

Complicated Gastric Ulcer and Jejunal Hematoma with Concomitant Warfarin Use: Analytical and Biomedical Case Study Report

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Abstract Intramural hematoma is commonly known as a hematoma that occurs in the wall of a structure, such as the bowel or bladder, usually resulting from trauma or, in rare situations, excessive anticoagulation. In particular, intramural gastrointestinal hematoma is part of a spectrum of gastrointestinal injuries. Vomiting and straining, endoscopic procedures, and bleeding disorders are essentially among the most common predisposing factors. However, it can also be an unusual complication of anticoagulation and/or thrombolysis therapy. The most common symptoms are retrosternal chest pain, dysphagia, and hematemesis. Intramural bowel wall hematoma is an often forgotten, rare clinical entity that may pose considerable diagnostic dilemma. Therefore, it is conspicuously imperative to diagnose this condition promptly and correctly in order to avoid unnecessary surgical intervention. In this rare presented case report, a 51-year-old woman having mitral valve prosthesis and mild heart failure under warfarin was admitted to the renowned Sahel General Hospital in Beirut in November of 2006 with massive upper gastrointestinal bleeding that was subsequently controlled by endoscopic sclerotherapy and medical management. About 12 h later, the aforementioned patient suffered a severe abdominal pain accompanied by a steep dropping in the hematocrit level. Moreover, an abdominal computed tomography scan showed thick jejunal hematoma of approximately 70 cm in length that was handled surgically. The patient had been incessantly observed for about 7 days, and subsequently discharged with therapeutic international normalized ratio (INR).

Keywords: CT, Hemorrhagic bowel syndrome, INR, jejunal hematoma, NSAIDs, warfarin

1. Introduction and Background

In the past several years, there have been an increasing number of reports, albeit rare and uncommon, of jejunal hemorrhage syndrome (JHS) [1,2,3]. This condition, also known as hemorrhagic bowel syndrome (HBS), bloody gut syndrome, dead gut, and enterotoxaemia, affects the small intestine, specifically at the level of the jejunum. The first preliminary reports of JHS in the scanned literature come from the early nineties in northeastern US, but since that time there have been confirmed cases reported throughout the US and the world [3,4,5]. The syndrome technically appears without evidential warning signs, and is frequently fatal. Furthermore, periods of concentrated problems are often interspersed by intervals free of any apparent disease, thereby complicating the situation. Although the JHS/HBS syndrome targets the jejunum of affected individuals, the most severely inflicted have more extensive involvement of the small intestine [4,5].

Intramural hematoma, especially jejunal hematoma (JHS), is a very rare disease. JHS especially affects the pediatric population more often than the adult population, whilst it occurs in both genders with a slight, but significant, male predominance in children [4,5,6,7]. It is well known that the duodenum is the most common site for JHS, with the first reported medical case appearing back in 1938, when it was thought to be a "pseudaneurysm between the mucosa and muscular layers." Furthermore, statistically more than 70% of cases are usually associated with trauma [6,7,8]. Interestingly, JHS is usually ruled out in child abuse cases if the problem had occurred prior to 5 years of age (Figure 1 and Figure 2).

2. Case Report Presentation

In our case, a 51-year-old woman, a smoker, had a metallic mitral valve installed since 2001 due to rheumatic heart disease with mild heart failure NYHA II under warfarin 4 mg (¾ tablet daily), furosemide 40 mg,

captopril 25 mg, amiodarone 200mg, and simvastatin 10 mg. Moreover, she had taken diclofenac 100mg daily for back pain for one week, ostensibly without the knowledge of her physician. Following that, she was presented to the emergency room with acute onset of hematemesis (fresh blood seven times), associated with continuous crampy localized epigastric pain starting few minutes before hematemesis, albeit without vomiting, anorexia, melena, rectorrhagia, weight loss, fever, chills or other respiratory or cardiac symptoms. In the ER, she was with tachycardia (P=100beats/min.), tachypnea(RR=24beats/min.), afebrile (37°C) with conserved BP (130/70mmHg). Moreover, she was pale, diaphoretic, and with dyspnea but conscious and well oriented. On auscultation, she had regular heartbeats, mitral click, and diffuse inspiratory and expiratory wheezes; her abdomen was essentially normal except that with mild epigastric pain on palpation, no hepatosplenomegaly, and no hepatojugular reflux [5,6,7,8,9,10]. Moreover, she had few ecchymosis on her four limbs with positive peripheral pulses.

laboratory tests were normal except that for prolonged PTT (120sec), international normalized ratio (INR; >9), high BUN and LDH, normal creatinine and hematocrit of 33 (Table 1).



