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Black Œsophagus as a Possible Complication of Cholangitis

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Abstract

Acute esophageal necrosis (AEN), also known as black oesophagus or necrotizing esophagitis is a very rare clinical disorder characterized by a striking endoscopic image of diffuse, circumferential, black-appearing in the distal esophagus that stops abruptly at the gastroesophageal junction on esophagogastroduodenoscopy, its etiology is likely unclear and multifactorial, Upper gastrointestinal bleeding is The most common presentation, the incidence reported is only 0.2%, the prognostic is poor and more the cases are fatal by comorbidities, We present a new case of acute oesophageal necrosis as a possible complication of cholangitis.

Keywords: Black œsophagus, Complication Cholangitis.

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INTRODUCTION

Acute esophageal necrosis (AEN), also known as black oesophagus or necrotizing esophagitis is a very rare clinical disorder characterized by a striking endoscopic image of diffuse, circumferential, blackappearing in the distal esophagus that stops abruptly at the gastroesophageal junction on esophagogastroduodenoscopy [1], its first described by Goldenburg and *al.* in 1990 [2] and later Gurvits classified AEN as a distinct syndrome in 2007, the incidence reported is only 0.2% [10].

its etiology is likely unclear and Various kinds of diseases are known to be associated with AEN. They include diabetes mellitus, malignancy, hypertension, chronic pulmonary disease, alcohol abuse, and trauma [3]. The physiopathology is multifactorial and most investigators suggested a combination of various mechanisms including ischemia, impaired mucosal barrier systems, and a backflow injury from gastric contents.

Upper gastrointestinal bleeding is the most common presentation including hematemesis, coffeegrounds emesis, melena, and blood loss anemia [5].

Complications may include stenosis or stricture formation in the distal esophagus, perforation, mediastinitis, and death. Overall mortality is largely related to the underlying medical condition and approaches 32%[1].

We present a new case of acute oesophageal necrosis as a possible complication of cholangitis

CASE REPORT

A 59 years old man, without a past medical history, complained of epigastralgia for 5 months, At hospital he started to vomit and he had fever, admission blood pressure was 120/60physical, his pulse was regular with an apical rate of 104 beats/min, temperature 38.8° C examination epigastric sensitivity, his laboratory findings showed an increase in his inflammatory markers (CRP at 127 an white cells at 13000), A lipase test mesure was normal, Elevated cholestasis parameters, blood glucose of 1.2 g/dl, an increased level of serum creatinine (49,4 g/l) and urea (1.7g/l) with hyponatremia(122mmol/l), CT scan, performed to rule out a mass of 47mm in the head of the pancreas with dilatation of the common bile duct and the main pancreatic duct, for suspected cholangitis due to bilary obstruction by the pancreatic tumour, patient Intravenous was given antibiotics, analgesia, IV proton inhibitor and fluids with antiemetics, restriction of oral intake.

One day after his admission, he developed coffee ground emesis, His hemoglobin dropped from 12.5 to 7.6 gm/dl, and emergent esophagogastroduodenoscopy (EGD) was done which revealed diffusely circumferential black pigmentation of the oesophageal mucosa above the gastroesophageal

junction with ulcerations of the distal thirds of the esophagus, biopsies were not taken, the patient died 2 days after the diagnosis.

DISCUSSION

Acute esophageal necrosis (AEN), also known as black oesophagus or necrotizing esophagitis is a very rare clinical disorder, First described by Goldenburg *et al.* in 1990 [2] and later Gurvits classified AEN as a distinct syndrome in 2007, the incidence is low, a 1 year prospective study of EDG have estimated the incidence at 0.2% [10], Gurvits in his review study have reported A total of 88 patients in the literature during the 40 years from 1965 to 2007, it has a male predominance(81%) with an average age of 67 years [4].

Its etiology is likely unclear and various kinds of diseases are known to be associated with AEN. They include diabetes mellitus, malignancy, hypertension, chronic pulmonary disease, alcohol abuse, trauma, systemic inflammatory response syndrome due to severe pancreatitis [3], and infections such as cytomegalovirus, Candida albicans and Klebsiellapneumoniae [11] have also been, Nasogastric intubation and endoscopic instrumentation with detergent use have been implicated as possible inciting events for the development of AEN [4]The physiopathology is multifactorial and most investigators suggested a combination of various mechanisms including ischemia, impaired mucosal barrier systems, and a backflow injury from gastric contents.

Most commonly reported reasons for admission were upper GI bleeding; other symptoms include abdominal pain, dysphagia, vomiting, epigastric pain, chest pain and a cardiovascular event or shock including tachycardia and hypotension [4].

AEN can be diagnosed endoscopically and is usually confirmed histologically; A biopsy is usually recommended but not required for a diagnosis of AEN.

The classic endoscpic appearance is a Circumferentially black appearing esophageal mucosa that stops abruptly at the gastroesophageal junction [4], other endoscopic findings may include gastric ulceration, dilated fluid-filled stomach, volvulus, gastric outlet obstruction and duodenitis or pyloric stenosis [12, 4].

There is no specific treatment, Development of AEN carries a generally poor prognosis and the goal of therapy should be directed at treating the coexisting medical diseases.

The majority of authors support a convervating approach consisting on intravenous hydratation, blood cell transfusion, Intravebous proton pump inhibitor, supportive parenteral alimentation and correcting the underlying disease, nasogastric tube inserstion is contraindicated due to risk of perforation [4, 1].

The prognosis of AEN is poor, mortality is high (31.8%) and is probably related to the associated conditions and secondary to complications as esophageal perforation and mediastinal infection [4].

After our literature analysis we did not find any case of AEN associated with cholangitits which make our case unique

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