

An unusual outcome of a giant ventral hernia

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ABSTRACT

يعتبر الفتق من المشاكل الجراحية الروتينية التي يمكن أن تحدث في أي فئة عمرية، بغض النظر عن الوضع الاجتماعي و الإقتصادي للمريض. نقدم هنا حالة نادرة لمضاعفات فتق في البطن أدت إلى متلازمة الأمعاء القصيرة. هذا أمر غير مألوف و نادر للغاية حسب الحالات المنشورة في المجلات العلمية. يصف هذا التقرير حالة رجل يبلغ من العمر 54 عاماً أتى إلى المستشفى بفتق ضخم و محشور في جدار البطن أدى إلى غرغرينا في معظم الأمعاء و إلى حدوث الأمعاء القصيرة.

Hernias are routine general surgical problems that may present in any age group, regardless of the patient's socioeconomic status. We present a rare case of a complicated ventral hernia leading to short bowel. This is an unusual case and is very rarely reported in the literature. This current case report describes a 54-year-old gentleman who presented to the hospital with a giant strangulated ventral hernia causing massive bowel ischemia and resulting in a short bowel. The literature on large abdominal wall hernias leading to short bowel is reviewed, and a discussion on short bowel syndrome is also presented.

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Ventral hernia is a routine surgical problem with significant economic and public health consequences, particularly if treated late or present in neglected patients. Strangulated hernias may lead to visceral damage that increases a patient's risk for

morbidity and mortality. These complications can be prevented if diagnosed earlier (when small), and they can be repaired as a day case with good outcomes. The literature on large abdominal wall hernias leading to short bowel is reviewed, and a discussion on short bowel syndrome is also presented. Our objective in presenting this particular case is to highlight the importance of timing in surgical intervention for such particular cases.

Case Report. A 54-year-old male was admitted to the Accident and Emergency Department at King Saud Medical City, Riyadh, Kingdom of Saudi Arabia, with a huge ventral hernia on June 14, 2013 at 11 PM. The hernia had markedly increased in size within the previous month, and for the past week it had become stony, hard, painful, and irreducible, as in **Figure 1**. These changes were also associated with vomiting and absolute constipation. The patient's detailed history also depicts that he had experienced a road-traffic accident 20 years earlier, which resulted in dysarthria and rendered him socially inactive. No other significant past surgical and medical history was available. He was not on any specific medications.

The physical examination revealed a dehydrated patient with a pulse rate of 104 beats/minute, but his blood pressure was within normal range. There was a huge ventral hernia, around 25 × 30 cm, almost equal to the size of a football, which was tense and tender, bulging more on the right side of the abdomen. Skin discoloration overlying the hernia was also noted. Bowel sounds were not audible and a rectal examination was unremarkable. A laboratory investigation revealed leukocytosis. Chest x-ray showed no air under the diaphragm. Abdominal x-ray showed dilated bowel loops with few air fluid levels. A diagnosis of strangulated ventral hernia was made. He was resuscitated with isotonic fluids. Intravenous cefuroxime and metronidazole were also given before surgery. After resuscitation, and once he provided consent and an intensive care unit (ICU) bed arrangement was made, he was shifted directly for operation from the emergency department without any further investigations or delays

within 2 hours from presentation to the emergency room. Laparotomy revealed that almost the entire small bowel and some part of the large bowel were protruding out of the abdomen in the hernial sac, featuring some hemorrhagic fluid. There was a constriction at the abdominal wall that was shelf-like, and there was pressure over the mesentery due to the tight neck of the sac, with a size of around 5 × 5 cm. The huge hernial sac was opened, containing offensive fluid and gangrenous bowel, mesentery, and omentum. The pressure within the hernial sac was so high that upon opening of the sac, hemorrhagic fluid came out like a shower. The gangrenous area included most of the jejunum, as well as the entire ileum, cecum, and the ascending and proximal half of the transverse colon, as in **Figure 2**. The constriction was divided and full trial was given (100%



Figure 1 - Huge ventral hernia with skin changes.



Figure 2 - Huge hernial sac opened showing gangrenous gut.

oxygen and warm sponging of gut) to regain the viability of the gut. A clear demarcation line was established. The gut was totally black, foul smelling, and had lost its sheen and luster; there was no peristalsis. Even after full trial, viability of the gut was not regained. Secondly, the mesentery was also black, and the vessels in the mesentery were gangrenous due to continuous pressure of hard shelf-like sac. In addition, the general condition of the patient was not good during either anesthesia or surgery due to the septic, gangrenous gut. While keeping of all of these things in mind, the decision was made to resect the gangrenous bowel and omentum. The gangrenous sac was also excised. After resection, only 60 cm of proximal jejunum remained, and this portion was clearly viable with all of its features, which was anastomosed to the remaining parts of the healthy transverse colon. There were no doubts concerning the remaining portion of the gut and mesentery, so a decision was made to perform primary anastomosis. No healthy parts of the gut were excised; however, clear cuts of healthy, safe margins for anastomosis were preserved. The patient subsequently ended up with a short bowel. Direct hernia repair was performed without prosthetic mesh. Histopathology showed a gangrenous bowel with clear margins. The postoperative course was uneventful. Initially, total parenteral nutrition (TPN) was started, and it was gradually overlapped with enteral feeding later on. There were a few episodes of diarrhea that were managed conservatively. He tolerated the gradual increase of enteral feed and was discharged from the hospital after 30 days, when he went completely off TPN, tolerating full enteral feeds. He was regularly followed in the outpatient surgical clinic with short bowel syndrome. He is currently on a combination of an enteral and normal diet. He is passing mixed-consistency stool 4 to 5 times a day. He lost approximately 30 kg of weight during the last 3 months. His dietary issues were managed with the help of a dietitian.

