Non-occlusive small bowel necrosis during gastric tube feeding: a case report

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Abstract Small bowel necrosis is known as a rare, but serious complication of jejunal tube feeding. We report a case of non-occlusive small bowel necrosis with gastric tube feeding. The patient had a moderate multiple trauma with head injury. Abdominal distension developed after several days of uneventful nasogastric tube feeding. At laparotomy patchy necrosis of the small bowel was found without signs of bowel obstruction or impaired mesenteric perfusion. The patient recovered after a prolonged ICU stay. In this case the large doses of clonidine, given due to an alcohol withdrawal syndrome, were suspected to be a major contributing factor to the development of the small bowel necrosis by impairing gut motility and mucosal perfusion. We conclude that, first, small bowel necrosis can occur after primarily uneventful enteral feeding, even with gastric feeding; second, clonidine can dramatically impair gastrointestinal function in critically ill patients by impairing gut motility and mucosal perfusion.

Keywords Small bowel necrosis · Gastrointestinal tube feeding · Gastric tube feeding · Clonidine · Case report

Introduction

Currently, early enteral feeding is advocated in intensive care patients in order to maintain the structural and functional integrity of the gut, and its potential beneficial effect on host defence [1, 2, 3]. However, the numerous potential advantages of enteral nutrition are not definitively proven [3]. Enteral nutrition itself can cause morbidity. Up to more than one half of the critically ill patients receiving enteral nutrition show signs of poor tolerance of the enteral feeding. The more common problems are functional, such as abdominal distension, diarrhoea, regurgitation or vomiting [4]. Small bowel necrosis is a rare (0–0.5 %) but serious complication of enteral feeding, with a high mortality (50–100 %) [5, 6]. The reports in the literature are all with jejunal feeding. We present a case of similar small bowel necrosis during gastric feeding.

Case report

A 61-year-old male was severely injured in a motorcycle accident. He had an initial Glasgow Coma Scale of 15, despite a frontal cerebral contusion. Other injuries included facial fractures and stable fractures of the thoracic and cervical spine without neurological deficits. After osteosynthesis of the craniofacial fractures, with major blood loss, but without a prolonged phase of hypotension or the use of catecholamines, he was transferred to the ICU with a tracheostomy and an intermaxillary fixation. Due to being nervous and disoriented he was sedated with propofol up to 300 mg/h. There were no cardiopulmonary problems. Enteral feeding was started on the first postoperative day (the second day after the trauma) per nasogastric tube with a standard formulation [Nutrodrip, (now Isosource)]. The initial rate was 28 ml/h. Including a 6-h pause during the night, this corresponded to 500 ml/day containing 500 kcal/day. The daily rate was increased by 500 ml each day, so that the goal of 2,000 ml/day was reached within 4 days. From the beginning metoclopramide 10 mg iv was given every 6 h. On the third postoperative day the patient remained disorientated. Due to a history of alcohol abuse and suspicion of alcohol withdrawal clonidine up to 150 μg/h was added for sedation. A second
CT-scan confirmed the frontobasal contusion without further injuries of the brain.

On the 6th postoperative day the abdomen was distended and meteoristic, but without signs of peritonitis. The plain X-ray of the abdomen showed a dilated small bowel (3 cm). There was no free air in the peritoneum. Enteral feeding was stopped. The next day distension and meteorism were more pronounced without any signs of peritonitis. The plain X-ray (Fig. 1) and the CT-scan (Fig. 2) of the abdomen showed a localised pneumatosis intestinalis in proximal parts of the small bowel. No vascular mesenteric lesions were seen in the angiographic CT-scan. Overnight the patient became hemodynamically unstable with metabolic acidosis and multiple organ dysfunction. Emergency laparotomy was performed. It revealed peritonitis of all four quadrants and patchy necrosis with several perforations of the proximal small bowel (50 cm below the ligament of Treitz). There was no obstruction of the gut or mesenteric vascular damage. Part of the small bowel (1.2 m) was resected, the remaining anastomosed end-to-end.

The postoperative course was complicated by an acute renal failure requiring haemodialysis during 11 days and by a critical illness neuropathy. The patient recovered after a 6-week ICU stay. The neurologic course was still dominated by disorientation and complicated by the critical illness neuropathy. Enteral feeding was successfully installed via nasojugal tube, 14 days after laparotomy. Two months after his accident the patient was transferred to a regular ward awaiting his neurological rehabilitation in a specialized clinic one week later.

**Fig. 1** Plain X-ray of the abdomen on day 7 showing the distension of small bowel. The *arrows* mark the double contour of the abdominal wall caused by the pneumatosis intestinalis

**Fig. 2** CT-Scan of the abdomen on day 7 showing intestinal distension. The *arrows* mark air bubbles within the intestinal wall indicating pneumatosis intestinalis

**Discussion**

Non-occlusive small bowel necrosis is a rare, but serious, and now well-known complication of enteral tube feeding after major abdominal surgery or trauma [5, 6]. To our knowledge, the present case is the first one demonstrating small bowel necrosis during gastric feeding. Several features are remarkable: first, the non-occlusive small bowel necrosis developed during gastric feeding; second, despite diffuse peritonitis at laparotomy the clinical presentation was surprisingly subtle, possibly due to the deep sedation; third, the α₂-agonist clonidine, given for suspected alcohol withdrawal, in addition to standard sedation, may have contributed to the intestinal distension and subsequent necrosis. It seems likely that the multiple perforations of the gut were associated with the bowel distension and impaired perfusion without a vascular occlusion, and that the peritonitis was secondary to the delayed surgical intervention.

Many clinical and pathological findings of non-occlusive bowel necrosis are similar to the necrotizing enterocolitis of premature newborns. Three main components are discussed in the pathogenesis [5]: metabolic stress of the nutrient-absorbing enterocytes by increased energy demand and dysmotility by gut wall distension may lead to microvascular ischemia. Bacterial overgrowth may cause mucosal injury and local inflammation by intraluminal toxins of bacterial nutrient fermentation.