Bowel Necrosis Associated With Early Jejunal Tube Feeding

A Complication of Postoperative Enteral Nutrition

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Hypothesis: Postoperative enteral nutrition may sometimes be responsible for severe complications such as mesenteric ischemia.

Data Sources: Studies in the English literature were identified by a computer-assisted search of the MEDLINE database using the key words “enteral feeding OR jejunostomy” AND “complications OR mesenteric ischemia.” Cited references of each retrieved paper were checked for relevance.

Study Selection: All reports of mesenteric ischemia in the setting of postoperative enteral feeding were included. In cases of multiple articles from the same institution with overlapping patients, the most exhaustive article was included.

Data Extraction: All reports were abstracted for number of patients, presence of preoperative comorbidities, development of perioperative hypotension or mesenteric occlusion, and outcome.

Data Synthesis: Nine studies were retrieved in which enteral feedings were responsible for bowel ischemia; we report an additional case. The common clinical picture is that of a patient without significant risk factors for mesenteric ischemia, which during the early postoperative course develops nonspecific abdominal symptoms and then rapidly progresses to septic shock and eventually to multisystem organ failure and death. Mesenteric ischemia may present in up to 3.5% of enterally fed surgical patients; the associated mortality approaches 100%. The lack of specific symptoms requires a high index of suspicion for diagnosis; prompt abdominal exploration and bowel resection are the only chance for survival.

Conclusions: The benefits of enteral nutrition outweigh the likelihood of severe complications; when mesenteric ischemia develops, early diagnosis is challenging and the prognosis is poor.

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Enteral feeding is believed to diminish stress response, improve immunity and wound healing, and significantly reduce septic complications after major upper abdominal procedures and in the setting of multisystem trauma. This most likely occurs by stimulating enterocyte growth, resulting in improved mucosal barrier function and decreased bacterial translocation. Motility studies that document return of small bowel peristalsis within hours after laparotomy provide the theoretical support for early postoperative enteral nutrition.

Enteral feeding has been reported to have very few complications, mostly diarrhea and aspiration; tube placement–related morbidity is uncommon and includes intraperitoneal leak, bowel obstruction, wound infection, and fistula formation. As a result, an increasing number of surgeons feed their patients postoperatively by means of a jejunal tube with little concern for complications. However, nonspecific signs of intolerance to tube feeding (diarrhea, nausea, bloating, abdominal pain) occasionally may progress to a syndrome of abdominal distension, hypotension, and hypovolemic shock and eventually to small bowel necrosis.

Our recent observation of a patient who suffered bowel ischemia secondary to enteral feeding stimulated us to review the existing literature to clarify pathophysiologic features, clinical features, and outcomes of a complication easily underestimated in its early presentation and with catastrophic results when not promptly addressed.

Report of a Case

A 54-year-old morbidly obese woman (body mass index [calculated as weight in kilograms divided by the square of height in meters], 49) with a history of hypertension and thyroid cancer presented with stage III esophageal adenocarcinoma. She underwent neoadjuvant chemotherapy and...
operative day. No autopsy was performed. The patient died of respiratory complications on the 25th postoperative day. Full-thickness necrosis of the small bowel was noted, especially at the site of the feeding tube and extending distally for 40 cm. The rest of the small bowel appeared viable. Doppler examination revealed patent mesenteric vessels both in the ischemic segment and throughout the remaining small and large bowel. No perforation, obstruction, or torsion at the jejunostomy site was noted. The patient underwent small bowel resection with primary anastomosis.

Esophagogastroscopy demonstrated a viable gastric tube with normal mucosa. A computed tomographic scan of the abdomen revealed mild ascites and distended loops of small and large bowel (Figure). We suspected an intra-abdominal source of sepsis and brought the patient to the operating room. On emergency abdominal exploration, full thickness necrosis of the small bowel was noted, beginning at the site of the jejunostomy and extending distally for about 40 cm; the rest of the small bowel appeared viable. The nasogastric tube output did not appear to contain enteral feedings but increased to 1000 mL/24 h; the enteral feedings were held. On the seventh postoperative day, the patient developed hypotension and worsening abdominal tenderness, shortly followed by respiratory and renal failure.

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We identified 9 articles in the English literature that described postoperative mesenteric ischemia in 31 patients in the absence of mechanical bowel obstruction (Table). Four articles specified the total number of patients exposed to enteral feeding during the observation period; the incidence of feeding-related bowel ischemia varied between 0.14% and 3.5%, the lowest incidence being observed in the largest population. Mesenteric ischemia occurred in patients of any age (mean, 48 years) operated on either for abdominal trauma or for elective organ resection. Comorbidities potentially responsible for low mesenteric blood flow such as diffuse atherosclerotic disease, diabetes, or congestive heart failure were usually absent; only a patient described by Gaddy et al had a medical history remarkable for 2 myocardial infarctions but did not have heart failure at time of presentation. The commonly described clinical picture was that of a patient who had undergone an abdominal procedure without complications and had been started on early enteral feeding through a jejunostomy or a nasojejunal tube. In the early postoperative course, nonspecific abdominal signs and symptoms (bloating, crampy pain, loss of bowel function) developed, rapidly followed by hyperdynamic shock and eventually multisystem organ failure and death. About 31% of patients suffered systemic arterial hypotension before or at the time of onset of abdominal signs. Exploratory laparotomies consistently showed bowel ischemia starting at the site of the feeding tube and extending distally for a variable distance. No author described occlusion of the mesenteric vessels. Mortality varied between 41% and 100%.

