Jejunal Diverticulosis: Review

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Abstract

Jejunal diverticulosis is a rare pathology (less than 5% of the general population). It often has a non-specific symptomatology making its diagnosis usually difficult. It may be complicated by diverticulitis, perforation, acute intestinal obstruction, gastrointestinal bleeding, or intestinal malabsorption. Conservative treatment of symptomatic jejunal diverticulosis may be performed with however poor outcomes. Surgery is then unavoidable. Short and limited intestinal resection should always be preferred to prevent short bowel syndrome. The main poor prognostic factors are diagnosis and surgery delays.

KEYWORDS : Jejunum; Diverticulum; Complications; Diagnosis; Treatment; Mortality.

INTRODUCTION

Diverticulosis of the small intestine was reported for the first time by Sommering and Baillie in 1794 in their autopsy studies [1]. In 1881, W. Osler [2] published the first clinical observation on this issue. Jejunal diverticulosis (JD) is a rare entity (1 to 4.6% of the general population) [3] whose evolution is often asymptomatic. Its diagnosis is difficult and often neglected or delayed because of the location of the disease and its nonspecific clinical presentation.

There is no international consensus for the management of JD today. The objective of this narrative review is to work out the appropriate modalities for the diagnosis and treatment of JD taking into consideration recent medical improvements.

EPIDEMIOLOGY

JD is very rare in children [4]. It is usually reported in patients aged between 50 and 70 years [5]. Men seem to be more affected (58%) than women [6].

JD can be associated with colonic diverticula (70-20%), duodenal diverticula (10 to 40%), and oesophageal or gastric diverticula (2%) [7, 8]. These associations suggest a common etiology.

ANATOMOPATHOLOGY

The jejunal diverticulum corresponds to a herniation of the intestinal mucosa and submucosa with no muscular layer in the diverticular wall. It is a "pseudo-diverticula", whereas Meckel congenital diverticulum is a true one as its wall has a muscular layer [9]. There are two kinds of acquired diverticula: the narrow collar type and the wide collar type [10].

JDs thrive at the weak points of the small intestine wall which are the intestinal micro vessels' entries (vasa recta). Therefore, the JD is strictly localized at the level of the mesenteric side of the small intestine [11]. The jejunal diverticulum of the anti-mesenteric side is a very rare model (only one case was reported) [12]. Its forming mechanism would be different.

The proximal jejunum is the preferred site of small bowel diverticula (75%) [13]. This would be attributed to the larger diameter of the vasa recta and their larger sites of penetration into the bowel wall [9].

The size of jejunal diverticula varies from a few millimeters up to 3 centimeters, but sometimes it exceeds 10 cm [14]. Generally, the jejunal diverticula are large and numerous in the proximal jejunum while they are few and small in the distal jejunum [13].

PHYSIOPATHOLOGY

Although its etiology is still uncertain, it is believed that the jejunal diverticulum develops because of intestinal peristalsis anomalies (intestinal dyskinesia) and high intra-luminal pressure [7]. These conditions can be promoted by an internal hernia or by some surgical procedures such as Roux-en-Y reconstruction or Omega loop [15, 16].

Intestinal dyskinesia is probably related to abnormal smooth muscle or to abnormal neuro-myenteric plexus, which are of 3 types: progressive systemic sclerosis, visceral myopathy, and visceral neuropathy [17]. Some authors suggested a link between bowel diverticulosis and

Neuromuscular diseases that are Cronkhite-Canada syndrome, Fabry disease, neuro-gastrointestinal mitochondrial encephalopathy, Elhers-Danlos syndrome, progressive systemic sclerosis, myasthenia gravis, primary or secondary amyloidosis and jejuna lipomatosis [18–26]. In addition, the relative lack of fat in the mesentery may be another contributory factor as it would further weaken the intestinal wall at vasa recta entries. JD may be also a hereditary family disease (most probably autosomal dominant transmission) especially among families with autoimmune diseases (2 cases were published [27,28]), or be associated with mesenteric malrotation that may cause an intraluminal hyperpressure [29].

