

SCIENTIFIC LETTER

Intestinal necrosis associated with enteral nutrition by jejunostomy*



Necrosis intestinal asociada a la nutrición enteral por yeyunostomía

Enteral nutrition (EN) should be considered as the option of choice in patients after surgery of the upper digestive or pancreatic tract, either via a nasojejun tube (NJT) or through surgical jejunostomy.¹ Despite its advantages (diminished stress response, improved immunity and wound healing, fewer septic complications), EN may not be tolerated by all patients, since it is associated with mild gastrointestinal discomfort and occasionally severe complications – particularly non-occlusive mesenteric ischemia (NOMI) – causing intestinal necrosis associated with EN,² a rare but potentially fatal complication (41–100%) of EN. At our center, jejunostomy placement for EN after major surgeries of the upper digestive tract is a common practice. We present a case in which a differential diagnosis of necrosis associated with EN was considered, with a review of the literature.

A 64-year-old male smoker with type 2 diabetes mellitus (T2DM) and chronic obstructive pulmonary disease (COPD) was diagnosed with ulcerated gastric adenocarcinoma in November 2018, in the context of admission due to upper gastrointestinal bleeding. The study of disease spread found no metastases. The patient was receiving treatment with Spiriva (tiotropium bromide) every 24 h, metformin 850 mg every 12 h and omeprazole 20 mg every 24 h.

Elective admission to general and gastrointestinal surgery was decided upon for total gastrectomy, with Roux-en-Y end-to-side esophagojejunal anastomosis 70 cm from the foot of the loop, and a jejunostomy catheter was placed for EN. The pathology report indicated moderately differentiated tubulopapillary adenocarcinoma infiltrating to the submucosa, with disease-free surgical margins.

With regard to the indication of postoperative nutritional support, the patient reported a variable regular body weight of 75–80 kg, no weight loss prior to admission, height 1.70 m, and a body mass index: 26.7 kg/m². Caloric requirements: 2130 kcal/day. Protein: 85.2 g. Nitrogen: 13.6 g.

Biochemistry: glucose 149 mg/dl (normal range 74–106), HbA1c 7.3%, creatinine 0.57 mg/dl (0.7–1.2), cholesterol

121 mg/dl (130–200), triglycerides 51 mg/dl (60–150), albumin 3.66 mg/dl (3.5–5.2), prealbumin 16.6 mg/dl (20–40), C-reactive protein (CRP) 43.2 (<5 mg/dl), ions and liver function profile within normal ranges, leukocytes 12,430/μl (4000–11,000), hemoglobin 11.7 g/dl (13–17.5), lymphocytes 1740/μl (1000–4500).

On the second postoperative day, tolerance was started with water at a rate of 21 ml/h via jejunostomy over 6 h, followed by a normocaloric (NC) and normoprotein (NP) EN formulation specific for diabetes mellitus, with low osmolarity (Nutrison Advanced Dison®: 300 mOsm/L, dietary fiber 3% total calorie value [TCV], 80% soluble and 20% insoluble) in continuous perfusion at 21 ml/h over 8 h. Tolerance was acceptable, allowing elevation of the rate to 42 ml/h over the following 8 h.

On the third postoperative day, with EN at 63 ml/h, the patient suffered clinical worsening, with the appearance of atrial fibrillation (AF) (reverted with amiodarone), abdominal bloating and pain. The abdominal CAT scan revealed pneumoperitoneum and oral contrast extravasation secondary to suture dehiscence at the esophagojejunal junction, with extensive intestinal pneumatosis and associated portomesenteric gas accumulation. Emergency surgery was decided upon, revealing dehiscence of the esophagojejunal anastomosis. No exact leakage site could be identified, due to difficulties in accessing the intrathoracic component. Necrosis of approximately one and a half meters of small intestine was observed, starting about 30 cm from the foot of the loop distally, practically at the point of insertion of the jejunostomy, with distal patches of ischemic appearance. The rest of the intestine was of normal appearance, though dilated. Resection of the one and a half meters of ischemic jejunum was performed, with jejunostomy in the left flank region and ileostomy (mucosal fistula) in the right iliac fossa, in addition to washing of the abdominal cavity and removal of the previous jejunostomy. The patient was admitted to the intensive care unit with signs of severe sepsis. Despite intensive support, the patient developed multiorgan failure refractory to treatment; life support was adapted accordingly, and death occurred on the fifth postoperative day. The family did not grant permission for post-mortem study.

The incidence of EN-related intestinal necrosis caused by non-occlusive mesenteric ischemia (NOMI) ranges from 1.2 to 1.7%.³ The available data correspond to retrospective case series in which risk factors for mesenteric ischemia, such as arterial hypertension, atrial fibrillation, smoking, hyperlipidemia, peripheral arterial disease and diabetes mellitus, are usually associated.⁴ Some of the factors were also present in our patient. The underlying pathogenesis may be explained by a non-occlusive reduction of arterial blood flow, most often due to primary splan-

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nic vasoconstriction.⁵ However, the association with EN is unclear, and appears to be multifactorial. It is assumed that the absorption of intraluminal nutrients increases the energy demands in metabolically stressed enterocytes, exposing the intestine to ischemic risk in hypoperfused patients. Other factors such as the use of high osmolarity formulas (>700 mOsm)⁶ or high fiber formulas may predispose to decreased gastrointestinal tract perfusion, as well as to motility disorders.⁷

In the series published by Al-Taani et al.,⁸ this complication only occurred in patients undergoing total gastrectomy, which poses a greater risk than esophagectomy.

The clinical presentation is very nonspecific, and there are no risk factors that can identify patients amenable to EN or clinical indicators allowing for an early diagnosis. The occurrence of abdominal pain and bloating or signs of ileus should cause us to suspect this condition and suspend EN, instead of making repeated changes in the EN formula or in the timing of administration⁹.

Exploratory laparotomy reveals intestinal ischemia starting at the site of the jejunostomy tube and extending distally to a variable distance with histopathological findings of distal transmural necrosis. There is no agreement on the optimum management of early EN via jejunostomy in the postoperative period with a view to eliminating the risk of associated necrosis, though close monitoring is required.¹⁰ Despite this complication, the use of EN after major abdominal surgery should not be questioned in seeking to reduce the risk. Rather, it is advisable to administer low osmolarity, no fiber EN formulas with a low initial infusion rate followed by gradual elevation until optimum nutrition is achieved. The above-mentioned complication should be considered early in cases of intolerance, in order to avoid fatal outcomes.

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Conflicts of interest

The authors state that they have no conflicts of interest.

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