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Case Report

Jejuno-colonic anastomosis to restore intestinal continuity in a case of intestinal failure due to type I short bowel syndrome

Luis Ernesto Suárez Luna*, Raúl Andrés Troya Malo, Guadalupe Jhoana Vázquez Ramirez, José González Quiroga, Karina Sánchez Reyes

Department of Gastrointestinal Surgery, UMAE Hospital de Especialidades CMN Siglo XXI, Mexican Institute of Social Security, Mexico

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***Correspondence:**
Dr. Luis Ernesto Suárez Luna,
E-mail: drlesuarez@gmail.com

ABSTRACT

Short bowel syndrome is a rare surgical complication associated with high morbidity and mortality, significant decrease in quality of life and increasing costs to the healthcare system. In some cases, this condition can be mitigated and even reversed if the intestinal transit is restored with a minimum of 75-100 centimeters of small intestine and a segment of the colon. Some of the surgical techniques available within autologous gastrointestinal reconstruction are Serial transverse enteroplasty (STEP), Longitudinal intestinal lengthening (LILT), and as a last resort, intestinal transplantation. We presented a case of intestinal failure due to type I short bowel syndrome who improved in clinical stage from D4 to D2 after intestinal continuity restitution was performed with a jejuno-colonic anastomosis.

Keywords: Short bowel syndrome, Intestinal failure, Jejuno-colonic anastomosis, Total parenteral nutrition, fistules, Intestinal continuity restoration

INTRODUCTION

Short bowel syndrome, defined as a total intestinal length below 200 cm, is the main cause of intestinal failure.[1] Intestinal failure is defined as a reduction in gut function under a minimum threshold necessary to allow appropriate absorption of nutrients, water and electrolytes.[2] This condition makes it necessary to supplement these nutrients by enteral or parenteral route to maintain nutritional status and growth.[2]

CASE REPORT

We present the case of a 61-year-old male, with family history of prostate cancer and hypertension, and a personal history of social alcohol drinking for 27 years. His past medical history includes blood transfusions without any adverse reactions. The patient denies any chronic or degenerative diseases. He has had multiple surgeries; 1990 open appendectomy, no records. 1990 exploratory laparotomy due to blunt abdominal trauma, with findings of grade III hepatic injury, post-traumatic pancreatitis and an unspecified duodenal injury treated with primary closure.

In 2014, intestinal obstruction treated with laparotomy and adhesiolysis, no intestinal resection was performed. In 2020, intestinal obstruction initially treated with laparotomy and adhesiolysis. Due to non-favorable course, he is re-intervened with a new laparotomy, finding a frozen abdomen that only allows a jejunostomy to be performed. The patient had unfavorable course of illness that manifested as acute abdomen, which lead to a new laparotomy. This time the surgeons found total ascending colon necrosis and perforation that was resolved with a right hemicolectomy with end-ileostomy. Seventy-two

hours later, due to septic shock, the patient is re-intervened with the following findings: grade IV of the Björk open abdomen classification, bowel perforation 30 cm proximal to jejunostomy and multiple intestinal fistulas. Surgeons performed primary closure of the perforation site, suture invagination of fistulas and abdominal lavage. He progressed to intestinal failure due to short bowel syndrome, classified as D4 in clinical classification. A year later, during follow-up and after showing no improvement in home parenteral nutrition requirements despite maximum medical therapy, surgical management is proposed attempting restoration of intestinal continuity.

We performed a new laparotomy, with findings of severe bowel adhesions grade 4 in Zülke classification, a colo-cutaneos fistula within 40 cm of proximal transverse colon, a bowel segment of 110 cm with 23 fistulas (both entero-enteric and entero-cutaneous) starting 90 cm distal to Treitz' ligament. We found intact descending and sigmoid colon as well as rectum. With these findings we performed a side to side anastomosis between the jejunum and transverse colon using a mechanical 60 mm GYA stapler, blue cartridge. This procedure was done with no incidents, and he presented a satisfactory postoperative course with no clinical or radiological signs of anastomotic leak, with adequate oral tolerance and no diarrhea. Histopathological study reports acute and chronic enteritis and peritonitis.

