

Intramural Hematoma of the Duodenum:

A Review of the Literature and Case Report

WALTER R. JONES, M.D., WILLIAM J. HARDIN, M.D.,
JESSE T. DAVIS, M.D., JAMES D. HARDY, M.D.

*From the Department of Surgery, University of Mississippi Medical Center,
Jackson, Mississippi*

INTRAMURAL hematoma of the duodenum is a clinical entity which has been recognized with increasing frequency during the past two decades. This condition was initially described in 1838 by McLauchlan, whose "false aneurysmal tumor occupying nearly the whole of the duodenum" was noted at the autopsy of a 49-year-old man who died of dehydration and duodenal obstruction.⁴⁷ Only ten cases of duodenal hematoma were reported over the ensuing 100 years.

In 1948 Liverud first described the radiographic findings associated with intramural intestinal hematoma in his report of a case involving the jejunum.⁴³ Later, in 1954, Felson and Levine²⁰ reported four cases of obstructing intramural duodenal hematoma, two of which demonstrated, on upper gastrointestinal contrast roentgenograms, the "coil spring" sign which they considered to be pathognomonic of the disease. Since 1948 one hundred and fourteen cases of intramural duodenal hematoma have been reported in the world literature, bringing the total to one hundred twenty-five. This paper presents a report of an additional case and a critical analysis of the one hundred twenty-five cases reported in the literature.

Case Report

A. F. (#88070), a six-year-old girl entered the University Hospital emergency room with a four-day history of abdominal pain. Four days

prior to admission she had been struck by a playmate in the upper abdomen with a broom handle. No contusions or masses were noted initially. Three days prior to admission the child complained of intermittent crampy abdominal pain which progressed in severity and frequency over the next 72 hours. She vomited six to eight times in the 36–48 hours prior to admission, and she had anorexia for the final 24 hours. There was a history of obstipation for five to six days despite oral laxatives given on the day prior to entry. On the night before admission she had had a "chill," and earlier that day the mother noticed a discrete exquisitely tender mass in the child's epigastric area. She had had two syncopal episodes on standing the day of admission. There was no history of hematemesis, melena, hematuria, epistaxis, diarrhea, familial hematologic disorders, previous medications, anti-coagulant therapy, or previous operations. Systems review, family history, and past history were otherwise non-contributory.

Physical examination revealed a well developed thin colored female in moderate distress with abdominal pain. She lay very still on the examining table, resisting all movement. Vital signs revealed pulse—140/min., respirations—28/min., and temperature—37.2° C. Pertinent positive findings were as follows: Bilateral tonsillar hypertrophy without exudate; no jaundice; marked generalized direct and rebound abdominal tenderness with extremely hypoactive bowel sounds of normal pitch; an exquisitely tender oval 4 × 5 cm. epigastric mass extending into the left upper quadrant; moderate voluntary abdominal muscle guarding; and bilateral pararectal tenderness (right greater than left) with soft hematest negative feces. Diagnostic impression on admission was ruptured spleen vs. rectus hematoma vs. intraabdominal hematoma.

Admission Laboratory Data. Hemoglobin—10.2 Gm.%, hematocrit—30%, white cell count—8,500/cmm. with normal differential, serum amylase—466 Somogyi units, and blood urea nitrogen—40 mg.%. The serum electrolytes, urinalysis, sickle

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cell preparation, and blood glucose were normal.

Radiographic Findings. Routine films of the chest and abdomen showed obliteration of the right psoas shadow. Emergency IVP—no visualization of the right renal shadow, good bilateral excretory function, and an abnormal gastric air shadow with extrinsic pressure defect on the greater curvature. Roentgenograms on the twelfth postoperative day showed distortion of the descending and transverse duodenum with mucosal edema and an area of narrowing in the descending duodenum.

