CASE REPORT

Black esophagus (acute esophageal necrosis) after spinal anesthesia

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Abstract Acute esophageic necrosis or black esophagus is an uncommon clinical entity that owes its name to the endoscopic view of the necrotic esophageal mucosa. It is always related with a critical medical condition and usually has an ischemic etiology. We report the first case of acute esophageal necrosis after a spinal anesthetic for partial hip joint arthroplasty. We discuss the underlying pathophysiological mechanisms.

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PALABRAS CLAVE
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Hemorragia masiva

Esófago negro (necrosis esofágica aguda) tras anestesia subaracnoidea

Resumen La necrosis esofágica aguda o esófago negro es una entidad clínica infrecuente que debe su nombre al aspecto necrótico de la mucosa esofágica observado durante una endoscopia digestiva alta. Se relaciona siempre con estados clínicos de gravedad y su etiología es habitualmente isquémica. Presentamos el primer caso de necrosis esofágica aguda tras anestesia subaracnoidea para la realización de una artroplastia parcial de cadera. Se discuten los mecanismos fisiopatológicos subyacentes.

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Introduction

Acute esophageal necrosis (AEN) or black esophagus is an uncommon clinical entity first described by Goldenberg in 1990. It owes its name to the endoscopic view of the necrotic mucosa. It may affect the esophagus from the cricopharyngeal crease to the gastroesophageal union, which is usually

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preserved. The differential diagnosis should include malignant melanoma, pseudomelanosis and acantosis nigricans. In addition, caustic ingestion must be ruled out because the endoscopic view of the injury may be undistinguishable. In some instances the cause of AEN remains unclear, although it is usually related to a critical medical condition. We report the first case of AEN after a spinal anesthetic for partial hip joint arthroplasty. We discuss the underlying pathophysiological mechanisms.

Case report

We present the case of an 87-year-old woman (75 kg, 152 cm, BMI 32.5 kg/m²) sustaining arterial hypertension and permanent atrial fibrillation (AF). No gastrointestinal bleeding was reported. Her chronic medications included acenocoumarol, diltiazem and occasional use of nonsteroidal antiinflammatory drugs (NSAIDs). She was admitted to the hospital because of a subcapital left hip fracture. Arterial pressure at admission was 130/70 mmHg and heart rate was 90 beats/min. Surgery was scheduled 48 h after admission.

In the operating room blood pressure was 140/70 mmHg and arrhythmic heart rate (AF) of 90 bpm. Preoperative blood test showed 30% hematocrit. INR was 1.4. Monitoring consisted in pulse oximetry, electrocardiogram and noninvasive blood pressure. After premedication with 1 mg of midazolam, we performed a spinal anesthetic with the patient in the right lateral decubitus at L4–L5 level with 0.5% hypobaric bupivacaine 9 mg and fentanyl 10 mcg, with a 25 G Whitacre needle. The patient was replaced supine and then, she suffered arterial hypotension and became unconscious. We tried to ventilate her but it was not possible so tracheal intubation was performed. Immediately afterwards, the patient suffered a massive upper gastrointestinal bleeding. Arterial hypotension worsened (blood pressure of 50/30 mmHg, heart rate 120 bpm). An infusion of intravascular crystalloids (1000 ml) and colloids (500 ml) was started together with norepinephrine. Blood test showed hemoglobin 5 g/dl, hematocrit 15.7%, 133,000/µl platelet count, INR 2.48, APTT 79 s, pH 7.04, pO₂ 472 mm Hg, pCO₂ 27 mm Hg, HNaCO₃ 7 mmHg, base excess −23. The patient was transferred to the Post-Anesthesia Care Unit (PACU), and 4 packed red blood cells (PRBC) units and 1600 ml fresh frozen plasma (FFP) were administered. After hemodynamic stabilization was achieved, an endoscopic exploration was performed. It revealed an esophagus with blackish mucosa and ulcerated areas with active bleeding, and AEN was diagnosed. The endoscopic study also showed ulcers in the antrum, bulb and second portion of duodenum with fibrin and absence of active bleeding. Treatment included pantoprazol 8 mg/h IV, digestive rest and parenteral nutrition.

A second endoscopic exploration 48 h afterwards demonstrated a significant improvement of the lesions, with visible signs of mucosa regeneration among the areas of esophageal necrosis. Biopsies were taken to confirm the diagnosis.

The patient's clinical condition improved progressively without rebleeding, and she underwent hemiarthroplasty under general anesthesia at the 5th day after bleeding. No intraoperative or postoperative complications occurred. The patient died ten days later due to upper gastrointestinal bleeding and secondary hypovolemic shock.

Discussion

A literature review was performed using Medline and afterwards relevant references from the published literature were retrieved. One hundred and twelve cases were identified, and upper gastrointestinal bleeding was the commonest presenting feature. The majority of cases were male patients and the mean age at presentation was 68.4 years. This review of the literature shows a mortality rate of 38%.

