Comentario a la nota clínica: Esofagitis necrotizante aguda en paciente inestable

Comment on the clinical note: Acute necrotizing esophagitis in an unstable patient

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Rodrigo M et al described a case of black esophagus (BE) in a 77-year-old male who developed severe hemodynamic instability, and BE was cited as rare condition[1]. The mentioned report is very interesting and well documented, but additional comments and illustrations from the study of a brazilian patient with coexistent BE and candidiasis could be appropriate to emphasize concerns about frequency rate and associated factors.

Although ischemic factors can play a pathogenic role, hemodynamically stable patients have presented with BE too. Worth of note, only about a hundred cases of this entity were reported since the first description in 1990[1-4]; notwithstanding, published necropsy data are suggestive that BE may be under diagnosed and/or under reported[2,5]. The aim of the present paper is to enhance the suspicion index about BE, which will have better outcome if early diagnosis and precocious treatment can be accomplished.

An 83-year-old male was admitted because of hypoglycemia, vomiting, dysphagia, and weight loss. He was in use of NPH insulin and furosemide to treat diabetes and hypertension and there was antecedent of an advanced invasive rectal cancer. The endoscopy study detected mild stricture, ulceration and black discoloration in distal esophagus (Fig. 1A), in addition to duodenal ulcers. Esophageal biopsy samples showed characteristic features of acute esophageal necrosis and ulcerated esophagitis (Fig. 1C), coexistent with local accentuated moniliasis (Fig. 1D). Rectosigmoidoscopy revealed an ulcerated infiltrative rectal tumor at 3cm from the anal limit, affecting ¾ of the lumen. The diagnosis of rectal carcinoma was confirmed by biopsy (Fig. 1D). TC showed perirectal invasion and liver implants. Remarkable laboratory data were hypoalbuminemia, accentuated anemia and leukocytosis with neutrophilia, in addition to hyponatremia and high platelet count[1-4]. These changes can be found in acute phase response (APR) and protein-energy malnutrition (PEM)[6,7]. He received gastric protection, total parenteral nutrition and intravenous fluconazole. Even with clinical improvement, the patient refused treatment for advanced cancer. After gastrostomy done because of esophageal stenosis, he was referred to home-care support.

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In spite of the malignant co morbidity and indicative features of APR and PEM, there was no circulatory instability, and the clinical evolution was favorable. Wallberg et al. reported similar favorable outcome of BE in a 75-old-year patient with sepsis[4], which is a major cause of systemic inflammatory response and circulatory failure. Common clinical features are dyspepsia, epigastralgia, hematemesis, melena, and vomiting, but the diagnosis suspicion of BE during endoscopy must be further confirmed by histological data. Circumferential black discoloration is often seen in distal esophagus, with mucosa erosions, ulcers, exudates, necrosis and vessel thrombi[1-4]. Differential diagnosis of BE includes acanthosis nigricans, melanoma, melanosis, pseudomelanosis, ocronosis and necrotic lesions due to caustic or corrosive agents[1-4].

Risk factors for BE include alcoholism, cardio circulatory disorders, pulmonary diseases, cirrhosis, diabetes, esophagitis, gastric obstruction, hiatal hernia, malignancies, malnutrition, and renal failure[1-4]. Interestingly, esophageal candidiasis or rectal cancer are associations very rarely reported. Malignancies found in patients with BE are: cancers from colon, biliary tract, hypofarynx, lung, ovary, pancreas, prostate, and rabdomyosarcoma. Esophageal stricture is the major complication[2,4].