Operation. Exploratory laparotomy was performed on the night of admission. At this time a 4 × 4 cm. subserosal, well organized intramural hematoma was evacuated from the distal second, third, and fourth portions of the duodenum and proximal jejunum by incising the serosa longitudinally and closing it with interrupted 000 chromic suture. A longitudinal 3 cm. jejunotomy was performed distal to the hematoma, and partial intussusception in this area thus reduced manually. The jejunotomy was closed transversely in two layers. The nasogastric tube was next advanced into the proximal jejunum. The pancreas and biliary tract appeared normal.

Postoperative Course. The postoperative course was uncomplicated; the Levin tube was removed on the fourth day and clear liquids were given on the fifth day. Postoperative laboratory data showed prothrombin time—30% and serum amylase—760 Somogyi units. On discharge from the hospital the serum amylase, BUN, CBC, and serum electrolytes were normal. She was discharged on the twelfth postoperative day asymptomatic, and thereafter remained free of symptoms on subsequent out-patient visits.

Incidence

No accurate estimate of the exact incidence of intramural duodenal hematoma has been made. While the disease is being recognized with increasing frequency, it still remains a relatively rare surgical entity. In Perry and Shaw's⁵⁹ analysis in 1893 of the findings at 17,652 autopsies at Guy's Hospital in London, only two cases of duodenal hematoma were found. Poer and Woliver,⁶³ in 1942, reviewed 1,476 cases of nonpenetrating abdominal trauma with 36 of these resulting in significant small bowel injury. Duodenal trauma was recorded in only 8% and no duodenal hematomas were reported. In 1964, Judd *et al.*³⁷ reviewed 75

cases of small bowel hematoma reported in the literature. Of these 75 cases, 36 (48%) involved the duodenum. Most authors^{77, 53} agree that the duodenum is involved in approximately 50% of the cases of intestinal hematoma and probably a higher percentage of the time when trauma alone is the etiologic agent.

The disease more frequently affects males than females. Only 28/123 (22.8%) patients were females. This difference has been attributed to the male's more frequent exposure to situations where physical trauma is likely to occur.

The young are more frequently affected than the elderly. About 51.7% (60/116) occurred in children less than 14 years of age, 63.8% (74/116) in patients less than 20 years of age, and 75% (87/116) in patients less than 30 years of age. The youngest patient reported was in a seven-day-old male infant²⁹ and the eldest a 76-year-old man.⁵

Etiology

Blunt abdominal trauma is by far the most common etiologic factor (Table 1). A history of previous blunt abdominal trauma was elicited in 74.2% (89/120) of cases. Frequently the trauma is so insignificant that it is forgotten by the patient and his family initially. The trauma characteristically occurs several hours to days prior to the onset of symptoms. In 70/124 reported cases, this lag period from abdominal trauma to onset of symptoms averaged 53.6 hours. Devroede¹⁷ pointed out that the trauma is most frequently of the localized type, as was true in our case.

The mode of onset and mortality rate seems to be changing with increasing closed abdominal trauma secondary to automobile accidents. Judd *et al.*,³⁷ first pointed out that duodenal hematoma appeared clinically as two distinct syndromes: One as high small bowel obstruction of insidious onset and, secondly the other as an acute abdominal lesion with obstructive symp-

TABLE 1. *Etiology*

Trauma—	89/124	Alcoholism—	12/124
Clotting defects—	9/124	Hiccoughs—	1/124
Anticoagulants—4		Ruptured aortic aneurysm—	1/124
Blood dyscrasias		Epilepsy—	1/124
Hemophilia—5		Small bowel capsule biopsy—	1/124
Henoch's Purpura		Mesenteric adenitis—	1/124
Unknown—no etiologic factor present—	11/124	Others not mentioned in this series	
Pancreatic disease—	14/124	Duodenal ulcer with hemorrhage	
Pancreatitis or evidence of—9*		Reduplication cyst of the small bowel with assoc. hemangioma	
Pancreatic ectopic tissue—1			
Pancreatic pseudocyst—3			
Pancreatic carcinoma—1			

* 10 other cases exhibited elevated amylase levels without other evidence of pancreatitis.

toms resembling acute appendicitis. The latter onset appears to be increasing in frequency as the severity of the trauma increases, and there is a concomitant increase in associated injuries (duodenal perforations, etc.) and overall mortality. Free-ark's²² recent review of nine cases supports the impression.