Discussion. Paraumbilical hernia repair is one of the most commonly practiced operations in the surgery department; giant abdominal hernias are less common.¹ Multiparous females are more likely to be affected.² Generally, hernia cases are operated on as day cases, except for instances when patients have a comorbid condition. However, if the patient has neglected the condition of his or her hernia, then these cases become challenging and require inpatient care.² In the current case report, a male patient had an enormous strangulated paraumbilical hernia. According to the patient, he initially had a small-sized hernia that gradually increased in size over a long period of neglect.

When he had severe symptoms of strangulated hernia, he presented to the hospital and underwent laparotomy. Most of his gut was gangrenous and, ultimately, the patient ended up with a short bowel - namely only the proximal jejunum remained, which was about 60 cm from the ligament of Trietz. Due to clear demarcation of the gut by a tight band, the rest of the area was free of gangrene. The margins were quite healthy, though they bled profusely, and the mesentery was also healthy in the spared area. As such, it was decided that the patient would not undergo a second exploratory procedure; rather, he would be followed at the clinic.

Early surgical treatment for such cases has good prognostic value.³ This report describes a case of a neglected hernia, which led to increased morbidity and a hospital stay. A giant paraumbilical hernia, if strangulated, may cause massive mesenteric ischemia.¹ Short bowel syndrome (SBS), which is also known by other names, such as short gut syndrome or, simply, short gut, is a malabsorption disease of the intestine caused by the surgical removal of the small intestine; it is sometimes due to the complete dysfunction of a large segment of the bowel.³ Short bowel syndrome usually develops when there is less than 2 meters (6.6 feet) of the small intestine left to absorb sufficient nutrients.⁴ Short bowel may be a temporary condition due to the adaptive property of the small intestine.⁵ Nutrients are not properly absorbed into the body (malabsorption); as a result, risk factors - including diseases of the small intestine (such as Crohn's disease) - may require surgery. In infants, necrotizing enterocolitis is a common cause. Although the majority of cases are acquired, some children are born with a congenitally short bowel.⁶ Short bowel syndrome can also be caused by a disease or injury that prevents the small intestine from functioning as it should despite a normal length. Specific nutrient deficiencies may occur depending on which sections of the small intestine were removed or were not functioning properly. The sites of nutrient absorption in the small intestine include the duodenum (which is the first section of the small intestine and where iron is absorbed), the jejunum (the middle section of the small intestine where carbohydrates, proteins, fats, and vitamins are absorbed), and the ileum (the last section of the small intestine where bile acids and vitamin B12 are absorbed). People with short bowel syndrome are also at risk for developing food sensitivities.⁶ Diarrhea is the main symptom of short bowel syndrome. Diarrhea can lead to dehydration, malnutrition, and weight loss. Other symptoms may include cramping, bloating, heartburn, weakness, and fatigue. Specific

nutrient deficiencies may occur, depending on which sections of the small intestine were removed or were not functioning properly.

There is no actual cure for short bowel syndrome.⁶ In newborn infants, the 4-year survival rate while on parenteral nutrition is approximately 70%. In newborn infants with less than 10% of their expected intestinal length, the 5-year survival rate is approximately 20%.⁶ A high-calorie diet that supplies key vitamins and minerals, as well as carbohydrates, proteins, and fats, is essential. Vitamin B12, folic acid, and increased iron should also be included in the diet to treat anemia. The main treatment for short bowel syndrome is nutritional support. Treatment may involve the use of oral rehydration solutions, parenteral nutrition, enteral nutrition, and medications. Oral rehydration solutions consist of sugar and salt liquids. Parenteral nutrition delivers fluids, electrolytes, and liquid nutrients into the bloodstream intravenously - through a tube placed in a vein. Enteral nutrition delivers liquid food to the stomach or small intestine through a feeding tube.

Some studies suggest that much of the mortality associated with this condition is due to a complication of the parenteral nutrition (TPN), especially chronic liver disease.⁴ Recent case reports have shown promising results in a type of lipid TPN feed known as Omegaven, in which the risk of liver disease is lower.⁷ In a process called intestinal adaptation, physiological changes to the remaining portion of the small intestine occur to increase its absorptive capacity. These changes include enlargement and lengthening of the villi found in the lining, an increase in the diameter of the small intestine, a slowdown in peristalsis, or the movement of food through the small intestine.⁸ Intestinal adaptation can take up to 2 years to occur.⁴ People with short bowel syndrome are also at risk for developing food sensitivities.⁸ Symptoms of short bowel syndrome are usually addressed by prescription medicine. These include anti-diarrheal medicine (for example, loperamide, codeine) to increase transient time in the intestine, vitamin and mineral supplements and L-glutamine powder mixed with water, H2 blockers and proton pump inhibitors to reduce stomach acid, and a lactase supplement (to improve the bloating and diarrhea associated with lactose intolerance). Surgery, including intestinal lengthening, tapering, and small bowel transplant may also be performed. Parenteral nutrition (or TPN - nutrition administered intravenously) or nutrition administered via a gastrostomy tube are typically required.^{8,9}

In conclusion, although promising, transplant of the small intestine has a mixed success rate, with a

postoperative mortality rate of up to 30%. The one-year survival rate is 90% and for the 4-year 60%.⁹ Timing in surgical intervention for such particular cases is very important by health care providers, to avoid such bad complications and treatment for these complications is not so promising.

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Case Reports

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