Acute haemorrhagic ischaemic colitis secondary to cocaine use

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DESCRIPTION

A man, in his mid-60s, with no significant medical history, presented with acute onset, sharp, stabbing abdominal pain associated with nausea and vomiting. On admission, he was haemodynamically stable, however, appeared unwell, in significant painful discomfort. Physical examination revealed a diaphoretic, tachycardiac man with bloodshot eyes, dilated pupils and a rigid abdomen with guarding.

On admission, laboratory investigations revealed haemoglobin (Hgb) 13.5 g/dl, an acute kidney injury (Blood Urea Nitrogen 25, creatinine 1.7) with lactic acidosis. A CT abdomen/pelvis reported findings of proctitis with rectal wall thickening and adjacent mesenteric stranding (figure 1). While in the emergency department, the patient's clinical status acutely deteriorated with the development of massive lower gastrointestinal (GI) bleeding, characterised by bright red blood per rectum. The patient was fluid resuscitated and started on vancomycin/cefepime for presumed infectious colitis. The patient continued to endorse abdominal discomfort with haematochezia. Serial complete blood counts (CBCs) revealed a down-trending Hgb to 9 g/dL. Stool testing returned negative for *Clostridium difficile*, enteric pathogens and ova/cyst/parasites. He was planned for colonoscopy to evaluate his abdominal pain and haematochezia.

Colonoscopy revealed severe, diffuse inflammation characterised by erythema, erosions, ulcerations with mucosal bleeding, through the descending colon, splenic flexure and transverse colon consistent with left-sided colitis (figure 2). Segmental colonic biopsies were taken during the procedure. The procedure was aborted for safety reasons at the hepatic flexure given the severity of inflammation characterised by friability and contact bleeding. Biopsies returned with colonic mucosal haemorrhage, mucosal necrosis with acute inflammation strongly suggestive of acute haemorrhagic ischaemic colitis with mucosal necrosis.

Stool testing and colonic biopsies excluded an infectious aetiology of his colitis. There was no clear aetiology or hypotensive period to account for haemorrhagic ischaemic colitis on pathology. The patient's presenting history was revisited and, on this occasion, he admitted to significant cocaine use just prior to the onset of abdominal pain. Urine toxicology returned positive for cocaine which in part, accounts, for his diaphoresis and markedly dilated pupils on admission. The patient was managed conservatively with fluid support and liquid diet, advancing as tolerated. His postprocedure hospital course was uneventful and the patient was discharged on day 4 of hospitalisation as his haematochezia resolved and haemoglobin remained stable. He was extensively counselled on continued cocaine use, risk of overdose and death and provided with necessary information on rehabilitation centres.

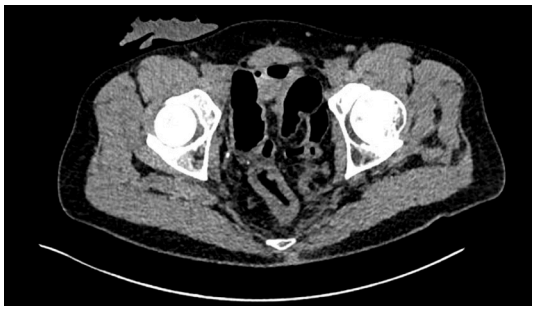

Figure 1 CT abdomen/pelvis with rectal wall thickening with mild adjacent mesenteric stranding suggestive of proctocolitis.

Cocaine is a highly addictive, powerful stimulant for the nervous system that can be snorted, smoked or injected. In the USA, 1 in every 5 overdose deaths were related to cocaine use with an estimated 24 486 deaths in 2021.[1] In 2020, cocaine use was reported by over 5 million Americans, which is almost 2% of the population.[2]

Cocaine toxicity leading to GI injury is of a multifactorial pathophysiology. Cocaine's direct vasoconstrictive effects via enhancement of a calcium flux across endothelial membranes result in ischaemia/infarction.[3] The sympathomimetic effects of cocaine via inhibition of presynaptic norepinephrine reuptake result in an accumulation of catecholamines which causes extensive mesenteric vasoconstriction with hypoperfusion and focal ischaemia of the intestines.[4] Furthermore, cocaine may cause thrombus formation and platelet aggregation via activation of the intravascular thrombosis cascades with additional hypoperfusion.[3] Notably, the direct prothrombotic effect of cocaine is not