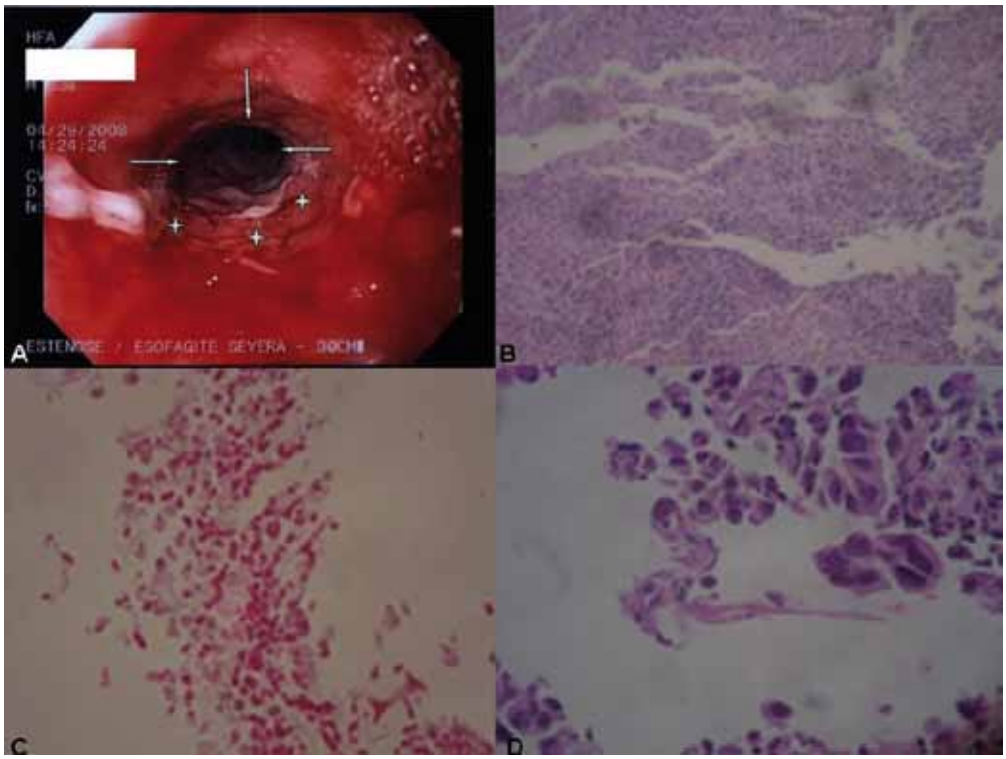

Figure 1A. Accentuated acute esophagitis, with fibrinous exudates, enanthema, edema and friable mucosa (stars); in addition to stenosis and circumferential black discoloration in the distal portion of esophagus (arrows).

Figure 1B. Acute necrotizing esophagitis, showing fibrin and necrotic material in the ulcerated lesion. Necrotic changes were limited to mucosa and submucosa (HE 100x).

Figure 1C. Esophageal candidiasis characterized by numerous spores of *Candida sp* (HE PAS 600x).

Figure 1D. Cells of the rectal infiltrative carcinoma showing cytoplasmic vacuolization (HE 600x).

Coexistent BE and candidiasis should be better cleared because: reports about this association are rare[5,8,9]; endoscopy disclose esophageal candidiasis in 1-8% of all patients examined[10]; fungi may be opportunistic agents in immunocompromised individuals[8-10], including those with APR and PEM associated with cancer or AIDS[5,8,9]. As occurred in the present case, the esophageal changes may undergo favorable resolution after management with proton pump inhibitors, sucralfate, and nutrition care, in addition to treatment of associated infections (Table 1).

Table 1. Comparative data from five patients with black esophagus associated with esophagitis by fungi.

Authors/ year	Patients	Main risk factors	Fungal agents associated with black esophagus	Fluconazole	Outcome
Hoffman et al, 1999	30y, male	AIDS	*Penicillium chrysogenum*	No (Ket + Nys)	†
Ben Soussan, 2002	68y, male	Prostate cancer	«Minor mycotic infection»	No	†
Ferreira et al, 2007	52y, male	Rabdomyosarcoma	*Candida albicans, Aspergillus, and Actinomyces*	Yes	†
Kim and Choi, 2007	34y, male	Ketoacidosis, DM	*Candida*	Yes	Favorable
Santos et al, 2009	83y, male	Rectal cancer, DM	*Candida sp*	Yes	Favorable

DM: diabetes mellitus; Ket: ketoconazole; Nys: nystatin; †: death.

As necropsy data have suggested that BE can be under diagnosed, case reports and letters to the editor could increase the awareness about this disorder.

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