The concept of early postoperative enteral nutrition is supported by the observation of a blunted reactive hypometabolic response and reduction of septic complications in patients receiving immediate feedings, possibly on
solely responsible for this problem. Systemic hypotension is also a potential complicating factor in this clinical setting, and several cases of mesenteric ischemia secondary to enteral feeding have been reported in the literature. The pathogenesis of ischemic necrosis secondary to enteral feeding is likely multifactorial, including intraluminal factors such as hyperosmolarity of feeding and intestinal bacterial overgrowth. The absorption of intraluminal nutrients increases energy demands in metabolically stressed enterocytes, therefore putting the intestine at risk for ischemia in patients with systemic hypoperfusion; however, the gut can survive periods of profound hypoperfusion with protective mechanisms such as the ability to increase oxygen extraction and to redistribute flow within the intestinal wall favoring the mucosa. Furthermore, the majority of patients described in the literature (Table) and the patient observed by us did not suffer major episodes of hypotension or hypovolemia during the operation or in the postoperative period. Therefore, it is unlikely that either early hypovolemic shock with incomplete resuscitation or episodic hypotension later in the patient’s course would be solely responsible for this problem. Systemic hypotension did occur in our patient after gastrointestinal symptoms were already evident and therefore was believed to be secondary to the intra-abdominal process and not the cause. Moreover, in our patient as well in previous reports (Table), mesenteric pulses were clearly present in the vascular distribution to the ischemic segments, essentially eliminating an occlusive vascular event as an explanation.

Bacterial contamination of tube feedings can result in abdominal cramping and distension, vomiting, fever, and hypotension. Clostridium difficile colitis and its associated diarrhea has been noted to occur with increasing frequency in patients receiving postpyloric feedings. However, pathologic signs of enteric bacterial invasion or positive stool cultures for C difficile have been conspicuously absent in all reported cases of small-bowel necrosis related to tube feeding, and so far no specific infectious agent has been linked with nonocclusive bowel ischemia. Significant bacterial overgrowth is likely to occur, especially when enteral feeding is administered for prolonged periods in the setting of ileus or in patients who are receiving H2 receptor blockers or may have undergone vagotomy. Increasing concentrations of luminal toxins derived from the overgrowth of bacteria could cause a mucosal-submucosal inflammatory response; this, coupled with intraluminal gas production from substrate fermentation, could set up a vicious cycle of inflammation, distention, and dysmotility that eventually may impair mucosal perfusion, resulting in ischemic injury.

The composition of the tube feedings may also play a central role in the development of bowel ischemia. High concentrations of carbohydrates may provide substrates for excessive bacterial fermentation and therefore worsen bowel distention, edema, and subsequent ischemia. By-passing the stomach with jejunal feedings eliminates the normal dilutional capacity of the stomach and duodenum, thereby exposing the mucosa to significant osmolar loads. In the setting of disordered peristalsis, this hyperosmolar load may promote the release of proteolytic enzymes and may cause rapid fluid shift into the bowel lumen with resulting bowel distension, capillary sludging,
and decreased perfusion. The supplementation of fibers to the enteral feedings may prevent overgrowth of pathogenic bacteria and septic complications; however, its role on the gastrointestinal motility and susceptibility to bowel ischemia is still unknown in the clinical setting.

The clinical presentation of feeding-related bowel ischemia is very nonspecific: early signs and symptoms include bloating, crampy pain, and loss of bowel sounds. Late in the course, massive abdominal distention, paralytic ileus, pneumonia intestinalis, and transmural bowel necrosis may develop; the progression of the clinical course mimics septic shock with possible progression to multi-system organ failure. Findings in plain films or computed tomographs of the abdomen are nonspecific: pneumonia intestinalis (88%), free fluid (38%), thickened/dilated loops of bowel (38%), or free peritoneal air (25%).

Most cases reported in the literature occurred in young patients (Table) where full-dose nutrition was tolerated for an average of 8 days prior to the development of bowel ischemia. In our case, the clinical picture developed more rapidly. With the data available from published reports, it is not possible to identify risk factors suggestive of diminished mesenteric blood flow that could be used to identify patients who are not candidates for enteral feeding or that would reliably diagnose this entity before bowel necrosis had occurred. The occurrence of abdominal pain and distension, high nasogastric output, and signs of intestinal ileus should result in immediate discontinuation of tube feeding rather than repeated attempts to alter the formula and feeding schedules. In the presence of worsening clinical parameters, early surgical exploration should be considered. Unfortunately, many patients who develop intestinal necrosis associated with enteral feeding are diagnosed late in their course, with resulting significant morbidity. Conservative therapy in these cases of bowel compromise increases morbidity and mortality. An early exploratory laparotomy with resection of all involved segments of the bowel is the only chance of cure.

CONCLUSIONS

Enteral nutrition through a feeding jejunostomy offers several advantages compared with total parenteral nutrition for the postoperative patient, and its benefits usually outweigh the likelihood of severe complications. Several authors have reported the association of small bowel ischemia and necrosis with catheter jejunostomy. Early signs of this syndrome are very nonspecific and clinical factors for identifying patients at risk are lacking. Distension is a nonspecific but ominous finding and should prompt discontinuation of tube feeding and close monitoring. A worsening general condition or sepsis mandates early operative intervention with resection of ischemic bowel as the only way to decrease morbidity and mortality.

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REFERENCES