Massive pan-gastrointestinal bleeding following cocaine use

Troy Emanuel Gibbons, Kadria Sayed, George Joseph Fuchs

Little Rock, USA

Background: **An 18-year-old adolescent with cystic fibrosis developed massive gastrointestinal bleeding.**

Methods: **History, physical examination, upper and lower endoscopy and wireless capsule endoscopy were performed.**

Results: **Upper and lower endoscopy did not reveal cause of persistent bleeding. Wireless capsule endoscopy revealed pan-gastrointestinal ischemic injury. Further discussion with the patient revealed recent cocaine ingestion.**

Conclusion: **Most reported cases of gut injury following cocaine abuse describe juxtapyloric and colonic injury; this case demonstrates that ischemic gut injury after cocaine use can be extensive and may be the reason for the associated high mortality.**

World J Pediatr 2009;5(2):149-151

Key words: **cocaine abuse;**
gastrointestinal bleeding;
wireless capsule endoscopy

Introduction

Cocaine is a naturally occurring alkaloid that is most noted as a highly addictive recreational drug. Cocaine is also an indirect agonist of the sympathomimetic nervous system that can inhibit the reuptake of catecholamines in the peripheral nervous system. This blockade of catecholamines at adrenergic nerve terminals can produce potent vasoconstriction of blood vessels, and therefore, reduce oxygen delivery to end organs. This effect can be accentuated by increased body oxygen demand resulting from tachycardia, hypertension, and the pyrogenic effects of cocaine. Gastrointestinal complications of cocaine abuse have been described only in the adult population. Most reports describe ischemic injury to the peripyloric or left colonic areas of the intestine. We report an adolescent who was shown to have pan-gastrointestinal ischemic mucosal injury following cocaine use. Such findings have not been previously described.

Case report

An 18-year-old adolescent with cystic fibrosis (CF) was hospitalized because of chronic persistent abdominal pain, constipation, progressive cough and progressive weight loss over the previous 4 months. Prior to this, the patient's health status remained relatively stable. There was no recent history of nausea, hematemesis, hematochezia, or melena. The patient acknowledged occasional use of marijuana and consumption of alcoholic beverages.

Physical examination revealed a scaphoid abdomen with mild general tenderness and no rebound. There was no organomegaly or masses. Rectal examination revealed an anal fissure and firm stools that were slightly positive for occult blood. The patient's admission hematocrit was 43.2%.

During the first 4 days of hospitalization, there was progressive improvement in the patient's health status with daily soft stools. On day 5 of hospitalization the patient was given a pass to leave the hospital. Upon return, the patient complained of increasing abdominal pain and was observed to have large, frankly bloody stools and the hematocrit fell to 27.3%. Coagulation studies revealed prothrombin (PT) and partial thromboplastin time (PTT) were within normal laboratory range with an international normalized ratio (INR) of 1.1. Over the next 48 hours, 4 units of packed red blood cells (PRBCs) were transfused owing to hemodynamic instability. An upper and lower endoscopy was then performed. There were no features of esophagitis or varices. The gastric mucosa had diffuse moderate-to-severe erythema particularly in the prepyloric area where a large superficial ulceration with an accumulation of fibrinous exudate was noted

Author Affiliations: Department of Pediatrics, Section of Gastroenterology, Hepatology, and Nutrition (Gibbons TE, Fuchs GJ) and Department of Pathology (Sayed K), University of Arkansas for Medical Science, Arkansas Children's Hospital, Little Rock, Arkansas, USA

Corresponding Author: Troy Emanuel Gibbons, MBBS, Department of Pediatrics, Section of Gastroenterology, University of Arkansas for Medical Sciences, College of Medicine, Arkansas Children's Hospital, 800 Marshall St., Slot 512-7, Little Rock, Arkansas 72202, USA (Tel: 1-501-364-1004; Fax: 1-501-364-6291; Email: gibbonstroye@uams.edu)

