Proceeding S.Z.P.G.M.I vol: 8(1-2) 1994, pp. 52-55.

Short Bowel Syndrome and the Surgeon: A Review With Case Reports

K.M. Durrani, G.M. Arain, Shaukat Ali
Department of General Surgery, Shaikh Zayed Hospital, Lahore

INTRODUCTION

Short Bowel Syndrome (S.B.S) or intestinal failure may be the end result of a wide variety of disease processes that impair the ability of the gut to adequately digest and absorb food. If untreated this will lead to progressive nutritional depletion and failure to thrive. In children short bowel syndrome results from congenital malformations, entero-colities[1]; whereas in adults vascular catastrophe resulting in bowel infarction requiring massive resection is one of the most common indications for long term total parenteral nutrition (T.P.N). The causes of mesenteric vascular disease include embolic and thrombotic occlusions, non-occlusive mesenteric ischemia, chronic mesenteric ischemia and venous thrombosis[2] Multiple resections for conditions like inflammatory bowel disease, patients with intestinal fistulae chronic bowel obstruction, are other examples of short bowel syndrome encountered in surgical practice[3].

We report two cases who were managed in the department of Surgery at Shaikh Zayed Hospital, Lahore over a period of 3 years from 1990-1993.

Patient 1

A 23 years old male presented in emergency with severe abdominal pain, more marked in left hemiabdomen, vomiting, abdominal distension and absolute constipation of 48 hours duration.

Patient was dehydrated, tachycardiac and febrile, (10 °F) with blood pressure 95/70 mmHg. He was tender in whole abdomen, more marked in left hemiabdomen with a positive rebound. Bowel sounds were sluggish and rectal examination was unremarkable.

Laboratory findings were, Hb 12-gm%, ESR 26 mm, TLC $17x^{10}$/L, Poly 70% lympho 28% Urea 38 gm/dl. S. Amylase 28 U/L serum electrolytes and liver function tests were in the normal range. Plain X-Ray abdomen showed multiple air fluid levels.

Patient was resuscitated and a laparotomy planned with a provisional diagnosis of strangulation/ peritonitis.

Exploration revealed an inflammatory mass involving the proximal small bowel wrapped in greater omentum. On taking down adhesions a 60 cm loop of proximal jejunum was found gangrenous. Rest of small bowel loops were densely adherent to each other and with areas of patchy necrosis, involving distal jejunum and proximal ileum. Mesentery of the small bowel was haemorrhagic with no pulsations.

Resection of nonviable small bowel about 45 cm from doudenojejunal flexure to about 30 cm from ileocaecal junction was carried out. End jejunostomy was established in the left lumbar region with closure of distal ileum, on account of doubtful viability of the proximal jejunum. Post operatively the patient was managed on total parenteral nutrition for 3 weeks providing 2000-2500 K-cal/day through a central (subclavian) line. His jejunostomy secretions were controlled with loperimide till, daily out put of thick intestinal secretions of about 800 ml was achieved. Then he was started orally on to low residue thin liquids gradually increasing in amount and shifting to low residue semisolid diet. He was discharged about six weeks after the operation. His weight at time of discharge was 40 Kg. He was tolerating semisolid diet well and his jejunosty out put being 600-800 ml/dl.

The histopathology revealed gangrenous small bowel with non-specific inflammation and thrombosis of the mesenteric vessels.

Three months after surgery he was brought to emergency with severe vomiting for the previous 48 hours. He was dehydrated, tachycardiac and tachypnic. The jejunostomy was not working and he was admitted in a grossly malnourished state.

This time his weight was 36 Kg, Hb. 10.7 gm% TLC $17.9x^{10}$/L, Urea 40 gm/dl serum amylase 17 U/L X-Ray abdomen showed few fluid levels.

He was rehydrated and parenteral nutrition commenced through a central venous line.

Two weeks later the patient was operated and jejunoileal anastomosis was established. Total length of small bowel was about 1.5 meters. Post operatively for about 6 weeks parenteral nutrition was continued via a central venus line. Low residue liquid diet was started during the 2nd post op. week, gradually switching over to semisolid diet and later discharged after six weeks.

On his last follow up six months post op. he was doing well and had gained about 10 Kg weight, thereafter he has not shown up.

Patient 2

A 21 years old male admitted through Accident and Emergency department for abdominal pain, distension, vomiting and absolute constipation of 12 hours duration.