CLINICAL MANIFESTATIONS

The clinical suspicion of JD remains difficult, and its diagnosis is often overlooked or delayed as it is usually done when the disease becomes symptomatic or complicated. Indeed only 30% of patients with JD are symptomatic, and less than 10% would have severe complications [30]. Noncomplicated JD symptoms are non-specific such as chronic postprandial abdominal pain, vague abdominal pain, nausea, vomiting, alternating diarrhea constipation, steatorrhea, weight loss, fever, and anaemia [6, 31]. The only definitive way to attribute these symptoms to JD is to get their resolution after surgical resection of the jejunal diverticula.

Clinical presentation of acute or chronic complications of JD is also not specific and variable: it is sometimes discreet but often misleading and may simulate colonic diverticulitis, appendicitis, or acute cholecystitis. Acute complications include massive bleeding, diverticulitis, perforation, and acute intestinal obstruction. Chronic complications are mainly malabsorption but also include intestinal dyskinesia and chronic intestinal hemorrhage [32]. It seems that the risk of complications is higher in patients with multiple jejunal diverticula.

COMPLICATIONS OF JEJUNAL DIVERTICULOSIS

Jejunal diverticulitis

Diverticulitis is much less common in JD than in colonic diverticula. It is probably due to the diverticulum's larger size, better intra-luminal flow, and relatively sterile jejunal content [33]. Perforation may happen (7%) and cause a local abscess or generalized peritonitis [34].

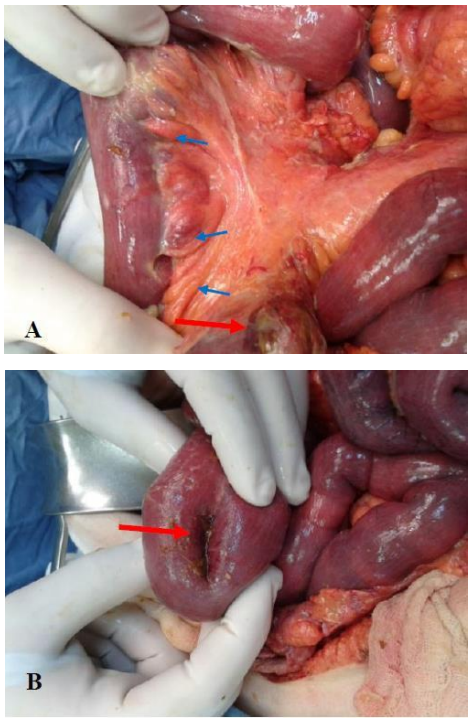

Figure 1: *Diffuse jejunal diverticulosis (A) (blue arrows) with abscessed and perforated diverticulum (red arrows) (A and B): this is a mesenteric abscess related to jejunal diverticulitis.*

The incidence of diverticulitis ranges from 2 to 6% [35]. Its mortality rate was high (24%) in the past then it declined sharply thanks to medical improvements [36]. Jejunal diverticulitis is rarely diagnosed preoperatively because of its nonspecific clinical presentation as acute abdominal pain with infectious syndrome and sometimes gastrointestinal bleeding. In addition, it is sometimes very difficult to distinguish, on a CT scan, between a perforated diverticulum and other differential diagnoses that have a similar image as an inflammatory process next to an intestinal loop. These differential diagnoses are

mainly intestinal neoplasms then Crohn's disease [37] and more rarely intestinal perforation by a foreign body, traumatic hematoma of the intestinal wall, and drug-induced intestinal ulceration [38, 39]. However, it is worth noting that the inflammation of Crohn's disease affects the whole intestinal wall circumference, unlike jejunal diverticulitis which causes asymmetric intestinal wall inflammation [40].

Perforation of jejunal diverticulum

The perforation of JD is rare (2.1 to 7% of diverticulitis) [41] probably because of intestinal intraluminal low pressures. Diverticulum perforation leads to generalized [42] or localized peritonitis. The latter usually ties in with a mesenteric abscess which is the most common presentation of this perforation (figure 1) [43]. As a rule, this wouldn't cause immediate peritoneal contamination. Therefore, diagnosis of such complications is delayed and would lead to a possible disastrous clinical evolution, particularly in debilitated and/or elderly patients. The other complications of diverticulum are digestive fistula, intra-peritoneal abscesses, and liver abscesses [36, 44].

From a pathophysiological standpoint, possible causes of diverticulum perforation are necrotizing inflammatory reaction (82%), progressive ulceration in diverticulitis, blunt trauma (12%), foreign body impaction (6%), and cocaine intoxication [31, 45].

