unusual lesion have been reported. Jewett et al. reviewed 182 cases of mostly children and young adults with a median age of 8 years [6]. IDH is usually a complication of blunt abdominal trauma [7–9]. The extraperitoneal position of the duodenum overlying the vertebral column and its tethering to the ligament of Treitz predisposes to injury during rapid deceleration, and it has been postulated that its rich submucosal vascular plexus may easily lead to haemorrhage [10–13]. In addition, bleeding disease [2, 14, 15] and anticoagulation therapy [3, 16] are risk factors even for spontaneous occurrence of IDH [17].

Several cases of IDH following endoscopy have been reported during the last years [10, 18]. Especially children with bleeding disorders or failure to thrive were supposed to be at risk for IDH [1, 15, 19, 20]. The first case after grasp forceps biopsy of the duodenum was reported in 1987 by Ghishan et al. in a 8.5-year-old child with failure to thrive [4].

The incidence of IDH is not known, but estimations are 1:1,250 upper gastrointestinal endoscopies [18]. So far only 28 cases have been published following endoscopic forceps biopsy, with a mean age of 18 years (median 13, range 2–63). More than two thirds were children or young adults, but only 6 had altered coagulation or platelet dysfunction [9, 13]. 7 out of 21 had platelet numbers below 70/μl (**table 1**). Complicated IDH, e.g. perforation, was suspected in two patients [18, 21] and led to immediate surgical intervention. Surgical management including exploration and evacuation was done in another 5 patients [14, 22, 23], including one who underwent ultrasound-guided drainage of the heamatoma [24].

This was the first case of IDH in our hospital during the last 20 years. It is remarkable that the girl did not have any risk factors for IDH such as bleeding disorder or anticoagulation therapy. However she had mild GvHD of the intestinal mucosa, which might also be considered as a predisposing factor [25]. Interestingly 9 patients out of 29 [14, 15, 26, 27] had a history of bone marrow transplantation or oncological disease (table 1); 4 of them died due to their primary disease. Moreover, 7 out of 9 had low platelet counts. Therefore a history of bone marrow transplantation or oncological disease seems to be a relevant risk factor. In addition, it has been suggested that the relatively fixed retroperitoneal third part of the duodenum is more prone to shear injury [28]. To avoid stripping of a great area of the mucosa and tearing of submucosal

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vessels by grasping with the biopsy forceps, it has been recommended to extend the biopsy forceps not more than 2–3 cm from the endoscope [1]. Even following this advice we were not able to prevent IDH in our patient. As mentioned above, failure to thrive was also suggested to be a predisposing risk factor for IDH [4]. However, regarding the published data we cannot confirm this conclusion.

The diagnosis of IDH is likely if symptoms of acute abdominal pain and vomiting occur within 48 h after duodenal biopsy, though also later appearance up to some days or weeks has been described [15]. Laboratory evaluations are unspecific and usually show only mild decrease of haemoglobin concentration. The symptoms are usually caused by duodenal obstruction [10, 12, 29]. If the ampulla of Vater is obturated, elevated amylase and lipase are found, indicating pancreatitis as a frequent complication [7, 12, 13, 18, 24, 30]. Therefore biopsies should not be taken near the papilla duodeni [18]. In our case there was only mild elevated lipase but a continuous retrograde bile outflow via the gastrostomy tube.

Imaging techniques are used to confirm the diagnosis and include upper gastrointestinal series, ultrasound, CT or MRI scan [31–33]. Ultrasound provides immediate information about the presence and the age of the haematoma. It is also used serially to observe the resolution of the haematoma [34]. Upper gastrointestinal series describe well the duodenal obstruction and may also demonstrate perforation. CT or MRI scan with oral contrast medium illustrate the exact extend of the haematoma and may suggest perforation by free fluid or extravasations of oral contrast medium. Since the presence of perforation leads to surgical treatment, these imaging techniques have to be performed immediately. However, the presence of perforation and haematoma has not been reported yet after biopsy, but after abdominal blunt trauma [12].