doi:10.1007/s12519-009-0030-5

(Fig. A) while colonoscopy revealed scattered, well-circumscribed islands of submucosal bright red hemorrhage with normal-appearing surrounding mucosa (Fig. B). No active bleeding was noted. Two more units of PRBCs were required to maintain hemodynamic stability following colonoscopy. As it was felt that the source of continued intestinal blood loss was not completely defined, a wireless video capsule endoscopy (Given Imaging, Yoneam, Israel) was performed. The study revealed multiple areas of well-circumscribed, deeply erythematous lesions with villi erosion and edema diffusely throughout the small intestine (Fig. C, D), from the proximal small intestine to the distal ileum. Passage of the capsule through the small intestine was markedly delayed (>8 hours), particularly through the distal small intestine where the lesions were most prominent and associated with significant edema and complete loss of villi. Histologically, gastric biopsy showed mucosal erosion with edema of the lamina propria and early re-epithelialization. Rapid urease test (CLOtest® Kimberly-Clark/Ballard Medical Products, Draper, USA) and histological evaluation for *Helicobacter pylori* were negative. Colonic biopsies showed prominent ischemic injury with necrosis.

Upon repeat questioning following the diagnostic procedures, the patient admitted recent cocaine use prior to hospitalization. Over the next 4 days, the abdominal pain gradually resolved and the patient slowly advanced to a regular diet without incident.

Discussion

Ischemic effects of cocaine on the gastrointestinal tract have been previously reported in adults and are associated with a high rate of mortality, ranging from 17% to 29% in the 2 larger case series documented in the literature.[1,2] In these case series and other retrospective reviews, cocaine-associated gastrointestinal injury involved the colon, the juxtapyloric region of the stomach, and proximal duodenum of the small intestine.[3-5]

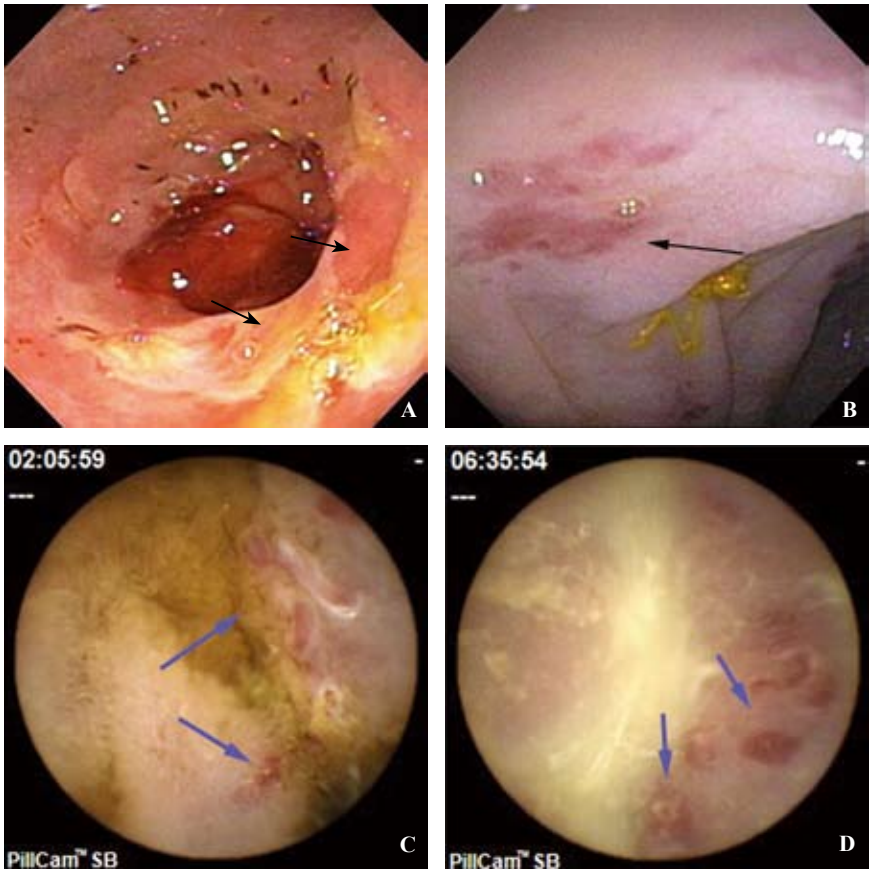

Fig. Endoscopic pictures of a large superficial ulcer in the pyloric channel covered with fibrinous exudates (**A**). Islands of submucosal hemorrhage with surrounding edema predominantly on the left side of the colon (**B**).Video capsule endoscopy pictures in the middle small intestine showing erosions with surrounding damage to villi (**C**) and distal small intestine with deep erosions, edematous mucosa, fibrinous exudate, and complete loss of villi (**D**).