Figure 1: Findings of 23 fistules during surgery.

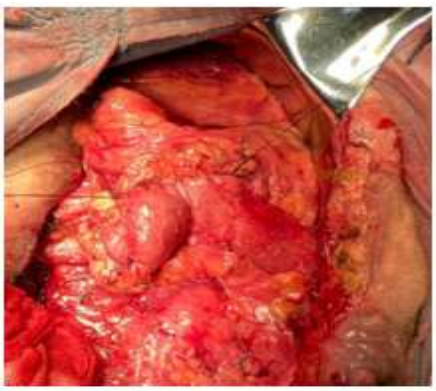

Figure 2: Anastomosis between jejunum and transverse colon.

Figure 3: Intestinal transit after surgery.

DISCUSSION

Normal intestinal length varies according to radiological, surgical or autopsy measurements, varying from 275 to 850 cm. Intestinal length less than 200 cm is an accepted definition for short bowel syndrome.[1] This syndrome is characterized by malabsorption secondary to loss of intestinal mass, frequently after surgical intestinal resections.[3] Clinical manifestations are diarrhea, steatorrhea, malnutrition, and dehydration. It is associated with high morbidity and mortality, as well as a decrease in quality of life and increased health-care costs.[4] After eating, absorption of most nutrients occurs within the first 100 cm of the jejunum. Bile acids, vitamin B12 and magnesium are absorbed in the final 100 cm of the ileum. Water and sodium are absorbed throughout the digestive tract, being tightly dependent on intracellular junction's permeability, which is greater in the jejunum compared to the ileum.

In the jejunum, water absorption depends on the concentration of sodium and glucose, which are absorbed by a difference in concentration gradient. While in the ileum, water is absorbed by an electromechanical gradient that allows greater reabsorption of fluids.[5] Between oral intake, in addition to gastric, pancreatic and biliary secretions, approximately 9 l of secretion pass through the small intestine, 7 l are absorbed by the small intestine and 2 of them pass through the ileocecal valve. A healthy large intestine absorbs approximately 150 kcal per day, but can absorb up to 1000 kcal per day in those patients with malabsorption. Approximately 75% of cases of short bowel syndrome develop after a single massive bowel resection; while the remaining 25% occurs after multiple resections.[5]

This syndrome is one of five conditions known to cause intestinal failure.[5] Intestinal failure was defined for the first time by Fleming and Remington in 1981 as a reduction in intestinal mass below the minimum necessary for proper digestion and absorption of food.[6] Recently, ESPEN

proposed a new definition: 'reduction in gut function below the minimum necessary for the absorption of macronutrients and/or water and electrolytes, such that intravenous supplementation is required to maintain health and/or growth'.[2] If this gut reduction is compensated solely with oral supplements, we use the term 'intestinal insufficiency'.[5] Intestinal failure can be further classified by functional aspects, physiopathological aspects and clinical aspects: functional classification is in accordance to Shaffer's initial proposal- (a) type I- acute short term condition, usually in the perioperative setting, associated or not to other pathologies that require intravenous supplementation for days or weeks; (b) type II- prolonged acute condition in metabolically unstable patients, such as open abdomen, enterocutaneous fistula or acute mesenteric ischemia that requires inter-disciplinary management and intravenous supplementation for weeks or months; and (c) type III: chronic condition in a metabolically stable patient, that requires intravenous supplementation for months or years.[5,7] Physiopathological classification recognizes 5 main conditions that lead to intestinal failure: (a) short bowel syndrome; (b) intestinal fistula; (c) intestinal dysmotility; (d) mechanical obstruction; and (e) extensive small bowel mucosal disease.[5]

After an extensive bowel resection is done, morphological and functional intestinal adaptation mechanism are carried out, thee are characterized by an increase in crypt's depth and elongation of the intestinal villi to provide greater absorptive surface. Also, hypertrophy of the mucosa and hyperplasia of the enterocytes can occur within the first 48 hours and continues for months to years, depending on certain factors, such as the length of the resected segment and the presence or absence of ileocecal valve and colon.