Other conditions which have been implicated in the etiology of intramural duodenal hematoma are listed below. Anticoagulant therapy,^{97, 75} blood dyscrasias,^{29, 19, 53, 97, 51} pancreatic disease, and alcoholism^{1, 40, 22, 83, 13, 21, 97} have all appeared in a significant number of cases. Other factors such as severe hiccoughs,²⁰ ruptured aortic aneurysm,^{59, 8} epilepsy,⁸³ mesenteric adenitis,^{91, 76} duodenal ulcer with hemorrhage,² Schoenlein-Henoch's purpura,^{2, 24} and reduplication of the jejunum with associated angiomatous malformation⁹³ have been reported in association with intestinal intramural hematomas.

An interesting etiologic factor was reported by Toblin⁸⁶ who reported a case of intramural duodenal hematoma following peroral small bowel biopsy using a biopsy capsule. An accompanying balloon was inadvertently inflated prior to transoral removal of the capsule from the duodenum, and resulted in the development of an intramural hematoma which required surgi-

cal excision. The shearing force produced on the duodenal mucosa by the inflated balloon and pre-existing areas of duodenitis were both implicated in the development of the hematoma, as the biopsy site was not in the area of hematoma formation.

Pathogenesis

Intramural hematoma has been reported in the esophagus, duodenum, jejunum, ileum, and colon.⁷⁷ Several factors are thought to account for the higher incidence of duodenal involvement. The relatively fixed retroperitoneal position of the duodenum and its adjacent anterior relationship to the lumbar spine renders it susceptible to blunt trauma of a crushing type. The fact that it is frequently filled with liquid contents⁶³ and bound in three areas by the pylorus, aortic-superior mesenteric vascular angle, and ligament of Treitz have led some to consider it to be a physiologically "closed loop," more susceptible to sudden increases in intraluminal pressure seen in abdominal trauma. The work of Williams and Sargent⁹⁶ tends to refute this hypothesis.

Additional factors mentioned include: (1) the rich submucosal vascular plexus in the duodenum, (2) the greatly shortened duodenal mesentery which adds to limitation of mobility, (3) the tangential in-

sertion of the mesentery of the duodenum and proximal jejunum, (4) the varying tensile strength of the three layers of the duodenal wall, and (5) the lack of a completely circumferential well developed serosal layer in the retroperitoneal portion of the duodenum with resultant decreased ability to tamponade active intramural hemorrhage.¹⁷

Several of the latter factors add evidence to the postulate that a shearing force, rather than crushing force, is responsible for intramural hematomas. The discussion by Toblin⁸⁶ supports this premise.

Judd *et al.*³⁷ and others attribute the delay in appearance of obstructive symptoms to a hyperosmotic effect of the liquefying hematoma similar to that responsible for the enlargement of a chronic subdural hematoma.

The prevalence of intramural duodenal hematoma in children has been attributed to two factors—the high, flared costal margin common in children and less well developed abdominal musculature. The latter factor is reemphasized by the prevalence of the disease in alcoholics.