The pain was severe, starting in the umbilical region and spreading to the whole abdomen. Past history was unremarkable.

Examination revealed signs of hypovolemia. His pulse was 110/m BP 70/40 mm, respiratory rate 28 and temperature 98.4°F. Abdomen was tense, tender and guarded, redound tenderness was positive. Rectal examination was unrevealing.

Laboratory findings were Hb 13 gm% TLC $15.9x10^9$/L serum electrolytes within normal range, urea 64 gm% creatinine 2.2 gm% S. Amylase 28 U/L.

X-Ray plain abdomen showed multiple air fluid levels. A provisional diagnosis of bowel strangulation was made.

Following initial resuscitative measures, laparotomy was performed through right paramedian incision. Peritoneal cavity contained about 1 liter dirty foul smelling haemorrhagic fluid, whole of the small bowel was gangrenous about 5 cm from the doudenjejunal flexure down to about 15cm from ileocaecal valve as a result of small bowel volvulus, the gut being rotated anti-clock-wise twice on its mesentery. There was a cyst present at the anti-mensenteric border of mid-jejunum. The gut was untwisted. The mesenteric vessels were found to be non palpable. So it was decided to resect the gangrenous small bowel.

The deudenum was mobilized completely and after mobilizing the ileocaecal function, end-to-end anastomosis between the duodenum and terminal ileum performed about 10 cm proximal to ileocaecal junction.

Parenteral nutrition through the central venous line was started post-operatively. Oral sips were started on 8th post op. day. Initially the patient was given isotonic fluids like glucose. The concentration of the fluids was increased gradually, and diarrhea controlled with loperimide Caps four hourly. The patient was discharged at 4 weeks with a tunneled polyurethane C.V. line and an advice on low residue diet with oral loperimide. His home parenteral nutrition regimen composed of 1000 ml 25% glucose with 500 ml 5% aminoacid solution 75 Meq Kcl + Inj. B. complex (5 ml) daily and 250-500 ml lipid solutions (10-20%) twice a week. Inj B12 1000 I.U. I/m every two months. The patient was followed up in the Out patient clinic for about one year and then was lost to followup. During this time he was hospitalized twice for change of central line due to catheter related complications.

DISCUSSION

Recent advances in the understanding of the adaptive responses, surgical techniques and parenteral nutrition has improved the survival of patients with short bowel syndrome[3-5].

In adults short bowel syndrome results after extensive resection of small intestine for mesenteric vascular occlusion, mid gut valvulous, chronic diseases traumatic or iatorgenic disruption of the superior mesenteric artery, neoplasm and radiation enteropathy.

The Clinical features of S.B.S include diarrhea, fluid and electrolyte deficiency and malnutrition. The severity depends upon the extent and site of the small bowel resected. Proximal bowel resection is tolerated much better than distal resections. The outcome depends upon the extent of resection, quality of the remaining gut and the nature of the underlying disease. The minimum length of the bowel for sufficient absorption is still controversial, however resection of 70-80% of the small bowel or a remaining intestine of 100 cm or less is associated with sever metabolic sequele, that require intensive intestinal support[6].

The pathophysiology of massive small bowel resection has been well documented[7-10]. A minimum of 100 cm of ileum is required for the complete absorption of bile salts. The ileum compensates for most of the absorptive functions but not for the secretion of enterohormones by the jejunum. Gastric hyper-secretion, greater after jununal resection results from the loss of these inhibitory hormones

secreted by the jejunum. Hypergastrinemia and hyper secretion occurs weeks to months after resection leading to peptic ulceration and steatorrhoea. Ileal resection and loss of ileocaecal valve results in disruption of enterohepatic circulation, which may lead to the formation of gall stones, and anemia from B12 deficiency.

Malabsorption of Vit. D, calcium and protein will lead to metabolic bone disease. (Osteomalacia and Osteoporosis), Vit K deficiency produces coagulation defects i.e. purpura, generalized bleeding. B-complex deficiency results in peripheral neuropathy.