Regarding the published data a conservative management of IDH should be preferred. It consists of nasogastric suction and intravenous fluids or parenteral nutrition [35]. The outcome of conservative treatment is good, as complete resolution of IDH generally occurs within 2–3 weeks (table 1) [18, 36, 37]. In contrast to other blunt or penetrating duodenal injuries there is no recommendation for immediate surgical intervention [38]. A surgical approach is necessary if perforation is confirmed or suspected and if there is no clinical improvement with conservative treatment, e.g. prolonged bowel rest without resolution of the haematoma. The best time for surgical intervention is still controversially discussed. It must be planned either immediately if perforation is suspected or after 7–14 days due to lack of clinical improvement or increasing parameters of cholestasis (table 1) [39]. Elevated amylase or lipase are often reported. However, in two thirds of patients (12 out of 18) conservative treatment was also successful (table 1) [1, 13, 14, 18, 19, 40, 41]. In all these complicated cases ultrasound is especially useful for monitoring IDH [6, 7, 32]. Several surgical approaches have been made, e.g. simple evacuation of the haematoma, resection of the lateral duodenal wall, exploration of the common duct or gastroduodenostomy [42]. Evacuation of the haematoma seems to be an effective treatment [35] and can also be achieved by a CT [43] or ultrasound-guided drainage procedure [18, 24]. Late complications, e.g. strictures, have not been reported [35], but chronic pancreatitis was described after IDH once [44].

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Conclusion

IDH is a rare complication after duodenal biopsy and occurs mainly in children. Especially patients with leukaemia or after bone marrow transplantation with low platelet counts are at risk for IDH. Every patient with abdominal pain and vomiting following upper gastrointestinal tract endoscopy with duodenal biopsy should be examined for IDH. Early diagnosis is important for appropriate treatment and identification of complications. Therefore MRI or CT scan are the preferred techniques and can be complemented by ultrasound or barium meal study. In most cases a noninvasive treatment is sufficient. Ultrasound is useful for monitoring the resolution of IDH. Evacuation of the haematoma either by ultrasound- or CT-guided drainage should be considered if there is no resolution of the IDH within 7–14 days. The prognosis of IDH is generally good; complete resolution without any complications was found in most cases.

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Table 1. Characteristics of patients with duodenal haematoma after endoscopic biopsy

