Small-bowel necrosis associated with jejunal tube feeding

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Objectives: To report 3 cases of small-bowel necrosis after jejunal tube feeding and to review the literature concerning this condition.

Design: A 5-year retrospective review.

Setting: A 560-bed university-affiliated tertiary-care teaching hospital.

Patients: Three patients who had bowel necrosis out of 386 who received jejunal tube feedings.

Results: The patients experienced small-bowel necrosis as a consequence of jejunal feeding. The ischemic necrosis was preceded by progressive abdominal pain, distension and high nasogastric output. All 3 patients required extensive small-bowel resection. Although survival was rare in previous reports, our 3 patients survived after prompt surgical intervention and small-bowel resection.

Conclusions: Although the death rate for this condition approaches 70%, timely recognition and surgical intervention can save the patient's life.

Objectifs : Présenter un rapport sur trois cas de nécrose de l'intestin grêle après un gavage jéjunal et revoir les écrits portant sur ce problème.

Conception : Révision rétrospective de cinq ans.

Contexte : Hôpital d'enseignement de soins tertiaires de 560 lits affilié à une université.

Patients : Trois patients victimes d'une nécrose intestinale sur 386 qui ont reçu un gavage jéjunal après une intervention.

Résultats : Les patients ont été victimes d'une nécrose de l'intestin grêle à la suite du gavage jéjunal. La nécrose ischémique a été précédée d'une douleur abdominale progressive, d'une dilatation et d'une production nasogastrique élevée. Dans les trois cas, il a fallu réséquer une partie importante de l'intestin grêle. Même si les cas de survie étaient rares dans les comptes-rendus antérieurs, les trois patients en cause ont survécu après une intervention chirurgicale rapide et une résection de l'intestin grêle.

Conclusions : Même si le taux de mortalité causé par ce problème atteint presque 70 %, l'identification rapide du problème et une intervention chirurgicale peuvent sauver la vie du patient.

Early postoperative enteral feeding has been advocated after major upper abdominal procedures, in multisystem trauma, as well as in cancer therapy and in the malnourished patient.[1] The advantages relate to a diminished catabolic stress response, improved immunity and better wound healing.[2] The enteral route has advantages over the parenteral route owing to enterocyte growth stimulation and an improved mucosal barrier thereby decreasing bacterial translocation.[3] Enteral feeding is also reported to be safer and more cost-effective than the parenteral route.[4] Using the small intestine as an access site for enteral feeding has been shown to reduce the incidence of aspiration pneumonia in comparison with the use of the stomach.[5] In addition, motility studies have documented return of small-bowel peristalsis within hours of the insult, whereas gastric motility lags 36 to 48 hours behind.[6]

Complications of enteral feeding by jejunostomy are reported to be approximately 5% and are related to diarrhea, nausea, crampy abdominal pain and distension. Catheter dis-

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Accepted for publication Jan. 20, 1998

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placement, leaking or obstruction are also relatively common.[7] Infrequently, however, nonspecific intestinal complaints of abdominal distension and high output from the nasogastric tube have been harbingers of a syndrome of hypotension, shock and extensive small-bowel necrosis. Between 1992 and 1996 inclusive we encountered this complication in 3 of 249 patients receiving continuous jejunostomy feeding through surgically placed feeding jejunostomy catheters.

Case reports

Case 1

A 65-year-old woman with Barrett's esophagus and severe dysplasia underwent transhiatal esophagectomy and placement of feeding catheter jejunostomy by the modified Witzel technique. The procedure was performed without complication and no episodes of perioperative hypotension were noted. Tube feedings were started on the first postoperative day with a balanced 300 mOsm/kg formula at a rate of 25 mL/h and were advanced by 25 mL/h every 12 hours to a rate of 75 mL/h. On the fifth postoperative day abdominal distension and increased nasogastric output developed requiring cessation of tube feeding. Fever, abdominal pain consistent with peritonitis, hypotension, and respiratory and renal failure quickly developed. An urgent laparotomy on postoperative day 5 revealed patchy, full-thickness, hemorrhagic necrosis of the small bowel, beginning 10 cm proximal to the jejunostomy insertion site and extending to the cecum. No perforation, obstruction, torsion at the jejunostomy site or major vascular occlusion was noted. The patient survived after multiple small-bowel resections and did well with no evidence of short gut syndrome at follow-up. Pathological examination of resected specimens revealed diffuse ischemic necrosis with areas of full-thickness necrosis interspersed with areas of preserved mucosal architecture.