The colon normally functions as a major site of water and electrolyte absorption and may also absorb some nutrients. The colon in the absence of small bowel may increase its absorptive capacity 3-5 times. Malabsorbed carbohydrates reaching the colon are fermented by bacteria to yield short chain fatty acids, which are absorbed in the colon and provide source of energy[11]. On the other hand, an intact colon is responsible for chloritic diarrhoea due to unabsorbed bile salts, reaching the colon, and excessive absorption of oxalates leading to oxalate stone formation. Hence patients with intact colon should avoid oxalate containing foods such as chocolate, cola drinks, tea, carrots, spinach, nuts etc. and the diet supplemented with oral calcium gluconate/lactate.

The bowel has an intrinsic capacity to adapt after resection. Multiple factors are responsible for successful intestinal adaptation. Luminal nutrients, trophic gut hormones pancreatic and biliary secretions are all required. There is increase in the calibre of the remaining bowel, hypertrophy of the gut wall, increase in villous length, the process taking weeks to months to complete[12].

The most important principle in the management of S.B.S. is prevention, which would mean an attempt at re-vascularization of the bowel, and limiting the bowel resection to minimum.

There are three post op. phases of short bowel syndrome[6]. The first phase is a period of fluid and electrolyte loss due to profuse diarrhoea. The watery diarrhoea decreases over 1-3 months. This phase is managed by the control of diarrhoea with medication, replacement of fluid and electrolytes and nutritional support through total perenteral nutrition via a central venous. line. The second phase is the period when most of the adaptation in the remaining bowel occurs. Diarrhoea stabilizes, and positive fluid and electrolyte balance may be achieved with oral intake. T.P.N. is continued during this phase, and minimal enteral feeding started to stimulate the gut.

The third phase of full adaptation may last for 3-12 months, there being improved tolerance to oral diets and positive balance of all nutritients may be achieved by entral feeding. During this phase tapering off of the T.P.N. is carried out. Not all patients can attain this stage of full adaptation.

H2 antagonists and drugs affecting motility like loperimide, codeine are helpful in controlling diarrhoea.

Enteral diets such as elemental diets (Vivonex, Flexicol) or polymeric diets (Isocal, Ensure) may be used. Initially these diets should be started in Iso-osmolar concentration and as the adaptation progresses the osmolarity, volume and caloric content is increased. Medium-chain triglycrides may be added as these are absorbed in the proximal bowel without miccelle formation. Vitamins esp. Fat soluble Vitamins, Ca, Mg are also required which may be given parenterally initially.

However for those patients with inadequate residual bowel length, home parenteral nutrition will have to continued indefinitely[13].

Nutritional support is recognized as an important therapeutic intervention to promote wound healing sustained traumatically, surgically or caused by debilitating illness.

Total Parenteral Nutrition (T.P.N). has a well established role in the following conditions in surgical practice[14].

1. To maintain life in patients with short bowel syndrome.
2. To improve the quality of life in patients with inflammatory bowel disease
3. To assist the healing of enterocutenous and pancreatic fistulae.

The efficacy of home parenteral nutrition is well established, but it involves a multidisciplinacy approach[2,15-19]. However the administration of T.P.N. for prolonged period is associated with high incidence of liver dysfunction. This is menifested by elevation of liver enzymes and histologically fatty infiltration of liver with cholestasis[20,21].

The surgeons in an attempt to treat this complex problem of intestinal failure have tried a number of procedures aimed at bowel lengthening,

increasing the transit time and even intestinal pacing. These include interposition of reversed segment recirculating loops, valve reconstruction, bowel lengthening but so far the results are variable[20,22-24]. Intestinal transplantation which is still experimental may one day provide ultimate solution to short bowel syndrome.

Despite the recent development of total parenteral nutrition and home parenteral nutrition, the long term survival of patients with short bowel syndrome is not satisfactory. This is mainly due to catheter related complications and hepatic steatosis. Till such time when a permanent solution to the problem of short bowel syndrome is found, the outcome depends upon the level and extent of bowel resection, process of adaptation, and last but not be least, the commitment of the team responsible for parenteral nutrition.