Global intestinal adaptation consists of three phases- (a) acute phase: occurs during initial 3-4 weeks and is characterized by metabolic disorders and significant intestinal losses. It requires close monitoring in hospital settings to avoid sequelae such as dehydration, acute kidney injury, acid-base abnormalities and electrolyte impairments; (b) adaptative phase: It takes from 2 months to 2 years and involves adaptation in the remaining small intestine to compensate and maximize absorption (enterocyte hyperplasia). Feeding should be mixed both enteral and parenteral; and (c) mantainance phase: it happens 2 years after surgery onwards, intestinal adaptation has already been achieved and the patient is fed enterally. Long-term complications are associated to mechanisms related to both gastrointestinal disease and total parenteral nutrition.

Kidney disease and stones

Events of severe dehydration due to high intestinal losses can lead to acute kidney injury, mainly in patients with end jejunostomy.[5] There is also an association between decrease in glomerular filtration rate in patients with total parenteral nutrition associated with bacteremia and fungemia. Calcium oxalate stones occur in patients with an intact colon due to an increase in absorption that occurs in this digestive tract portion.

Dehydration and electrolyte impairment

These are also risk factors to develop acute kidney injury, kidney stones and acid-base impairment. The most common electrolyte disorder is hyponatremia followed by hypokalemia.

Lactic acidosis

It is due to fermentation of carbohydrates in the colonic remnant, with a consequent elevation in D-lactate production. Even though the mechanism is unclear, elevation of D-lactate produces neurological symptoms. Treatment is aimed at avoiding carbohydrate intake and antibiotics against D-lactate producing bacteria such as metronidazole, vancomycin, and clindamycin.[5]

Metabolic bone disease

Secondary to TPN use, patients may develop osteopenia, osteoporosis or osteomalacia. Serum concentrations of vitamin D, PTH and biochemical biomarkers of bone turnover should be monitored to avoid these complications.[8]

In some cases, intestinal failure in the adaptive phase can be reversible if it meets any of the following characteristics: (a) more than 35 cm of small intestine along with a jejuno-ileal anastomosis and an intact ileocecal valve and colon; (b) more than 60 cm of small intestine with jejuno-colonic anastomosis; (c) more than 115 cm of small intestine with an end jejunostomy.

Whenever possible, intestinal continuity should be restored since a residual small intestine of 75 cm or more, anastomosed even to a colonic remnant, carries a reported probability of up to 69% of weaning from parenteral nutrition at one year of follow-up.[9] Analogous gastrointestinal reconstruction is proposed in cases of persistent intestinal failure and should only be performed in specialized centers by surgeons with expertise in these procedures.[10] In case of advanced liver disease, intestinal transplantation should be carried out directly.[11] These procedures are based on intestinal residual length, function, disease origin, stomas, blind loops, or colonic remnant.[12] In case of decreased motility and intestinal dilation, procedures such as STEP, LILT or Bianchi technique are considered.[13]

In case of rapid intestinal transit without dilation, a small bowel segment reversal is suggested to slow nutrient's flow but this procedure is contraindicated in case of bacterial overgrowth.[14] Intestinal transplantation is indicated in patients who will require lifelong parenteral nutrition with associated irreversible complications, such

as impending liver failure, main hepatic vein thrombosis or recurrent catheter associated sepsis.