Case 2

A 57-year-old man who had adenocarcinoma of the distal esophagus, underwent transhiatal esophagectomy and placement of a feeding catheter jejunostomy by the modified Witzel technique. The procedure was performed without complication and no episodes of perioperative hypotension were noted. Tube feedings were started on the first postoperative day with a balanced 375 mOsm/kg formula at a rate of 25 mL/h and were advanced by 25 mL/h every 12 hours to 75 mL/h. This rate was maintained for 24 hours and was advanced further to 100 mL/h on the fourth postoperative day. That same day, the patient had abdominal distension and an increase in nasogastric output. The patient's condition rapidly progressed with fever, abdominal pain consistent with peritonitis, hypotension, renal and respiratory failure with evidence of pneumatosis intestinalis on computed tomography. Emergency laparotomy showed patchy, hemorrhagic, full-thickness necrosis of the small bowel, beginning at the J-tube insertion point and extending distally over most of the jejunum and ileum. No perforation, obstruction, torsion at the jejunostomy site, or major vascular occlusion was noted. The patient survived after multiple, segmental small-bowel resections and in follow-up showed no evidence of short gut syndrome. Pathological examination revealed segmental ischemic necrosis with areas of submucosal congestion consistent with ischemia and full-thickness necrosis in other areas.

Case 3

A 71-year-old woman who had carcinoma of the sigmoid colon and colovesical fistula underwent resection. She was left with an end colostomy. Two years later she presented with a left flank mass and small-bowel obstruction. At the time of laparotomy the proximal jejunum was obstructed by this mass, which was suspicious for recurrent carcinoma. A gastrojejunostomy was constructed to bypass the obstruction. Gastrostomy and feeding jejunostomy catheters were placed using the Stamm and modified Witzel techniques respectively. The procedure was performed without complication and no episodes of perioperative hypotension were noted. Tube feedings were started on the second postoperative day with a balanced 300 mOsm/kg formula at a rate of 25 mL/h for 48 hours. The rate was advanced to 50 mL/h on the fifth postoperative day. On the seventh postoperative day the patient had abdominal pain and distension, and increased gastrostomy tube output. The tube feedings were discontinued. Abdominal pain consistent with peitonitis rapidly developed along with hypotension, respiratory failure and lactic acidosis. Emergency laparotomy revealed patchy, full-thickness small-bowel necrosis beginning near the jejunostomy insertion site and extending distally. The intervening bowel was healthy and there was no evidence of perforation, obstruction, torsion at the jejunostomy site or vascular occlusion. The patient survived after 2 small-bowel resections and ileostomy formation and a relook laparotomy within 48 hours requiring a further small-bowel resection. Pathological examination of the excised specimens revealed grossly distended, dilated intestine with mucosal ulceration in some areas and extensive full-

thickness necrosis in other areas. There were also foci of intramural hemorrhage, inflammatory infiltrate and ischemia.

Discussion

Enteral feeding by catheter jejunostomy is quite common in modern surgical practice and the frequency of related complications is relatively low.[7] The most commonly encountered complications are those related to the gastrointestinal tract such as diarrhea, abdominal cramping and distension and mechanical problems related to the catheter itself such as occlusion, dislodgement or leaking.[8] There have been 5 reports describing 18 cases of a more serious and highly lethal complication of extensive small-bowel necrosis in the early postoperative period related to jejunostomy catheter feedings. Thompson[9] first reported this complication in 1983. He described the case of a 20-year-old man who experienced necrosis of his entire small bowel 4 to 5 days postoperatively while receiving jejunal catheter feedings. Gaddy and associates[10] reported 5 patients in whom extensive small-bowel necrosis developed between 3 and 15 days of beginning jejunostomy tube feedings. All of these patients died of related complications. Smith-Choban and Max[8] reported small-bowel necrosis in 5 patients receiving continuous jejunal tube feedings. Four of these patients were noted to have necrosis of the entire small bowel, the fifth had segmental, patchy necrosis. All were without evidence of obstruction or vascular occlusion. All 5 patients died. Recently, Schunn and Daly[11] reviewed previously published cases and reported 4 patients of their own who had small-bowel necrosis between 6 and 11 days after jejunostomy catheter insertion. Two of these had patchy necrosis and both survived whereas 2 others had subtotal small-bowel necrosis at exploration and both died. Three further reports have been described by Myers, Page and Stewart[1] in which 2 patients exhibited pannecrosis of the entire small bowel; both died. A third patient, explored laparoscopically, showed segmental, patchy ischemia, was treated nonoperatively and survived.

Although the rate of occurrence of this problem appears to be relatively low, the resulting high death rates suggest that further consideration should be given to potential causes. A multifactorial cause is likely. In the early postoperative period, jejunal feedings, because of osmolar load, bacterial overgrowth or metabolic breakdown products, may cause bowel distension, impaired blood flow with mucosal injury and resulting ischemia.