Figure 1. Barium meal of duodenal hematoma



Figure 2. Ultrasound of duodenal hematoma

In further evaluating her complicated condition, an EGD urgently performed revealed the presence of cardiac ulcer with a clot and severe antral gastritis (Figure 3, Figure 4, and Figure 5); a hemostasis with adrenaline injection was performed and the patient was subsequently transferred to the ICU for close observation. Furthermore,

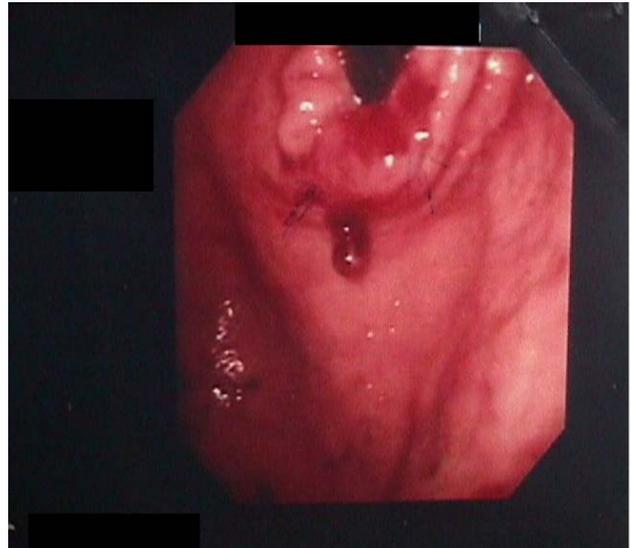


Figure 3. Gastroscopy of the case showing cardiac ulcer with a blood clot



Figure 4. Gastroscopy of the case showing intragastric bleeding

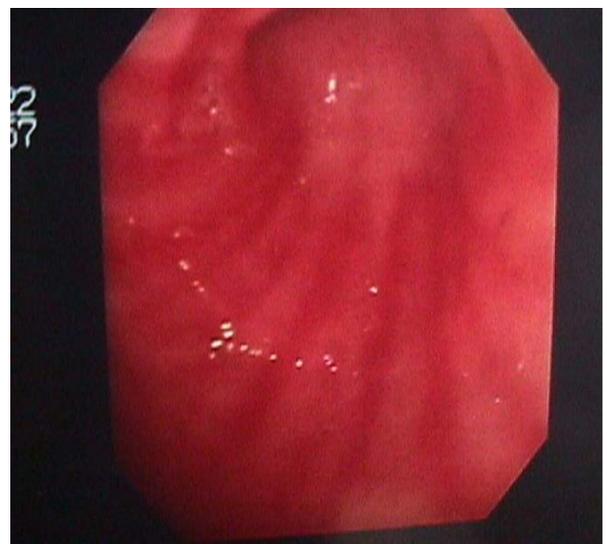
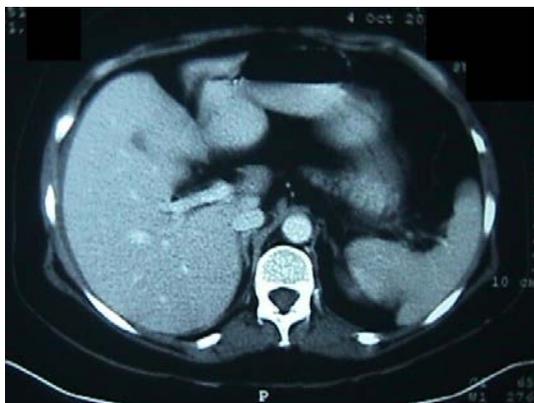


