CLINICAL NOTE

Bruising of the esophagus as a cause of gastrointestinal bleeding in a case of heatstroke

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ABSTRACT

Alterations in blood clotting are a frequent complication of serious heatstroke and may result in gastrointestinal bleeding. We report the case of a 26-year-old man who was admitted to our hospital with symptoms of hyperthermia associated with encephalopathy and disseminated intravascular coagulation (DIC) after prolonged exposure to sunlight. He presented hematemesis, after which he was diagnosed with having a bruising of the esophagus that took up the upper and lower thirds, there being no other lesions in the stomach or duodenum. After supportive treatment and following the resolution of the underlying pathology, the endoscopy-revealed injuries healed with a complete normalization of the esophageal mucosa. Esophageal submucosal bruising is an exceptional cause of hematemesis in serious heatstroke not previously described in the literature.

Key words: Heatstroke. Esophageal bruises. Disseminated intravascular coagulation. Esophagus. Gastrointestinal bleeding.

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INTRODUCTION

Heatstroke is a serious, potentially fatal illness characterized by a rise in body temperature to around 40 °C. The clinical and metabolic signs that arise from this situ-

ation include tachycardia, hyperventilation, arterial hypotension, respiratory alkalosis, and lactic acidosis. Hypophosphatemia and hypokalemia are common in the initial symptomatic stage. All these disorders are the result of multi-organ failure, which occurs in this pathology and then progresses to encephalopathy including delirium, convulsions or coma, rhabdomyolysis, acute renal failure, acute respiratory distress syndrome, myocardial damage, hepatocellular injury, ischemia or intestinal infarction, and hemorrhagic complications, particularly disseminated intravascular coagulation (1).

We report the case of a young patient who showed signs of gastrointestinal bleeding of esophageal origin as a complication of heatstroke.

CASE REPORT

A 26-year-old male was admitted to the emergency department of our hospital for loss of consciousness, sinus tachycardia, serious dehydration, and hyperthermia of 40 °C. At the time of admission a blood test showed Hb 14.9 g/dL, leukocytes 8660/μL (63% neutrophils, 30.9% lymphocytes), platelets 139,000/μL. Prothrombin activity was 59%, fibrinogen 380 mg/dL, and cephalin time 27.6 seconds. The following stood out from the biochemical study: glucose 169 mg/dL, urea 38 mg/dL, creatinine 2.5 mg/dL, CPK 733 IU/L, Na 135 mmol/L, K 4.2 mmol/L, Cl 97 mmol/L. Arterial blood gases: metabolic acidosis. Within 24 hours after admission, platelets were 13,000, but coagulation times could not be measured due to incoagulable blood samples. Creatinine 1.7 mg/dL, CPK 2034 UI/L, total bilirubin 2.3 mg/dL, AST 883 UI/L, ALT 932 UI/L.

After his admission into the Intensive Care Unit he was diagnosed with serious heatstroke, which throughout its evolution resulted in complications such as distribu-

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Fig. 1.- Multiple submucosal bruises in the upper esophageal third. *Múltiples hematomas submucosos en el tercio superior esofágico.*

Fig. 2.- Detail of bruises, which appear like well-delimited, red papulas over the esophageal mucosa. *Detalle de los hematomas, que se muestran como pápulas rojas bien delimitadas sobre la mucosa esofágica.*

tive shock, acute renal failure, serious hepatic insufficiency, and disseminated intravascular coagulation. On the day of his admission he was suffering from massive hematemesis, for which a gastric endoscopy was undertaken.

Fig. 3.- Multiple erosions, some of them with a fibrinous base, and submucosal bruises in the lower esophageal third. *Erosiones múltiples, algunas con fondo fibrinoso, y hematomas esofágicos en el tercio inferior del órgano.*

Endoscopic examination revealed arytenoid folds and Killian's bundle of an edematous and congestive nature, with a violet coloring suggestive of submucosal bruises. In the upper esophagus, multiple submucosal bruises were observed, with the largest being approximately 2.0 cm in size, and a petechial hemorrhage extending to the mid esophagus, which showed no lesions. The lower esophagus revealed a pattern of submucosal bruising similar to the proximal esophagus, with multiple erosions on a fibrinous base, and evidence of sloughing and tearing of the GE junction mucosa at the cardia.

The stomach and duodenum contained old blood and coffee-ground remnants, but was otherwise normal, lesion-free in its mucosa.

Another endoscopy was undertaken twenty days after the first one, with normal results of the examination except for the presence of an isolated angioma in the gastric canal.

DISCUSSION

Impaired coagulation is a cause of hemorrhagic phenomena in heatstroke. Our patient showed laboratory data suggesting disseminated intravascular coagulation, which worsened during the first few hours after admission. The mechanism underlying impaired coagulation has not been clearly defined, but its multi-factor origin has been proposed, which is initially explained by cell necrosis with a massive release of tissue thromboplastin,

which would activate both coagulation pathways (2). It has been more recently suggested that a direct activation of platelets by heat could be in the origin of this coagulopathy (3), and endothelial activity or damage, with a release of cytokines and pro-coagulating factors, has been documented (4). Severe hepatic failure with a reduction of prothrombin activity contributes to the presence of hemorrhagic phenomena (5).

Impaired coagulation persisted during the following days, with the patient requiring transfused blood derivatives and manifesting as hematic secretions through tracheostomy, mouth, and melanic stools.

Gastrointestinal bleeding has been described as one of the signs present in most severe cases, arising from mesenteric ischemia because of a reduction of splanchnic blood flow. Moreover, cases of ischemic gastritis through mucosal vasoconstriction have been recorded (6). Prolonged hyperthermia has been linked to edema of the intestinal wall, and occasionally petechial bleeding (7). In reviewing the literature available we found no cases of gastrointestinal bleeding from heatstroke being caused by esophageal submucosal bruising.

In the first endoscopy, our patient showed a normal gastroduodenal mucosa, and hemorrhagic signs were well confined to the proximal and distal esophageal thirds. In our opinion, the development of submucosal bruising in the digestive tract would be more directly related to coagulation disorders (disseminated intravascular coagulation with severe thrombocytopenia and/or serious hepatic insufficiency) than to organ hypoperfusion. We do not know why the mid esophagus was free of lesions.

The patient responded satisfactorily to the support

treatment established, and the esophageal lesions regressed to their original state within a few days.

To conclude, esophageal submucosal bruising is a possible sign of serious heatstroke that had never been described before, and may be a cause of hematemesis. The disordered coagulation that occurs in this situation is secondary to disseminated intravascular coagulation and/or serious hepatic insufficiency. Lesions are reversible once the underlying pathology has been resolved.

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