Case	Reference (first author)	Age (years)/ sex	Indication	Amylase	Lipase	Bilirubin, mg/dl	Platelet count, $\times 10^9$/l	Treatment	Indication	Oral intake
1	Ghishan [4]	8/F	failure to thrive	normal	n.r.	n.r.	285	conservative		d 14
2	Ben-Baruch [20]	6/M	suspected coeliac disease	normal	n.r.	n.r.	n.r.	conservative		n.r.
3	Zinelis [1]	23/M	malabsorption	+++	n.r.	5.1	normal	conservative		d 17
4	Szajewska [10]	11/M	suspected GERD	n.r.	n.r.	n.r.	normal	conservative		d 14
5	Karjoo [41]	14/F	chronic diarrhoea, abdominal pain	++	+++	n.r.	normal	conservative		d 19
6	Lipson [14]	15/M*	abdominal pain, nausea, vomiting	+++	+++	n.r.	404	surgical (d 14)	biliary obstruction, no clinical improvement	
7	Lipson [14]	32/F*†	abdominal pain, nausea, vomiting	+++	+++	11.0	50	surgical (d 21)	biliary obstruction, unchanged size of haematoma	n.r.
8	Lipson [14]	11/M*	abdominal pain, nausea, vomiting	++	++	2.3	31	conservative		n.r.
9	Lipson [14]	36/F*	epigastric pain, substernal burning	n.r.	n.r.	7.3	54	conservative		d 5
10	Ramakrishna [15]	5/F*	abdominal pain, nausea, vomiting	n.r.	n.r.	n.r.	55	conservative		d 41
11	Ramakrishna [15]	12/M*†	diarrhoea	+	+	26.4	65	conservative		–
12	Guzman [18]	13/M	suspected coeliac disease	+++	+++	normal	320	surgical (d 1)	suspected perforation	d 7
13	Guzman [18]	13/F	abdominal/retrosternal pain	++	+++	n.r.	normal	conservative		d 21
14	Worynski [27]	23/M*†	nausea, vomiting	n.r.	n.r.	n.r.	46	conservative		–
15	Sollfrank [23]	56/M	n.r.	n.r.	+++	n.r.	n.r.	surgical (d 12)	increasing parameters of cholestasis and inflammation, pain	
16	Camarero [36]	4/F	suspected coeliac disease	normal	n.r.	n.r.	n.r.	conservative		n.r.
17	Sgouros [19]	32/F	diarrhoea	++	++	n.r	normal	conservative		d 21
18	Lloyd [24]	18/F	suspected coeliac disease	+++	n.r.	n.r.	n.r.	ultrasound-guided drainage (d 15)	no clinical improvement	n.r.
19	Diniz-Santos [13]	6/F	abdominal tenderness	++	+++	n.r.	279	conservative		d 10
20	Chen [37]	39/M	n.r.	n.r.	n.r.	n.r.	n.r.	conservative		d 7
21	Borsaru [9]	10/F	abdominal pain, anaemia	n.r.	+++	n.r.	n.r.	conservative		n.r.
22	Kwon [22]	63/F	haematemesis	+++	n.r.	n.r.	161	endoscopic (d 16)	no improvement	n.r.
23	Chen [26]	17/M*†	epigastric pain	n.r.	n.r.	n.r.	56	n.r.		–
24	Galea [21]	30/M	suspected coeliac disease	++	n.r.	n.r.	n.r.	surgical (d 1)	peritonism	n.r.
25	Dunkin [40]	5/M	suspected eosinophilic oesophagitis	normal	normal	n.r.	normal	conservative		d 6
26	Dunkin [40]	2/F	suspected rejection after small bowel transplantation	+++	+++	n.r.	normal	conservative		n.r.
27	Dunkin [40]	13/M	diarrhoea, failure to thrive	++	+++	9.5	n.r.	conservative		n.r.
28	Antoniou [34]	5/F	suspected coeliac disease	++	n.r.	n.r.	320	conservative		d 10
29	Grasshof (this report)	5/F*	failure to eat, vomiting	n.r.	++	n.r.	388	conservative		d 10

* Patient with leukaemia or after bone marrow transplantation. † Patient died. d = day; n.r. = not reported; GERD = gastroesophageal reflux disease. + = Slightly elevated; ++ = elevation 2–3 times above normal; +++ = elevation 3 times or more above normal.

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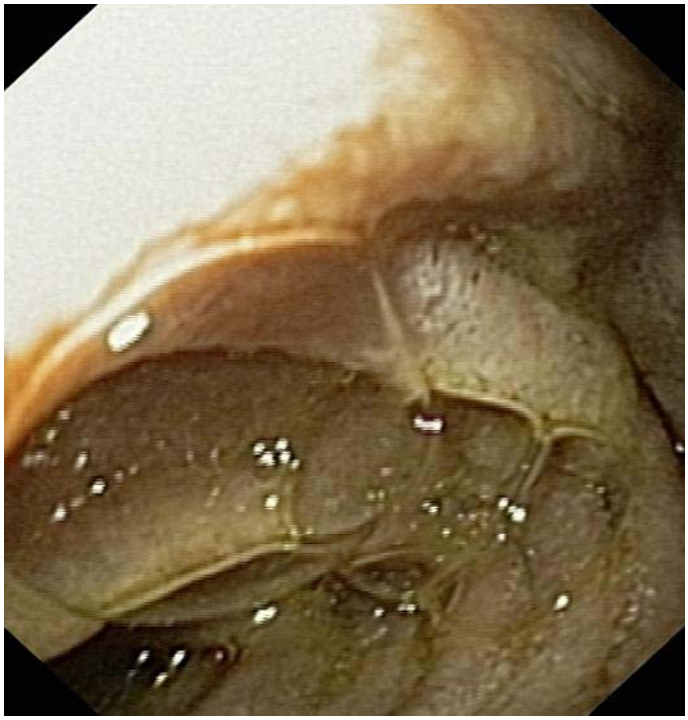

Fig. 1. Endoscopic picture of the duodenum performed with an Olympus N180 video endoscope. The mucosa displayed only mild duodenitis with intestinal lymphangiectasia.