In our case and in two previous reports^{70, 26} partial intussusception of the intestinal mucosa distal to the hematoma was found. As was postulated by Devroede,¹⁷ this phenomenon explains the presence of the “coil spring” sign distal to the intramural hematoma on radiologic contrast study. The “crowding of the edematous valvulae conniventes” mentioned by Felson and Levine²⁰ probably results from the pressure of the leading edge of the intussusceptum. Intussusception has also been reported in conjunction with intramural ileal hematoma arising from Henoch’s purpura.^{2, 24}

Diagnosis

Clinical Findings

History. The classical history of intramural duodenal hematoma is as follows:

The patient is usually a child who has had mild abdominal trauma three to four days prior to admission and who developed intractable bilious vomiting about 24 to 48 hours after the trauma. A history of melena or hematemesis is usually not present unless the patient has a clotting defect or the hematoma has evacuated itself into the bowel.^{19, 37, 34, 53, 88, 97, 51, 29} There may be a history of mild fever, and dehydration is usually present. In cases surveyed, 95.8% (114/119) had symptoms of obstruction. Of five remaining cases, four had acute abdominal signs from acute trauma and were immediately surgically explored.^{22, 7, 17} A history of abdominal pain is always elicited, the pain being in the right upper quadrant when the first, second, and third portions of the duodenum are involved, and in the left upper quadrant when the fourth portion of the duodenum or proximal jejunum are involved. There may also occasionally be testicular and scrotal pain if a retroperitoneal hematoma is present¹⁷ and shoulder pain if free peritoneal blood is present.⁸ Weight loss has been reported in more chronic instances.^{4, 5} The patient may complain of constipation but passes flatus without difficulty.

Physical examination usually reveals a lethargic dehydrated child with direct abdominal tenderness and frequently a tender upper quadrant abdominal mass. The mass occasionally becomes palpable after admission to the hospital or after induction of general anesthesia. An abdominal mass was palpable in 40% (44/110) of cases reviewed. Jaundice was present in 6% (7/116). There is no abdominal distention and bowel sounds are usually normal unless there is free peritoneal blood. Abdominal guarding is usually present. A “gastric splash” may be detected in the region of gastric dilatation⁷³ and the psoas sign is occasionally positive. On at least one occasion, the hematoma was palpable on rectal examination. The stool is usually hematest

TABLE 2. *Associated Biliary and Pancreatic Disease*
40/116 (34.5%) patients had signs of biliary and/or pancreatic disease

19/116 (16.4%)—Biliary Involvement	30/116 (25.9%)—Pancreatic Involvement
9/116—Distended common bile duct and gallbladder	9/116—Pancreatitis at surgery
7/116—Jaundice	19/116—Elevated serum amylase
1/116—Alcoholic cirrhosis	6/116—Fat necrosis
9/116—Increased serum bilirubin	3/116—Pancreatic pseudocyst
1/116—Fatty liver	1/116—Peripancreatic hematoma
	1/116—Pancreatic carcinoma
	1/116—Ectopic pancreatic tissue in duodenal wall with a cystic pancreas

negative. Contusion of the abdominal wall is rare.

Laboratory findings reveal a leukocytosis of 10,000 to 30,000 cells/cmm.⁵³ Anemia was present in 23% (20/86) of cases, but hemoconcentration secondary to dehydration was the rule. A hemolytogram should be performed to rule out clotting defects. Increased serum bilirubin was present in 7.8% (9/116) of cases, arising from a combination of hemolysis and ampullary obstruction from the hematoma. An elevated serum amylase was found in 16.4% (19/116) of cases, resulting from direct pancreatic trauma, ampullary obstruction by hematoma, or a combination of both. Serum electrolytes indicated a typical hypochloremic, hypokalemic metabolic alkalosis when the vomiting was protracted.

Radiologic evaluation of intramural duodenal hematoma should be diagnostic in most instances. Roentgenograms may show gastric dilatation, air or air-fluid levels in the proximal duodenum with no air in the remainder of the small bowel, extrinsic pressure defect on the greater curvature of the stomach, air contrast filling defect in the duodenum, obliteration of the psoas shadow, or abdominal mass.⁴³ Barium contrast roentgenograms may show an intraluminal filling defect in the duodenum and the diagnostic "coil spring" sign of Felson

and Levine.²⁰ Upper gastrointestinal x-rays were taken in 61.6% (66/107) of patients and 39.3% (42/107) were diagnostic, revealing the "coil spring" sign. An additional 19.6% (21/105) showed duodenal obstruction. X-rays may also show a "picket fence" configuration^{37, 72, 97} with or without a coil spring sign. This pattern has been frequent in association with intramural intestinal hematomata resulting from clotting defects with anticoagulant therapy or hemophilia. Intravenous pyelogram may show ureteral displacement,⁸² and in several instances, an IV cholangiogram has been used to demonstrate obstructive changes in the biliary tract.³⁴