Apart from any peritonitis, JD may cause chronic pneumoperitoneum by air diffusion through the semipermeable membrane of the diverticulum thin wall. This also may be due to diverticular micro perforation [42]. Jejunal diverticulum perforation may lead to high mortality rates ranging from 21 to 42% [46].

Acute intestinal obstruction

Intestinal obstruction remains the rarest complication of JD (2.3 - 4.6%) [14]. It may be due to intestinal dyskinesia (10-25%) resulting in what is called a "functional acute bowel obstruction" or a "pseudo-obstruction" [30]. Otherwise, it is usually due to a mechanical obstruction cause that may be as follows:

- A large diverticulum or an inflammatory pseudotumor relevant to jejunal diverticulitis leading to an extrinsic intestinal compression [47].

- Intestinal intussusception caused by an enterolith formed in the jejunal diverticulum [47].

- Adhesion band formed mainly due to repeated outbreaks of jejunal diverticulitis usually pauci-symptomatic. This band would cause a small bowel volvulus [14, 48, 49].

- An intestinal fibrous stenosis is generally due to multiple jejunal diverticulitis flare-ups [50].

- Stenotic jejunal tumor developing inside the diverticulum [51].

Enterolith formation within the jejunal diverticulum is rare: real enteroliths derive from Choleric acid (product of conjugation of

bile acids) and fake enteroliths form by impaction of food debris in the diverticulum. Small enteroliths are sometimes dislodged and transit through the colon uneventfully while large stones can cause obstruction: it is an "enterolithical ileus" like a gallstone ileus [52].

Diverticular bleeding

Hemorrhage complicates 2 to 8.1% of JD [53]. JD causes less than 5% of rectal bleeding [54]. The pathophysiology of diverticular bleeding is the same regardless of the location of the diverticulum on the digestive tract. Hemorrhage is usually due to arterial erosion by a diverticular ulcer, diverticulitis, an enterolith, or drug intake (NSAIDs). It can be occult bleeding with iron-deficiency anemia.

Vascular lesions such as arteriovenous malformations and venous ectasia, which are the most common causes of bleeding in the small intestine, can rarely coexist in the jejunal diverticulum (only 2 cases were reported in the literature) [55, 56]. Furthermore, there are other causes of bleeding from the jejunal diverticulum such as ischemic colitis, ectopic pancreas, and small bowel tumors (adenoma, adenocarcinoma, endocrine tumour) which may also exist in non- diverticular intestine.

Malabsorption

It is the most common complication of JD. It is due to intestinal stasis and bacterial overgrowth [57]. Malabsorption would result in Vitamin B12 deficiency (megaloblastic anemia) and/or in steatorrhea due to deconjugation of bile acids and disturbance of fat absorption. This triad (JD, megaloblastic anemia, and steatorrhea) was described for the first time in 1933 [58].

Malignancy in jejunal diverticulosis

Malignancies such as leiomyosarcoma, adenocarcinoma, endocrine tumors, and stromal tumors may rarely arise in the Jejunal diverticulum [51, 59]. The risk is higher in patients with multiple jejunal diverticula.

EXPLORATIONS

Abdominal x-ray

It can show distension of jejunal loops and air-fluid levels within large diverticula. JD may take on the appearance of an air-filled structure on the mesenteric border of the proximal small.

Intestine [60]. An opacity (calcification) is suggestive of a possible enterolith [52], but unfortunately, it also suggests biliary and urinary stones.

Small bowel follow-through (SBFT)

Before the advent of abdominal computed tomography (ACT), the SBFT was the gold standard for the diagnosis of JD [57]. However, it is prohibited if complicated JD is suspected. The diverticulum appears in SBFT as a gas clarity (bubble) or as a pediculate barite collection on the mesenteric border of the jejunum and in continuity with the intestinal lumen [57].

Echo-doppler (ED)

The ED would be very helpful in diverticular bleeding because of its good sensitivity (73.7%),

high specificity (97.1%), high positive predictive value (87.5%), and high negative predictive value (93.2%) [61].

Multi-slice computed tomography (MSCT)

Nowadays, the MSCT is the reference imaging tool for the diagnosis of JD as well as its possible complications [38] (figure 2). It may show jejunal diverticulum as:

- a sac on the mesenteric side of the small bowel, or
- a focal and asymmetric thickening of the intestinal wall or
- an inflammatory pseudo-tumor or
- an abscess next to a jejunal loop with infiltration of the surrounding mesenteric fat [62].

