Intestinal adaptation in short bowel syndrome: A case report

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Abstract

Short bowel syndrome is a clinical entity that includes loss of energy, fluid, electrolytes or micronutrient balance because of inadequate functional intestinal length. This case report demonstrates the case of a woman who compensated for short bowel syndrome through intestinal adaptation, which is a complex process worthy of further investigation for the avoidance of dependence on total parenteral nutrition and of intestinal transplantation in such patients.

Introduction

In this article, a case of a woman with an odd clinical presentation is described. The patient was finally diagnosed to have intestinal ischaemia and was treated surgically for that reason. As a result, she had to confront the consequences of short bowel syndrome, which were however compensated by the innate mechanism of intestinal adaptation. This patient was hospitalised in a Greek province and refused any referral to a specialised centre.

Case report

A 78-year-old woman with a history of hypertension and coronary artery disease under treatment presented with intense pain of acute onset in the back and lower abdomen. She also complained of malaise and oliguria. Her vital signs were: blood pressure (BP) = 150/100 mmHg, heart rate (HR) = 80 bpm, and temperature (T) = 40 °C. Clinical examination of the abdomen revealed normal bowel sounds, a slight tenderness in the inguinal region, and tympanic sounds. Acute renal failure was diagnosed based on laboratory assessment. Ultrasound examination revealed renal pelvic dilatation especially in the left kidney without presence of kidney stones. Considering the above, the patient was hospitalised in the nephrology department. Within the next 2 days, an attempt to insert pigtail ureteral catheters was made, but it unfortunately failed. Under these circumstances and in combination with the patient’s clinical status deterioration, the patient underwent bilateral nephrostomies. Despite this handling, the patient showed only a slight amelioration of her symptoms and laboratory assessment. The continuing pain led the therapeutic team to order a computed tomography (CT) scan, which revealed an image of ileus and intramural gas in the small intestine. The above finding imposed the diagnosis of intestinal ischaemia. During the following urgent operation, the patient underwent enterectomy (40 cm of small intestine left) and jejunostomy. An attempt to regulate fluid and electrolytes (especially Na+ and K+) homeostasis was made in the postoperative period. The patient received total parenteral nutrition (TPN) for almost 45 days postoperatively with subsequent nutrition per os. On the 30th and 58th postoperative days, an endoscopic examination with a gastroscope through the jejunostomy was performed. The endoscopic image (Figs. 1 and 2) indicated intestinal adaptation with an increase of the inner intestinal diameter and growth of the villi. After the patient was discharged, she was advised to visit the hospital twice a week for the first 2 months and once a week for the following months. According to laboratory results, she was hospitalised to regulate fluid and electrolyte balance. The patient died 18 months postoperatively due to acute heart failure.

Discussion

The above clinical case is presented in order to reveal the capability of the intestine to adapt both structurally and functionally.
with loss of a significant length, because only 40 cm of small intestine could be saved. Although the length of the remaining bowel necessary to prevent dependence on TPN is approximately 100 cm in the absence of an intact and functional colon, or 60 cm in the presence of a completely functional colon, the degree of adaptation and TPN dependence may be highly individualised. Patients with a jejunostomy are at increased risk of TPN dependence, and those with a jejunal–ileal anastomosis are less likely to be TPN dependent. In this case, not only is the remaining bowel much shorter than needed for intestinal adaptation, but also the patient underwent intestinal adaptation in the presence of a jejunostomy.

A group of experts on the subject convened to develop the following definition: “Short bowel syndrome results from surgical resection, congenital defect, or disease-associated loss of absorption and is characterised by the inability to maintain protein-energy, fluid, electrolyte, or micronutrient balances when on a conventionally accepted, normal diet” [1]. Short bowel syndrome is usually defined anatomically as <30% of normal intestinal length (75 cm in children and 200 cm in adults), although the absorptive function does not always correlate with residual bowel length. A variety of physiologic, cellular, and molecular responses serve to enhance the absorptive function (Table 1). The remaining intestine compensates for the surgically diminished length through an increase in villus height, crypt depth [2–6], bowel calibre, and length. An increase in the proliferation of crypt cells, coupled with a decrease in apoptosis and an augmentation of the cellular progression along the crypt–villus axis, is responsible for the increase in villus height and a total increase in DNA, RNA, and protein content. Proliferation therefore requires a supply of polyamines, putrescine, spermidine, and spermine, which are organic cations, influencing DNA, RNA, and tissue synthesis [7]. At the same time, an increase in the expression of transport proteins (Na+/glucose cotransporters, Na+/H+ exchangers) in
Factors influencing intestinal adaptation. (Table 2) in an attempt to promote intestinal adaptation and help patients wean off parenteral nutrition. Strategies used in humans include either the treatment with glutamine and growth hormone [9,10] or the administration of glucagone-like peptide [11,12]. The effectiveness of these treatment options remains to be proved by multicentre studies, otherwise evidence remains weak. Furthermore, magnifying endoscopy for monitoring intestinal adaptation is practicable and reliable.

To sum up, intestinal adaptation is a complex process that compensates for the loss of digestive and absorptive capacity in the remnant intestine. In case this process is inadequate, a lifelong dependence on parenteral nutrition will ensue or the need for intestinal transplantation will arise. Both alternatives may have serious negative effects on humans. Nevertheless, given the low patient survival and quality of life associated with other treatments for irreversible intestinal failure, it is imperative that research continues into the optimisation of the adaptation. A thorough understanding of the mechanisms that drive intestinal adaptation will be essential in the development of novel and innovative therapies that result in saving lives.

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References