Nonocclusive mesenteric ischemia has been postulated as a cause by some who have previously described this complication.[10] Patients receiving continuous enteral feeding are at potential risk for low flow mesenteric states. In man the splanchnic blood flow increases by approximately 40% after meals.[12] Continuous feeding would require an elevated blood flow to the splanchnic bed to meet increased metabolic demands.[10] Previous reports describe coexisting risk factors that may contribute to decreased mesenteric blood flow in patients receiving continuous enteral feeding. These are low cardiac output, hypotension, atherosclerotic vascular disease and congestive heart failure.[8,10] It has been proposed that because the ischemia only encompasses the distribution of the superior mesenteric artery and that the lesions were typical of ischemic necrosis, a vascular insult was likely.[10] Although this may be a contributing factor, it is unlikely to explain the problem. In the majority of reports describing this complication, including our own, cardiovascular risk factors for a mesenteric low flow state have been absent in most patients, who have been as young as 18 years. In fact we found that the development of hypotension was secondary to the onset of the other well-described symptom prodrome, and hypotension is more likely a consequence than a cause of this complication. Previously reported cases[11] have described demonstrable mesenteric pulses in the distribution of the ischemic segments, and findings were quite similar in our series, essentially eliminating an occlusive vascular event as an explanation.

It has been suggested that infarction of the bowel may be secondary to distension of the gut by the feedings, resulting in secondary hypoperfusion of the gut wall and mucosa.[10,11] Bypassing the stomach with jejunal feedings eliminates the normal dilutional capacity of the stomach and duodenum thereby exposing the mucosa to unphysiologic osmolar loads.[13] This may be an important contributing factor because the osmolality of the formulas used ranged from 300 to 375 mOsm/kg in our patients, a concentration that would be considered relatively isotonic in the stomach. Jejunal feedings likely present a relatively hypertonic load to the jejunum causing large fluid shifts and contributing to gut distension. Postoperative ileus may also contribute to the problem as this complication appears to be limited to patients in the postlaparotomy period. Two of our patients underwent esophagectomy requiring relatively long operating times and small-bowel manipulation. Small-bowel necrosis has not been described in patients having a less invasive, percutaneous catheter placement in similar clinical settings. The resulting local bowel distension causes secondary sludging in the microvasculature and decreased perfusion.[10] This is supported by the obser-

vation that in most cases the necrosis was limited to bowel that was perfused with the feedings, and the more proximal bowel was spared. Furthermore, it has been suggested that bowel perfused with hyperosmolar tube feedings is more vulnerable to ischemic injury in general.[8]

Bacteria within the bowel lumen may directly contribute to this problem. Bacterial overgrowth may occur in these patients who are often receiving H_2 blockers or may have undergone vagotomy as did our patients who had esophagectomy. Excessive fermentation of carbohydrates may contribute to bowel distension, local vasospasm, disruption of mucosal integrity and resulting ischemia.[11] Bacterial contamination of tube feedings has been described and can result in abdominal cramping and distension, vomiting, fever and hypotension.[14] Bowel injury may occur as a result of direct bacterial invasion or bacterial toxins, leading to mucosal injury.[11] Contamination of feedings is an unlikely cause in our series as all formulas are carefully stored and monitored for expiry dates.

The pathogenesis of small-bowel necrosis following jejunal tube feeding is very similar to necrotizing enterocolitis in the newborn. The majority of cases of necrotizing enterocolitis occur after feedings have been initiated. A multifactorial model of pathogenesis has been suggested in necrotizing enterocolitis, implicating ischemia, infection and enteral factors.[15] In the clinical setting several factors probably participate and one stimulus may generate an initial mucosal injury; subsequently a variety of infectious and inflammatory events may contribute, resulting in extensive bowel injury. This may well provide an acceptable explanation for this rare but lethal complication of enteral feeding.

Summary and conclusions

Continuous jejunal catheter feeding is a widely accepted means of providing nutritional supplementation to the surgical patient and has a relatively low reported complication rate. However, small-bowel necrosis has been reported associated with a death rate approaching 70%. Of 18 cases reported to date, all were preceded by a syndrome of abdominal pain and distension with associated high nasogastric tube output that progressed to hypotension, shock and multisystem failure. If these symptoms should develop, enteral feedings should be discontinued and the patient monitored closely for progression of this syndrome. If bowel ischemia is suspected, early operative intervention with aggressive resection of ischemic bowel appears to be the only way to save the patient. Further consideration should be given to the enteral feeding regimens including the osmolality and the rate of volume increase used in the early postoperative setting. Although no procedure, however simple, is without risk, the benefits of a feeding jejunostomy tube greatly outweigh the likelihood of occurrence of small-bowel necrosis. It is crucial, however, to be aware that this potential complication exists so timely recognition and intervention may alter its lethal course.

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