The MSCT also allows ruling out other abdominal inflammatory conditions such as colonic diverticulitis, Crohn's disease, and appendicitis.

A recent meta-analysis showed that abdominal angiostatin is effective and very precise in diagnosing or excluding active gastrointestinal bleeding with high sensitivity (79-100%) and specificity (85-100%) [63, 64].

Mesenteric angiography (MA)

The MA has a diagnostic profitability of 61-72% in patients with active bleeding [53]. However, it can only detect bleeding with an outflow of at least 0.5 ml/min. Angiographic pathognomonic sign of diverticular bleeding is the extravasation of contrast medium into the diverticulum. MA offers also the possibility of super-selective embolization (success rate of 58-100%) however with a risk of intestinal ischemia (0-7%) and rebleeding (15%) [64].

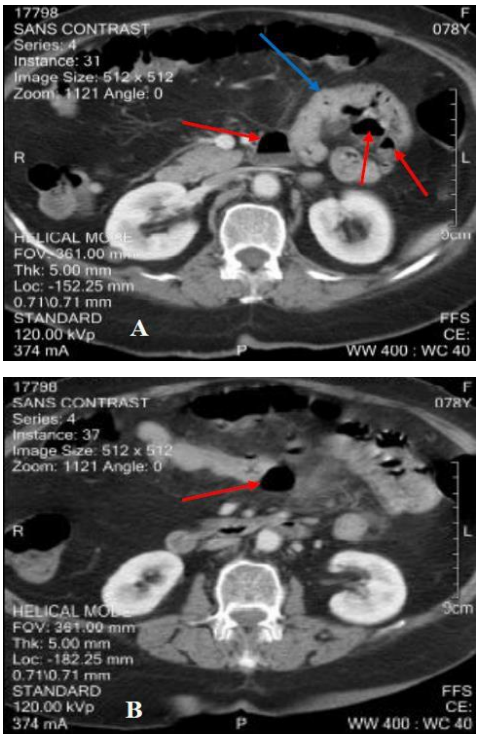

Figure 2**:** *Abdominal CT-scan showing multiple jejunal diverticula (A and B) (red arrows) with thickening of the first jejunal loops wall (A) (blue arrow) suggesting a jejunal diverticulitis.*

Red blood cell scintigraphy (RBS)

RBS can be used to detect and localize gastrointestinal bleeding even if they are of very low outflow (0.1ml/minute) with high sensitivity (93%) and specificity (95%) [64, 65]. However, it is still considered an accessory tool usually needed when the source of intestinal bleeding is still unknown after endoscopy and abdominal angiostatin [64, 66].

The combination of RBS (locates the bleeding) and MA (embolization) is highly efficient in diagnosing and treating hemorrhagic JD [33].

Endoscopic explorations

The enteroscopy is efficient in finding out the cause of gastrointestinal bleeding inherent to the small intestine, but it is useless in emergencies. The double-balloon enteroscopy (DBE) is significantly more effective than conventional enteroscopy in the diagnosis of small bowel lesions ($p < 0.0001$) [67]. It is also better than the video capsule endoscopy (VCE) which remains an excellent exploration method of obscure gastrointestinal bleeding particularly those of small bowel origin [68-70].

TREATMENT

Emergent surgery is required in 8-30% of patients with complicated JD [17]. The total laparoscopic approach is possible and it is safe and efficient even for complicated JD [43].

It is worth noting that diverticula may spring afresh after surgical resection of all jejunal diverticula [71].

Asymptomatic JD

Asymptomatic diverticula usually do not need any treatment. Nevertheless, surgery should be considered for large diverticula with dilated intestinal loops because of the high risk of complications [8].

Symptomatic uncomplicated JD

Medical treatment is usually sufficient otherwise; resection is indicated [5].

Jejunal diverticulitis

Management of uncomplicated diverticulitis with hemodynamic stability requires fasting and systemic antibiotherapy (oral antibiotics are debatable). Surgery has to be considered if medical treatment fails [38]. Considering the high-risk recurrence and other JD complications in this case, surgical resection is highly recommended [3, 7].

