H109 Black Esophagus and Fatal Cocaine Intoxication: An Unusual Combination

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Learning Overview: The goal of this presentation is to highlight the case of a 68-year-old Caucasian male found dead at home with a massive hematemesis. Autopsy revealed the presence of acute esophageal necrosis and toxicological investigations identified the cause of death in acute cocaine intoxication.

Impact on the Forensic Science Community: This presentation will impact the forensic science community by describing Acute Esophageal Necrosis (AON), also known as "black esophagus," as a rare and potentially lethal complication of cocaine abuse.

Introduction: AON, also referred to as acute necrotizing esophagitis and "black esophagus," is a pathological condition characterized, at endoscopic examination, by a diffuse full-circumference black appearance of the esophageal mucosa, which usually affects the distal portion of the organ and abruptly terminates at the gastroesophageal junction. It is a rare disease, with a prevalence found up to 0.2% of cases in some autopsy studies. During endoscopic investigations, its prevalence varies from 0.001% to 0.2% of cases. The etiology of AON is multifactorial, probably linked to a simultaneous presence of tissue hypoperfusion due to impaired circulation and low-flow states, alteration of mucosal protective factors and repair mechanisms in the presence of debilitation and malnutrition, and topical chemical lesions resulting from reflux of gastric acids due to conditions of esophagus-gastroparesis. The patients usually present with signs of upper gastrointestinal bleeding, such as hematemesis and melena. Other symptoms such as epigastric pain, nausea, vomiting, dysphagia, chest pain, and syncope may also be present. Multiple associated comorbidities are commonly found, the main ones being hypertension, diabetes, dyslipidemia, and widespread vascular atherosclerosis. Findings on physical examination are usually related to an underlying medical condition. Tachycardia, hypotension, shock, fever, cachexia, hypoxia, and abdominal pain are some of the signs that may be present, but none of these are specific for esophageal necrosis. Diagnosis is made by endoscopy. A biopsy is also needed to rule out infections and other causes of esophageal necrosis. Clinical management classically involves treatment of the underlying disease, intravenous fluid administration, concentrated red blood cell transfusions, absolute fasting, proton pump inhibitors, and sucralfate suspension. The evolution of the clinical picture largely depends on the severity of the underlying disease.

The possible complications of "black esophagus" include perforation of the wall with consequent mediastinal infection, narrowing or stenosis of the organ lumen, and duodenal ulcers. The mortality rate can reach up to 32% in the presence of severe comorbidities.

Case Presentation: A 68-year-old man at home was found in cardiac arrest with profuse hemoptysis. The man had a previous history of heart disease with pacemaker placement, diabetes mellitus treated with insulin, and renal failure undergoing dialysis treatment. The death was confirmed onsite by the medical staff. An autopsy was requested to clarify the cause of death.

Results and Discussion: The autopsy revealed a blackish discoloration of the esophageal mucosa at the mid-distal segment, up to the gastro-esophageal junction, with a typical "black esophagus" appearance. The presence of slight thickening of the coronary wall with diffuse atheromatous plaques was also observed, without significant stenosis of the lumens. Histological examination revealed the presence of contraction band necrosis of myocardial tissue, extensive areas of myocardiosclerosis and foci of chronic inflammation of the pericardial adipose tissue. The esophageal mucosa was extensively de-epithelialized, with foci of chronic inflammation. Immunohistochemistry on the esophageal samples (CD3, CD20, BCL2, BCL6, CD10, Ki-67, CKAE1/AE3) was performed. The toxicological screening test carried out on cadaveric blood was positive for the presence of benzoylecgonine (the main inactive metabolite of cocaine), showing a quantity of 530ng/ml, and negative for other substances of abuse. The data was then confirmed by qualitative-quantitative analysis performed with gas chromatography/mass spectrometry. The cause of death was therefore attributed to cardiogenic shock following acute cocaine intake and gastrointestinal hemorrhage due to acute esophageal necrosis.

Conclusion: In the literature, there are very few cases of cocaine abuse associated with the onset of acute esophageal necrosis and gastrointestinal bleeding. In this regard, the case presented is of considerable interest as it supports the hypothesis that the intake of cocaine, causing vasoconstriction, can further compromise the esophageal blood circulation and trigger massive necrosis of the mucosa, especially in predisposed subjects.

Black Esophagus, Cocaine, Intoxication

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