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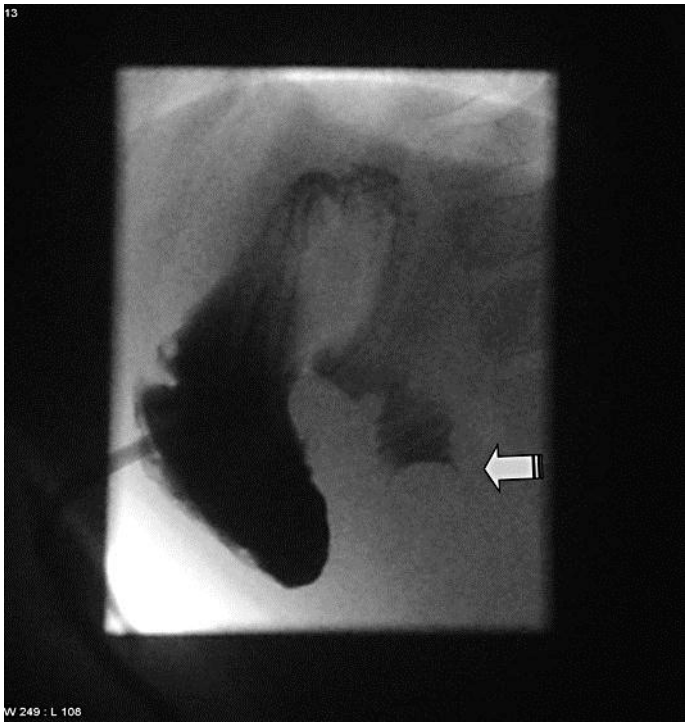

Fig. 2. Upper gastrointestinal series demonstrates complete duodenal stenosis by a sharply demarcated mass (arrow).

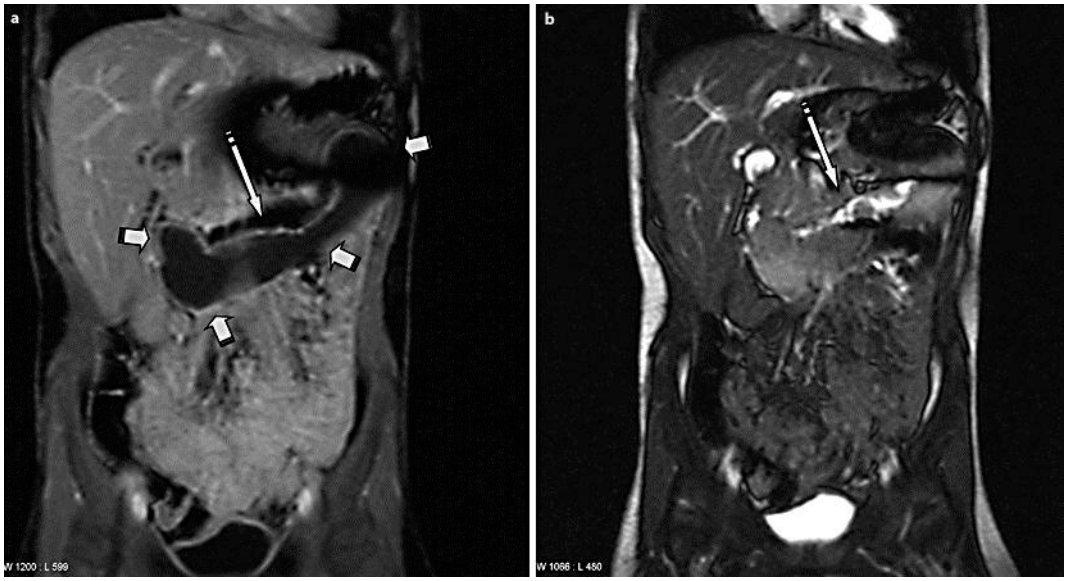

Fig. 3. Coronal MRI of the duodenal wall haematoma. The haematoma is clearly shown as a hypointense lesion in contrast-enhanced T1 Flash 2D (short arrows) (**a**) and moderately hypointense in STIR (**b**). Note the compression of the original duodenal lumen to Treitz ligament (long arrows).

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