Perforated JD

A conservative treatment based on intravenous antibiotherapy should be considered first for mesenteric or peri-diverticular abscesses. Patients may also undergo whenever needed CT-guided percutaneous drainage. On the other hand, the surgical approach (taking away the perforated diverticulum) becomes mandatory in the following situations:

- A generalized peritonitis
- Failure of
- Infeasible percutaneous drainage

Immediate digestive anastomosis should be avoided in shocked or high-risk patients and jejunostomy must then be rationally performed [7, 57].

Two surgical techniques are currently prohibited as they are Unsafe and significantly increase (3 times) the risk of death. These techniques are simple diverticulectomy, the suture of the diverticulum perforation, and burying the perforated diverticulum [72]. Paradoxically, it seems.

reasonable to use one of these techniques if the perforated diverticulum is close to the duodéno-jejunal angle because an intestinal anastomosis at

this level would be difficult to manage.

Extensive intestinal resection is prohibited because of a short bowel syndrome risk. Thus, in case of extensive JD, intestinal resection limited to the perforated diverticulum is recommended.

Haemorrhagic JD

Double balloon enteroscopy and embolization are very seducing approaches for management of jejunal diverticula bleeding [73]. Endoscopic haemostasis is burdened with a high mortality rate because of high risk of bleeding recurrence. Contrariwise embolization has a success rate of 85% and seems to be the best alternative in debilitated and high anaesthetic risk patients [64, 74]. However, surgical resection of the involved intestinal loop with immediate anastomosis remains the reference treatment of haemorrhagic JD. Unfortunately, preoperative diagnosis of the precise source of bleeding is rarely done [3, 53]. Thus, for patients with diffuse JD, intraoperative endoscopy via enterotomy would be helpful to locate the source of bleeding with a success rate of 76% [75]. In case of obvious colonic diverticula bleeding, jejunal diverticula should be resected whenever possible. Preoperative abdominal angioscan may be useful in patients with colonic and jejunal diverticula so as to avoid unavailing colectomy [53]. Immediate and long-term cessation of bleeding after resection is the certainty criterion that the source of bleeding is the jejunal diverticulum.

JD with acute intestinal obstruction

JD with intestinal obstruction may be managed with gentle digestive aspiration but due to high failure rate surgical approach is oftenly performed [47, 76]. In case of enterolithical ileus, the surgeon must attempt to manually crush the enterolith and to push its fragments in the distal colon [47]. Otherwise, an enterotomy may be carried out close to upstream or downstream of the obstruction site. Laparoscopic handling of enterolith (crushing, mobilization and extraction) is not recommended because it is potentially traumatic for congestive intestine. Moreover, laparoscopic complete verification of all jejunal diverticula is illusive [45, 52].

In case of intestinal obstruction without enterolith, the rule is intestinal resection with immediate anastomosis. Any adhesive band should also be sectioned. Complications as perforation, necrosis or stenosis necessarily require intestinal resection [76].

JD and malabsorption

Megaloblastic anaemia is classically treated by parenteral administration of Vitamin B12. Broad-spectrum antibiotics covering enteric flora (Rifaximin, Metronidazole, Amoxicillin / Clavulanic acid, ciprofloxacin and tetracycline) reduce efficiently diarrhoea and abdominal distension [77]. These antibiotics are typically prescribed for two weeks. Their rotating use is necessary to prevent microbial resistance.

MORTALITY - PROGNOSIS

JD is a benign pathology with a good prognosis in general. The mortality rate of complicated JD ranged from 21 and 30% but thanks to medical improvements, recent publications reported much lower mortality rate (0-5%) [78, 79].

Elderly patients, comorbidities, severe complications, diagnosis delay and mainly late surgery for diverticular perforation are associated with poor outcomes [33].

RESEARCH PRIORITIES CONCERNING JEJUNAL DIVERTICULOSIS

Medical researchers should try answer to these questions:

1) Is there any genetic test to detect JD in high-risk patient with neuro-myenteric plexus pathology?

2) What is the optimal combination of medical imaging that is most effective for diagnosing symptomatic and complicated JD ?

3) Is there any indication for small bowel transplant in diffuse JD ?

CONCLUSION

Clinical data of JD are always nonspecific and thus unsubstantial for its diagnosis. Therefore, MSCT and less frequently DBE and VCE are clearly contributing. Conservative treatment of symptomatic JD may be performed with however poor outcomes. Surgery is then unavoidable. Short and limited intestinal resection should always be preferred to prevent short bowel syndrome. The main poor prognostic factors are diagnosis and surgery delays.

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