Black esophagus has an incidence ranging between 0.008% and 0.2% in endoscopies performed in long series of patients. According to Yasuda et al. the incidence may reach 6% of the patients with upper gastrointestinal bleeding, so it might be more common than suggested. In fact, Jacobsen et al. report an incidence of esophageal necrosis of 10.3% in a consecutive hospital autopsy specimens during one year.

The etiology of AEN is unclear but it is probably multifactorial since in all the described cases several factors appeared involved. Many studies have reported the possibility of ischemic etiology but the esophagus is a relatively well vascularized organ: the cervical esophagus by the superior and inferior thyroid arteries bilaterally, the intrathoracic esophagus by arteries arising from the aorta, and the abdominal esophagus by branches of the left gastric and splenic arteries. The anastomotic network protects esophagus from a significant localized ischemic injury. However, the association of esophageal damage during hypovolemia and the improvement after hemodynamic stabilization together with the fact that the distal esophagus, the less vascularized area, is the more often involved in AEN supports this explanation. A case of AEN caused by anticardiolipin antibodies was described where transmural esophageal necrosis was not limited to the mucosa and submucosa, a typical feature of AEN; anticardiolipin antibodies being associated with thromboembolic induced ischemia. These antibodies were negative in our patient.

Esophagitis due to Candida, herpes virus I, cytomegalovirus and Lactobacillus acidophilus is suspected in some cases.

The combination of transient ileus, large amounts of gastric acid secretions and gastroesophageal reflux (perhaps worsened by NSAID intake in some previous reports) may play a relevant role in the onset of AEN.

Hypothermia, diabetic ketoacidosis, hyperglycemic hyperosmolar coma, aortic dissection and hypersensitivity to antibiotics, especially ampicillin, gentamycin and cefoxitin, have also been evoked as precipitating causes of AEN. In our case, the relationship between arterial hypotension and AEN is supported by previously reported cases of femur fracture patients suffering ischemic esophagitis.

To our knowledge there is only one case reported in anesthesia journals. The case being of a patient who underwent abdominal lymphoma resection and suffered multiorgan failure. The upper endoscopy performed due to acute anemia revealed AEN.

Reported risk factors include age, male sex, cardiovascular disease, hemodynamic compromise, gastric outlet obstruction, alcohol ingestion, malnutrition, diabetes, renal insufficiency, hypoxemia, hypercoagulable state, and trauma. The damage is probably of multifactorial origin, but ischemia is the final mechanism. Overall, acute esophageal
necrosis should be viewed as a poor prognostic factor, associated with high mortality.

In the case described, several predisposing factors (advanced age, occasional use of NSAIDs, immobilization) and a precipitating cause (arterial hypotension) may explain AEN. The incidence of arterial hypotension after spinal anesthesia ranges from 15% to 33%. According to Hartmann et al., our patient fulfilled many of the variables associated with arterial hypotension, as high BMI (32.5 kg/m²), arterial hypertension, urgent surgery and a possible high sensory block (the need for orotracheal intubation made impossible to evaluate this issue). Furthermore, our patient was treated with diltiazem and chronic long-term antihypertensive therapy was identified as having an association with a hypotension.

The most common presenting symptom in the awake patient (that was not the case) is usually upper gastrointestinal bleeding, followed by nausea and vomiting, epigastric pain, dysphagia and fever.

Treatment is supportive, including hemodynamic stabilization, proton pump inhibitors at high doses and short-term parenteral nutrition with bowel rest for at least 48 h. There is no consensus in the literature regarding the need for broad-spectrum antibiotic therapy. Some clinical situations that may make it advisable are perforation, rapid clinical deterioration, sepsis or immunological compromise. Nevertheless, prophylactic use of empiric antibiotics in sterile necrosis is generally not warranted. Nasogastric tube insertion should be avoided or guided through endoscopic techniques. Surgical treatment might be necessary whenever transmural necrosis appears or in those cases with esophageal perforation (esophagectomy).

The diagnosis can be made on an endoscopic basis. The pathological study shows necrosis of mucosa and submucosa with an inflammatory infiltration, partial destruction of muscle fibers and occasionally thrombosed vessels. The presence of acute necroinflammatory infiltrate and the failure to detect any sign of infection by fungus or bacterial agents in the biopsy supported the clinical diagnosis of AEN in our patient.

Possible complications include esophageal stenosis (10–15%), perforation (7–10%), recurrence of AEN, mediastinitis and abscesses (6%). The prognosis is dependent on the patient's age and comorbidities rather than solely the severity of esophageal injury. Mortality remains high (33–55%).

In conclusion, black esophagus is an unusual disease, whose pathophysiology remains unclear. We should be aware of its existence.

Conflict of interest

The authors declare no conflict of interest.

References