Intestinal transplantation is a high-end procedure that requires specialized centers and multidisciplinary teams.[15]

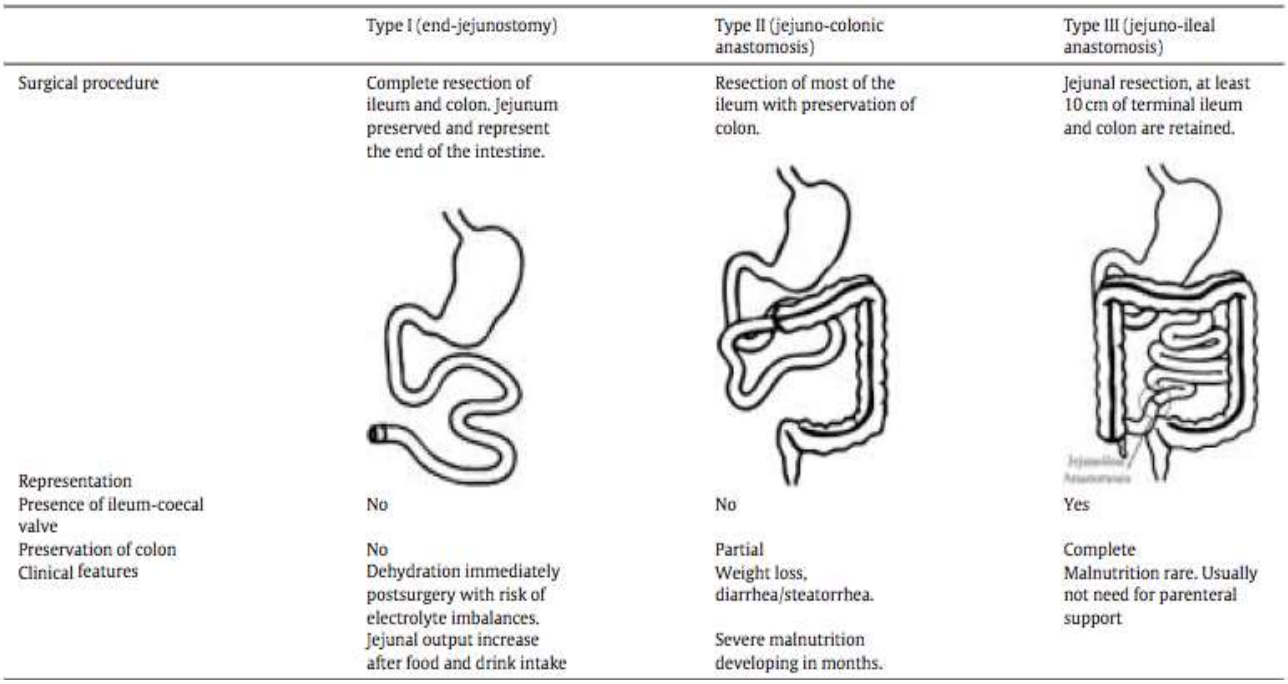

Figure 4: Anatomical classification of short-bowel syndrome, adapted from Massironi et al.[3]

Table 1: Clinical classification of intestinal failure.

IV energy supplementation (kcal/kg)	Volume supplementation			
	<1,000 (1)		<1,000 (1)	
0 (A)	A1	0 (A)	A1	0 (A)
1-10 (B)	B1	1-10 (B)	B1	1-10 (B)
11-20 (C)	C1	11-20 (C)	C1	11-20 (C)
>20 (D)	D1	D2	D3	D4

CONCLUSION

Intestinal failure is an uncommon condition that can lead to severe impairments and an important reduction in quality of life and high health system costs. Patients with intestinal failure may have severe and chronic complications associated with increased morbidity and mortality. Aside from achieving and adequate nutritional intake, patients with intestinal failure can be subject to surgical interventions that can improve their clinical stage and help them wean off parenteral nutrition thus, reducing their comorbidities and health systems costs. The patient presented in this case had a clinical stage improvement from D4 to D2 after the intestinal continuity restitution was done. Jejuno-colonic anastomosis can be a surgical option for patients with intestinal failure as illustrated in this case.

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