Differential Diagnosis. Primary diagnostic considerations are intestinal intussusception and ruptured viscus. Diagnostic possibilities included acute appendicitis (7/101), acute surgical abdomen secondary to trauma (7/101), perforated hollow viscus (6/101), retroperitoneal hematoma (4/101), abdominal mass (3/101), pancreatitis (3/101), intraperitoneal hemorrhage (2/101), peritonitis (2/101), duodenal tumor, gastroenteritis, rectus hematoma, intestinal duplication, annular pancreas, duodenal polyp with intussusception, retroperitoneal mass, ruptured ectopic pregnancy, congenital syphilis, and delirium tremens.

Operative Findings

Surgical operation of patients with intramural duodenal hematoma disclosed free blood in the peritoneal cavity in 33.7% (29/86) of cases.

The portions of duodenum involved with hematoma were similar to those reported by Devroede¹⁷: first portion 15.7% (17/108), second portion 79.6% (86/108), third portion 72.2% (78/108), fourth portion 42.6% (46/108), and proximal jejunum 21.3% (23/108).

Webb and Taylor⁹¹ proposed that intramural hematoma be considered one of a group of traumatic duodenal injuries, consisting of perforation or disruption of the duodenum, contusion of the duodenum with or without formation of intramural hematoma, and retroperitoneal paraduodenal hematoma with duodenal obstruction.^{84, 85} The latter two conditions require similar management, but duodenal perforation or disruption requires more extensive surgical repair and has a high mortality rate. Because of the increase incidence of intramural hematoma from abdominal trauma,²² a search for retroperitoneal duodenal perforations (emphysema, bile staining, or abscess formation) is essential.

Organized or liquefied intramural hematomata (Black pudding⁷⁹) were found subserosally in 65.6% (61/93) of cases and submucosally in 31.2% (29/93) where simple evacuation is possible. About 5.4% (5/93) were intramuscular and could not be adequately evacuated. Associated retroperitoneal hematomata were present in 8.6% of cases.

Associated biliary or pancreatic involvement (Table II) was first reported by Glass²⁹ in 34.5% (41/116) of cases (biliary 16.4% and pancreatic 25.9%). Distention of the gallbladder and common bile duct occurred in 7.8% (9/116) of the surgically explored cases, and gross evidence of acute

traumatic or obstructive pancreatitis was found in 5.2% (6/116). Pancreatic pseudocysts were present in 2.6% (3/116). In addition, one each of the following was reported: alcoholic cirrhosis,⁹⁷ fatty liver,⁴⁰ carcinoma of the pancreas,⁵⁶ peripancreatic hematoma,²² and ectopic pancreatic tissue in the duodenal wall associated with a multicystic pancreas.⁵

The presence of non-viable bowel or associated injury to other viscera was evaluated in reported cases of intramural duodenal hematoma. There were seven in which bowel of questionable viability was found, (7/119—5.9%), in five resection of the involved area was performed, in two diverting gastrojejunostomy. One of the latter was thought to have infarction of the duodenum at a second operation and was treated by gastrojejunostomy without resection.⁸⁸ Duodenal perforation or complete disruption occurred in 4.2% (5/119) of cases. Three duodenal perforations were treated by closure, evacuation, and gastrojejunostomy,²² and one duodenal disruption by evacuation, gastrotomy, and feeding jejunostomy,³ another required a Whipple procedure.⁸⁸ Serosal laceration were found in 5% (6/119) of cases. There was a total of 15.9% (19/119) of duodenal hematomata with associated duodenal injuries. Five (26.3%) of these patients died, two of whom had been operated upon. In another 42.1% (8/19) bowel resections were performed without mortality.