REFERENCES

1. **Leong GM, Drew JH.** Necrotizing entrocalitis: A 15 year experience. *Aus NZ J Obst-Gynae 1987;* **27**: 40-4.
2. **Muller C, Borriello R, et al.** Parentral nutrition support of a patient with chronic mesenteric artery occlusive disease. *Nutr clin Pract 1993;* **8**: 73-7.
3. **Scott NA, Irwing MH.** Intestinal failure - the clinical problems Dig. Disease 1992; 10(5): 249-57.
4. Hehir DJ; Gorey TF. Intravenous Nutritional support and the Surgeon: Where next. *Ir J Med Sci 1992;* **161**: 401-3.
5. **Matuchansky C, Massing B, Jeejeebhay KN, et al.** Cyclical parenteral nutrition. *Lancet 1992;* **340**: 588-92.
6. **Rombeau JL, Ralondeli RL.** Enteral and Parenteral Nutrition in patients with enteric fistulas and short bowel syndrome. *Surg Clin N Am 1987;* **67**: 551-71.
7. **Wright HK, Tilson MD.** Short gut syndrome, Pathophysiology and treatment. *Curr Probl Surg 1971;* **8**: 1-51.
8. **Tilson MD.** Pathophysiology and treatment of short bowel syndrome. *Surg Clin N Am 1980;* **60**: 1273-1284.
9. **Mackenzie GW, Bolleau GR.** Short gut syndrome a review with a case report. *Can J Surg 1973;* **16**: 3-11.
10. **Sawyers JL, Herrington JL, Jr.** Short gut syndrome. *Surg Annual 1972;* 4: 273-84.
11. **Philips SF, Giller J.** The contribution of Colon to Electrolytes and water conservation in Man. *J Lab Clin Med 1973;* 81: 733-46.
12. **Betzhold J, Howard L.** Enteral Nutrition and Gastroentestinal disease. In: **Rombeau JL, Caldwell MD (eds).** *Clin Nutrition* Vol I, pp. 338-361. Entral and tube feeding. Philadelphia WB Sanders 1984.
13. **Burnes JU, O'Keefe SJ, Flaming CR, et al.** Home parenteral nutrition, 3 years analysis of clinical and laboratory monitoring. *J Parent Enteral Nutr 1992;* **16**: 327-32.
14. **Buzby GP.** Overview of randomized clinical trials of total parentral nutrition for malnourished surgical patients. *World J Surg 1993;* **17**: 173-7.
15. **Weser E.** Short bowel syndrome in Bayless Tm (Ed) Current Therapy in Gastroenterology and liver diseases. Philadelphia BC Decker. 1986. PP
16. **Skaer TL.** Total parentral nutrition, clinical considerations. *Clin Ther 1993;* **15**: 272-82.
17. **Steriger E, Srp F, Helbey M, et al.** Home Parenteral nutrition. In: **Rambeau JL Coldwell MD (Eds).** *Clin Nutrition* Vol II, pp. 654-659. Parenteral Nutrition Philadelphia WB Souders. 1986
18. **McCrae JD, O'Shea R, Udine LM.** Parenteral Nutrition. Hospital to home. *J Am Diet Assoc 1993;* **93**: 664-70.
19. **Bisset WM, Stapleford P, Lang S, et al.** Home Parenteral nutrition in chronic intestinal failure. *Arch Dis child 1992;* **67**: 109-14.
20. **Lindor KD, Flaming CR, Abroms A, et al.** Liver function values in adults receiving total parenteral nutrition. *JAMA 1979;* **241**: 2398-2400.
21. **Langrehr JM, Reilly MJ, Banner B, et al.** Hepatic steatosis due to T.P.N.: The influence of short gut syndrome, refeeding and small bowel transplantation. *J Surg Res 1991;* **50**: 335-43.
22. **Muraji T, Tsugova C, et al.** Surgical management of massive involvement of the small bowel and of short gut syndrome. *Nippen Geka- Gakkai-Zasshi 1980;* **89**: 1382-6.
23. **Boeckman CR, Traylor R.** Bowel lengthening for short gut syndrome. *J Paed Surg 1981;* **16**: 996-7.
24. **Jee-Jeebhay KN.** Therapy of short gut Syndrome. *Lancet 1983;* **25**: 1427-30.

The Authors:

K.M. Durrani,
Associate Professor,
Department of General Surgery,
Shaikh Zayed Hospital,
Lahore.

G.M. Arain,
Registrar
Department of General Surgery,
Shaikh Zayed Hospital,
Lahore.

Shaukat Ali.
Department of General Surgery,
Shaikh Zayed Hospital,
Lahore.

Address for Correspondence:

K.M. Durrani,
Associate Professor,
Department of General Surgery,
Shaikh Zayed Hospital,
Lahore.