Figure 5. Gastroscopy of the case showing antral gastritis

Table 1. Patient's laboratory results

Laboratory Results on Admission	
Hemoglobin	11
Hematocrit	33
Platelets	200,000
BUN	62
Creatinine	0.9
INR	> 9
PTT	120
MCV	88.6
LDH	295
Na	140
K	4.5
Cl	105
HCO ₃	25
SGPT	50
SGOT	45
GGT	30
Phosp Alk	35
Amylase	38
Lipase	33

About 12 h later, the patient started to complain of diffuse severe abdominal pain with observable decrease in the hematocrit level from 33 to 27, but no more overt bleeding had occurred. Two units of PRBC were transfused and an urgent abdominal computed tomography (CT) scan showed the presence of a thick mural jejunum with free intra-abdominal fluid going with blood and intramural hematoma (Figure 6, Figure 7, and Figure 8). The patient subsequently underwent surgery with a jejunal resection of 70 cm and drainage of intra-abdominal blood. Medical management was undertaken with vitamin K once, four fresh frozen plasma, blood transfusion of another two units of PRBC, IV fluids, and close observation of the urine output and vital signs [8,9,10,11,12,13].

**Figure 6.** CT scan showing a thick jejunum of 15 mm.**Figure 7.** CT scan showing a thick jejunum**Figure 8.** CT scan showing an intra-pelvic fluid collection

Moreover, the patient's INR decreased gradually from 9 → 5 → 3 → 2, then 1.3; at the INR value of 2 heparin was administered with close watching of PTT. On day five post-surgery, she was clinically stable, having bowel movement, and fluid intake was started gradually, and she was transferred to the regular floor for another 3 days, while enteral feeding has been activated and sutures removed. Ambulation was commenced and warfarin resumed with low dose to be discharged under 2mg warfarin daily with an INR of 2.5. Another outpatient follow-up was performed almost two weeks after her discharge. The patient was stable, with no tangible complaints, and her physical exam was essentially normal, her INR was 2.5, and hematocrit 34. A monthly follow-up was performed with no apparent changes [12,13,14,15].

3. Discussion

Intramural jejunal hematoma is characterized by a hemorrhagic episode that starts within the submucosa of the jejunum. The collection of blood forms a hematoma and may eventually progress to dissection of the submucosal layer [16,17,18]. JHS is part of a spectrum of gastrointestinal injuries that range from local mucosal tears to transmural rupture of the jejunum. The several subtypes of JHS are categorized by the nature of the hemorrhage: i) Traumatic; ii) Emetogenic; iii) Abnormal hemostasis-related; iv) Aorta-related; and v) Spontaneous [19-25]. In particular, spontaneous submucosal hematoma of the jejunum is further sub-classified into factors that increase the tendency of bleeding such as drugs or an underlying acute or chronic disease [1,3,10,12,22,26-30].

Although risk factors vary, the most common predisposing factors for JHS are endoscopic procedures, vomiting and straining, and bleeding disorders. JHS is more commonly seen in middle-aged patients, with a slight predominance in females. The clinical stages of JHS manifestation are evaluated according to the degree of luminal involvement [25-30]. Stage I and Stage II are characterized by isolated hematoma and hematoma with surrounding tissue edema, respectively. Stage III involves a hematoma with edema and compression of the lumen,

while Stage IV is the complete obliteration of the jejunal lumen with formation of hematoma, edema, and organized clot formation (Table 2) [31,32,33,34,35].

Table 2. Causes of bowel hematoma.