Associated injuries viscera other than duodenum were few. One small liver laceration was reported,⁷³ one ruptured bladder,²² and several mesenteric and retroperitoneal hematomas. As mentioned, direct pancreatic trauma was occasionally encountered.

Active intraabdominal or retroperitoneal hemorrhage was uncommon and was found in only two operations for intramural duodenal hematomas.^{80, 15}

TABLE 3. *Surgical Procedure*
96/120(80%) cases underwent surgery

Initial Surgical Procedure in 96/120 Cases	Instances in Which Subsequent Surgery Was Required	Instances in Which the Procedure Was Done at Subsequent Surgery*
52/96 (54.2%)—evacuation of the hematoma	7/52 (13.5%)	none
16/96 (16.6%)—evacuation and GJ (gastrojejunostomy)		3/18
8/96—evacuation, bowel resection, GJ, duodenal closure (two had biliary bypasses)	4/8 (50%)	3/18
4/96—nothing done	none	none
4/96—GJ only	none	5/18
3/96—gastrostomy and evacuation	none	none
3/96—gastrostomy, evacuation, and feeding jejunostomy	none	none
3/96—closure perforated duodenum	none	none
3/96—appendectomy—normal appendix	2/3 (66.7%)	none
2/96—internal drainage of pancreatic pseudocyst	none	none
2/96—evacuation and reduction of intussusception	none	none
1/96—attempted unsuccessful evacuation of hematoma	none	none
1/96—pyloroplasty and GJ	1/1 (100%)	none

* 18 patients required reoperation. In seven instances, procedures other than those mentioned above were performed.

Management

Management of intramural duodenal hematomata has been evaluated from several aspects. Conservative non-surgical treatment was employed initially in 40.9% (47/115) of cases. Of these, 11.3% (13/115) were successful, four being patients with clotting defects or taking anticoagulants. Twenty per cent (23/115) were treated conservatively and later required operation for obstructive symptoms, and 9.6% (11/115) were treated without operation and subsequently died.

Surgical intervention was employed in 80% (96/120) of cases and 18.8% (18/96) of these required reoperation (Table 3). Simple evacuation of the hematoma was used in 54.2% (52/96) and was successful in 86.5% (45/52). The most frequent indication for reoperation after simple evacuation was continued obstructive symptoms, and the most frequent reoperation was

gastrojejunostomy with or without further evacuation of the hematoma. Hematoma evacuation and gastrojejunostomy were combined initially in 16.6% (16/96) of cases and was successful in 75%, four reoperations were done for malfunction of the gastrojejunostomy. In 4/96 patients no procedure was performed at operation and no further therapy was required. Bailey and Akers³ advocated hematoma evacuation, gastrostomy, and feeding jejunostomy in combination, and this worked well in three of ninety-six patients. As mentioned, in 8.3% (8/96) bowel resections were performed and reoperation was required in four.

Biliary decompression with cholecystojejunostomy or choledochojejunostomy was required on three occasions.^{15, 16, 58} Duodenal perforations were present in three instances and were closed.²²

Drainage of the hematoma cavity with a rubber drain resulted in development of

a duodenal valve with postoperative duodenal obstruction on one occasion, and most authors agree that drains are not necessary.⁵³

It appears from the data that conservative therapy with nasogastric suction and fluid and electrolyte replacement should be instituted initially when the hematoma is induced by clotting defects or when obstructive symptoms and x-ray changes are minimal. Exploratory laparotomy with simple evacuation of the hematoma with or without added gastrojejunostomy is statistically the most effective method of cure and decreasing morbidity.

