**Cocaine-induced ischemic colitis**

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**INTRODUCTION**

Ischemic colitis in young adults is an uncommon phenomenon. Among the nonocclusive etiologies, the use of cocaine can be cited as a cause of this kind of lesion, since it is demonstrated that this drug might cause cardiovascular alterations, such as myocardial infarction, arrhythmias and cerebrovascular events, regardless of the way it was used. The gastrointestinal consequences of cocaine use are infrequent but, when they occur, they can manifest as gastroduodenal ulcers, mesenteric infarction and intestinal ischemia. The proposed mechanisms are multifactorial and include direct toxicity of the intestinal mucosa, mesenteric vasoconstriction and alteration of the platelet aggregation, which generate focal ischemia. The clinical picture associated with the drug use is generally evident between twenty-four to seventy-two hours of consecutive use. The inflammatory process and the ischemia occur mostly in the distal colon.1,2

**OBJECTIVE**

Report a case of ischemic colitis in a young male who is a cocaine user and presented abdominal pain and hematochezia.

**CASE REPORT**

Male, 43 years old, presenting diffuse abdominal pain, with no signs of peritoneal irritation, and a history of recent and prolonged use of inhalatory cocaine. He was submitted to abdominal CT that showed no alterations. There was insufficient improvement with analgesics, evolving to hematochezia and discrete leucocytosis after a week. A colonoscopy was performed which revealed linear and irregular ulcers, edema and enanthema in the sigmoid colon (Figure 1). The biopsy brought evidences of chronic colitis with rare superficial ectasic capillaries, not dismissing ischemic colitis. The cardiologic propaedeutics with echocardiogram and scintillography showed no alteration. Improvement was observed with the clinical treatment. A colonoscopic control was performed after four months of clinical support and suspension of the drug use, when a total regression of the previous endoscopic alterations became evident (Figure 2).

**DISCUSSION**

Intestinal ischemic complications associated with the use of cocaine are rare. They could be explained by the vasoconstriction activity of the drug, which depends on its capacity of maintaining the intestinal alpha-adrenergic stimulation, inhibiting the re-uptake of dopamine and noradrenalin in the presynaptic membrane. Besides, with the use of cocaine there is also an increase in the influx of calcium in the endothelial membrane. Both mechanisms cause vasoconstriction and, consequently, a decrease in the blood intake. It is a fact that cocaine increases platelet aggregation and the formation of thrombi through the increase of tromboxane A2 and the decrease of eicosanoids. Studies on coronaries evidence the direct endothelial damage, and other studies on animals demonstrate direct damage to the intestinal mucosa. In the literature review it can observed that cases of ischemic colitis that are secondary to the use of cocaine do not depend on the way the drug is administered (intravenous or inhaled) and, in general, the patients are frequent consumers of substantial quantities. In the great majority of cases, the clinical picture initiates with abdominal pain and small digestive bleeding. In cases when a high digestive endoscopy was performed prior to a colonoscopy, some endoscopic alterations have been noticed, such as edema of the gastric mucosa, diffuse enanthema, ulcerations and subepithelial hemorrhages.1,2

**CONCLUSION**

In patients with a clinical history of abdominal pain, hematochezia and cocaine use, the hypothesis of ischemic colitis must be considered, even without clinical evidences of cardiac or neurological alterations.

**REFERENCES**