Causes of bowel hematomas	Examples:
Platelets dysfunction	Glansman's thrombasthenia Bernard-Soulier Sd
Platelets deficiency	ITP / TTP HUS / Leukemia Severe hypersplenism
Drugs	Heparin / warfarin / thrombolytic
Trauma	Abdominal / endoscopic manipulation/ forceful vomiting
Vasculitis	Polyarthritis nodosa / henocho schonlein purpura / Ehlers-Danlos Sd

Speaking of which, warfarin is extensively used for therapeutic and prophylactic purposes [1-5]. The most important complication of the anticoagulation treatment with warfarin is bleeding. It is associated with various hemorrhagic complications, including hematuria, gastrointestinal bleeding, intracerebral hemorrhage, soft tissue hematoma, epistaxis, and retroperitoneal hematoma. Bleeding, which is presented as an intramural hematoma of the small intestine, is a rare complication that is seen in 1 out of approximately 2,500 patients. Excessive anticoagulation with warfarin is the most common cause of spontaneous, intramural small bowel hematoma (JHS). Other risk factors may include hemophilia, idiopathic thrombocytopenic purpura, leukemia, lymphoma, myeloma, chemotherapy, vasculitis, pancreatitis, and pancreatic cancer. The presentation can vary from a mild abdominal pain to intestinal obstruction, and an acute abdomen. If suspected pre-operatively, the diagnosis usually requires CT scan for its confirmation. Most of the patients can be treated non-operatively, ostensibly with a good outcome [12,13,14,15,25-32].

Anticoagulation and/or thrombolysis therapy is an unusual cause of JHS, with only a handful of documented cases, especially in the absence of endoscopic trauma or bleeding disorders. Chest pain, dysphagia/odynophagia, and hematemesis are the most common initial symptoms of JHS [30-35]. There are approximately 35% of patients who present with this triad of symptoms, while at least 50% of patients present with at least two of the above symptoms. As such, it is important to differentiate JHS from several ischemic events because although it is a relatively benign condition, it could be worsened by anticoagulation therapy. In the setting where anticoagulation and/or thrombolysis are necessary, such as a PE, it may be beneficial to perform periodic reassessments for new-onset chest pain and dysphagia/odynophagia so JHS can be recognized and managed earlier [36,37,38].

As we have indicated earlier on, intramural bowel hematomas are rare and the JHS condition in particular is usually a very rare entity. The causes of bowel hematoma are essentially classified as follows: i) Platelets dysfunction and deficiency; ii) Drugs; iii) Trauma; and iv) Vasculitis (see Table 1). The diagnosis is practically based

on clinical (symptoms and signs of abdominal obstruction due to mass effect or ileus post bleeding) and radiological findings [38-42]. Furthermore, enhanced CT is considered one of the best diagnostic methods. In addition, ultrasound and barium meal are also useful (see Figure 1). On the other hand, endoscopic studies are usually performed in inconclusive cases [1,5,8,12,16,25,28,33,37].

Interestingly, the treatment is usually surgical therapy (first line after 1970s) as radial incision of the bowel for evacuation or excision with or without bypass surgery. Conservative therapy was used as the first line therapy before the 1970s, mainly in small isolated cases via putting the patient with rest bowel, nasogastric (NG) suction, and central hyperalimentation, or via CT-guided percutaneous aspiration with somatostatin use in severe pancreato-duodenal damage or periodic closing NG (usually to test and stimulate duodenal transit) [35-45]. Moreover, it is well established that complications are secondary to surgical treatment, including: i) Pancreatitis (traumatic/post-operative); ii) Iatrogenic mucosal leakage; iii) Intestinal ileus; and iv) Malabsorption of resection of long bowel. More importantly, prognosis is usually good and this depends on the associated comorbidities and the site or length of the affected bowel [17,18,19,22-27].

In returning to our case, the patient was under warfarin and had taken non-steroidal anti-inflammatory drugs (NSAIDs) as she came with upper gastrointestinal bleeding. The cause was determined by the endoscope findings as "NSAIDs-induced gastric ulcer and severe gastritis." At that time, the patient was treated by endoscopic sclerotherapy with medical management, in addition to the correction of her prolonged INR. Few hours later, she complained of abdominal severe pain with a drop in her hematocrit level [1,20]. The possible diagnostic observations at that time were including the following: i) Rebleeding from the same ulcerogenic site; ii) Irritation of the gastric mucosa by nasogastric tube; iii) Bleeding from another site; iv) Perforation of the ulcer; and v) Vague abdominal pain due to patient's problem with dilution of hematocrit [28-36].