Mortality

There have been fifteen deaths in 124 instances of intramural duodenal hematoma, an overall mortality rate of 12.1% (15/124). Eight of the first eleven reported patients to 1937 died. Four of these eleven patients were operated upon, the initial successful operation by Meerwein³¹ in 1909, and three of the four patients survived. Since 1937 there have been seven deaths summarized as follows, the first five were managed conservatively without operation.

- 1) 76-year-old chronically ill man with longstanding symptoms of obstruction.⁵
- 2) 40-year-old man with pancreatitis.³⁰
- 3) 36-year-old alcoholic man with delirium tremens and pancreatitis.⁴⁰
- 4) 19-year-old man with obstructive symptoms and severe dehydration.²²
- 5) 35-year-old alcoholic man with epilepsy, jaundice, and pancreatitis.⁸³
- 6) 42-year-old alcoholic man who died of sepsis and peritonitis postoperatively from repair of a rupture duodenum.²²
- 7) 32-year-old alcoholic woman with pancreatitis who died postoperatively of sepsis.²²

Recent deaths from intramural duodenal hematoma have occurred primarily in high risk patients frequently treated without operation.

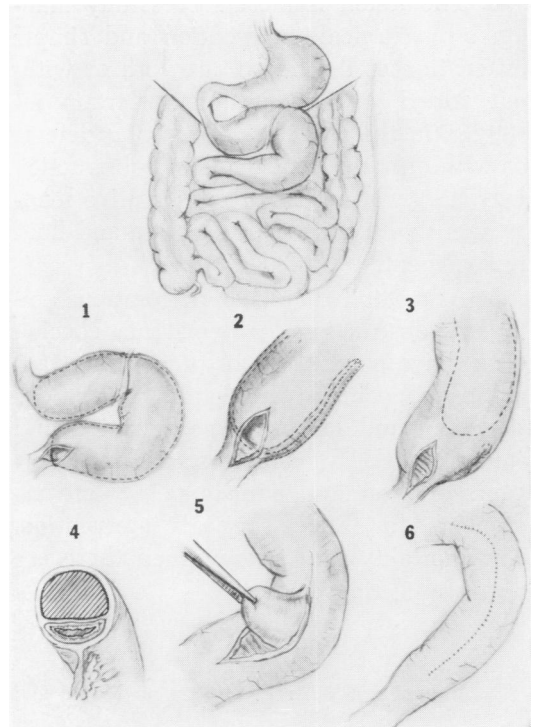


FIG. 1. Duodenal and jejunal intramural hematoma *in situ*. 2. Leading edge of intussuscepting intramural hematoma presenting at jejunotomy site. 3. Intussusceptum reduced. 4. Cross section of bowel demonstrating subserosal hematoma. 5. Hematoma being extracted through a longitudinal serosal incision. 6. Completed serosal closure.

Summary and Conclusions

1. A case of intramural hematoma of the duodenum has been presented and 125 cases reported in the world literature have been reviewed.

2. Intramural duodenal hematoma is a surgical disease of increasing clinical importance. The onset of symptoms is changing from that classically described to a more dramatic course when severe closed abdominal trauma is involved.

3. The diagnosis should be made clinically and radiographically in a major percentage of cases. The classical "coil spring" sign seen on upper gastrointestinal x-rays can probably be attributed to partial intussusception of bowel wall distal to the hematoma.

4. The lesion is most successfully managed by surgical intervention and simple evacuation of the hematoma with or without added gastrojejunostomy. Certain instances in the presence of clotting defects or with only minimal duodenal obstruction may be given a trial of conservative management with nasogastric suction and fluid replacement.

5. Associated duodenal perforation, ampullary obstruction by hematoma causing obstructive pancreatitis or preexisting traumatic or alcoholic pancreatitis, biliary obstruction, and long-term cicatrization and stenosis of the duodenum secondary to organized intramural hematoma^{36, 41} are arguments for early surgical intervention. The mortality rate is low when there are no complicating diseases.

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