Only 7 cases of small intestinal injury with subsequent outcome have been reported, 3 of whom died (43% mortality);[6-8] 6 were diagnosed by laparotomy while 1 was diagnosed at postmortem. An additional 3 cases were reported by Ellis et al;[2] however, the outcomes of these cases were not defined.

In addition to the patient's young age, this case is unusual in that it is the first description of pathologic involvement of cocaine ingestion throughout the small bowel, an observation made possible by video capsule endoscopy. This finding is in keeping with the postulation that gut injury occurs because of potent vasoconstriction of the mesenteric arteries and their branching vessels. Ischemic injury seemed maximal in the distal small intestine. Our patient's delayed intestinal transit time was likely due to the ischemic effects on gut motility that was further aggravated by the significant edema seen in the distal small intestine.[9] To our knowledge, this patient is the only reported adolescent case of cocaine-induced ischemic injury of the small intestine.

The long interval between inhalation of cocaine and the intestinal bleeding is unexpected; however, similar temporal associations have been reported, usually with chronic use. The patient described social use of an alcoholic beverage during temporary leave from the hospital. The combination of alcohol and cocaine could lead to the production of cocaethylene which has a prolonged half life and could contribute to prolonged episodes of vasoconstriction.[10] We conclude that cocaine abuse can be associated with severe pan-gastrointestinal injury which may account for the high associated mortality. While cocaine abuse with subsequent gastrointestinal bleeding is rare in the pediatric population, the pediatrician should consider this as differential diagnosis especially if there is a history of illicit drug abuse. This case also demonstrates the versatility of wireless capsule endoscopy in defining small intestine pathology.

Funding: None.
Ethical approval: Not needed.
Competing interest: No benefits in any form have been received or will be received from any commercial party related directly or indirectly to the subject of this article.
Contributors: Gibbons TE proposed the study and wrote the first draft. Sayed K analyzed the data. All authors contributed to the design and interpretation of the study and to further drafts. Fuchs GJ is the guarantor.

References

1 Linder JD, Mönkemüller KE, Raijman I, Johnson L, Lazenby AJ, Wilcox CM. Cocaine-associated ischemic colitis. South Med J 2000;93:909-913.
2 Ellis CN, McAlexander WW. Enterocolitis associated with cocaine use. Dis Colon Rectum 2005;48:2313-2316.
3 Abramson DL, Gertler JP, Lewis T, Kral JG. Crack-related perforated gastropyloric ulcer. J Clin Gastroenterol 1991;13:17-19.
4 Yang RD, Han MW, McCarthy JH. Ischemic colitis in a crack abuser. Dig Dis Sci 1991;36:238-240.
5 Niazi M, Kondru A, Levy J, Bloom AA. Spectrum of ischemic colitis in cocaine users. Dig Dis Sci 1997;42:1537-1541.
6 Nalbandian H, Sheth N, Dietrich R, Georgiou J. Intestinal ischemia caused by cocaine ingestion: report of two cases. Surgery 1985;97:374-376.
7 Freudenberger RS, Cappell MS, Hutt DA. Intestinal infarction after intravenous cocaine administration. Ann Intern Med 1990;113:715-716.
8 Muñiz AE, Evans T. Acute gastrointestinal manifestations associated with use of crack. Am J Emerg Med 2001;19:61-63.
9 Radhakrishnan RS, Xue H, Weisbrodt N, Moore FA, Allen SJ, Laine GA, et al. Resuscitation-induced intestinal edema decreases the stiffness and residual stress of the intestine. Shock 2005;24:165-170.
10 Landry MJ. An overview of cocaethylene, an alcohol-derived, psychoactive, cocaine metabolite. J Psychoactive Drugs 1992;24:273-276.

Received May 21, 2008
Accepted after revision July 18, 2008