On physical examination, she was hemodynamically stable, as there was no bleeding from NG tube, and abdomen was soft, with no rebound tenderness with diffuse abdominal pain on palpation. Furthermore, no hematuria or other sites of bleeding were observed. Laboratory tests revealed that the hematocrit decreased from 33 to 27. In addition, an abdomino-pelvic CT scan eliminated the presence of free intra-abdominal air, therefore, the possibility of perforation was eliminated, but it showed a thickness of approximately 15mm in the jejunum with free fluid intra-abdominally going with blood hematoma. The diagnosis of JH as another site of bleeding probably explained the drop in hematocrit and the patient's recurrent abdominal pain [10,11,12,20,21,42-45].

Promptly, the surgeons decided to undergo the surgery to prevent bleeding and avoid intestinal necrosis. A jejunectomy was performed on the following day with resection of 70 cm from the small bowel, in addition to the suction of free blood in the abdomen. The pathologic examination of the resected part confirmed an intramural bleeding with no presence of abnormal cells, any underlying vasculitis, or other malignant processes [1-5].

Finally, the routine management of gastrointestinal bleeding was performed (hydration, blood transfusion,

bowel rest, correction of INR, and urine output). During her observation in the ICU, the patient was stable hemodynamically almost all the time, with no more pain, NG bleeding, and per rectum or hematuria. Moreover, the physical exam was normal with mild wheezes due to her underlying chronic obstructive pulmonary disease (COPD). She passed gas on the third post-surgery, and her hematocrit level was stable. An enteral feeding was started gradually with good tolerance, and she was subsequently transferred to a regular floor following 5 days at ICU. On the floor, she started moving with reintroduction of warfarin in order to put her INR in the therapeutic range. Few days after, her INR was two and she was discharged home one week later with a therapeutic dose of warfarin. Several follow-ups done at two, four weeks, and 3 months following discharge demonstrated normal physical exam, no more complaints, and therapeutic INR [1,20-23].

4. Conclusions and Remarks

Intramural hematoma of the jejunum is a rare complication of anticoagulation therapy. In 1965, Walter Goldfarb published a series of eleven patients with intestinal hemorrhage that was related to oral anticoagulation therapy [1,2,3,4,5]. Warfarin toxicity remains the dominant cause, accounting for the vast majority of patients. The small bowel is affected in up to 85% of the occurrences of haematoma, with the jejunum being the most affected region, in contrast to the post-traumatic findings that affect the duodenum more. The incidence of spontaneous intramural haematoma is reported to be 1 per 2,500 anti-coagulated patients. The mean age at presentation in one recent series of 13 patients was 64 years; 15% of the patients in this series had multiple hematoma [1,2,3].

The presentation can vary from mild, vague abdominal pain to intestinal obstruction and an acute abdomen. The hemorrhage is usually located in the submucosal layer of the bowel and it originates from a small vessel that produces slow bleeding. Hemorrhagic ascites can be present and it is related to the leakage of blood from an engorged, thickened and inflamed bowel wall, with the submucosal bleeding extending into all the layers. In addition to intramural bleeding, intraluminal, intramesenteric and retroperitoneal hemorrhage can also occur, especially when the duodenum is involved. Abdominal CT is the key for its diagnosis, with the characteristics including circumferential wall thickening, intramural hyperdensity, luminal narrowing, intestinal obstruction, and hyperdense ascites [15,16,17,18]. The first step in the treatment of acute intramural small bowel hematoma is the discontinuation of the anticoagulant medication and the correction of the coagulation parameters with fresh-frozen plasma and vitamin K. Operative intervention is only indicated if there is significant intraluminal hemorrhage, bowel perforation or ischemia [18,19,20,21,22].

As noted, small bowel hematoma is a rare clinical entity. It should be considered in any patient on long-term anticoagulation therapy, who present with an acute abdomen. CT, especially non-enhanced CT, is a valuable tool in the diagnosis of this condition. An early diagnosis is crucial, because most of the patients can be treated non-operatively, with a good outcome.

Authors' Contributions

All authors have squarely and equally contributed to developing the experimental, theoretical and statistical aspects of this article.

Declaration of Competing Interests

The authors declare that they have no competing interests.

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