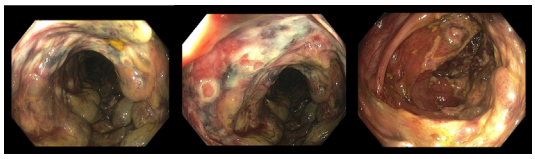

Figure 2 Endoscopic photograph of colonic mucosa demonstrating mucosal bleeding, erythema, scattered erosions and ulcerations consistent with severe colonic ischaemia.

typically from the drug itself, but rather from cocaine cut with levamisole resulting in a cocaine/levamisole-induced vasculopathy.[5] There is an interval time of 1 hour to 2 days from drug use to symptom onset, usually characterised by abdominal pain and bloody stools.

Colonic ischaemia is an uncommon medical condition that carries significant morbidity and high mortality. The incidence of colonic ischaemia is about 22.9/100 000 person-years with notable risk factors including elderly population, mesenteric vascular atherosclerosis, atrial fibrillation, cardiac disease, shock, long distance running, severe dehydration, hereditary and acquired thrombophilia, and illicit substance abuse.[6] Affected patients typically present with sharp abdominal pain, that is typically out of proportion to physical signs, accompanied by nausea and vomiting. Physical examination findings include abdominal tenderness on palpation, rebound tenderness (Blumberg's sign), rigidity and guarding. In the late phase, patients can develop bloody diarrhoea, fever and shock.

The superior mesenteric artery (SMA) supplies the bowel from the distal duodenum to the proximal two-thirds of the transverse colon. The inferior mesenteric artery (IMA) supplies the large intestine from the distal one-third of the transverse colon to the superior aspect of the rectum. Colonic perfusion can be compromised by changes in systemic circulation or by anatomic or functional change in local vasculature.[7] The 'watershed' areas namely the splenic flexure (Griffiths point) and rectosigmoid junction (Sudek's point) are prone to ischaemia as the regions between the two major arteries supplying the colon. Watershed areas account for nearly 70% of ischaemic colitis cases given their limited vascular collateralisation.[6]

Intestinal ischaemia occurs when there is at least a 75% reduction of intestinal blood flow for more than 12 hours.[8] Ischaemic colitis typically requires a low flow state, usually in patients whose vascular bed is already compromised by fixed atherosclerotic obstruction in the branches of the SMA and IMA. Hence, ischaemic colitis is more common in elderly patients with haemorrhagic shock when compared with younger trauma victims with the same degree of shock. Cocaine use would typically increase organ perfusion by increasing the mean arterial pressure and cardiac output, however, taking into account the vasoconstrictive effects of cocaine, it may paradoxically cause ischaemia by triggering mesenteric vasospasm. The pathophysiology of cocaine toxicity leading to ischaemic colonic injury via catecholamine induced mesenteric vasospasm is of a similar pathophysiology as cocaine induced coronary vasospasm causing myocardial infarction.[9]

Mesenteric vasospasm causing hypoperfusion and hypoxia leads to necrosis of the mucosal villi followed by transmural, mural or mucosal infarction. In response to injury, the intestinal walls initially become congested, subsequently appearing edematous, friable and haemorrhagic. As sloughing of infarcted mucosa occurs, there is increasing damage and exposure of bowel capillary vessels which in the late stage of colonic ischaemia presents with rectal bleeding.

The usual differential of rectal bleeding with abdominal pain includes infectious, inflammatory (Crohn's disease, ulcerative colitis) or ischaemic. Ischaemic colitis is usually suspected in elderly patients, especially those with underlying cardiovascular disease. Cocaine use is a life-threatening cause of ischaemic colitis and should be included in the differential diagnosis of any patient presenting with abdominal pain and bloody diarrhoea. Patients with cocaine use associated colitis have a significantly higher mortality than patients with ischaemic colitis (26% vs 7.7%).[10] Cocaine colitis is an easily missed diagnosis and a history of illicit drug use and/or urine toxicology is a vital component of the diagnostic work-up.

Patient's perspective

Been doing coke all my life, this time I went heavy. I've tried stopping before, but you'd never understand how hard it is.

Learning points

- Cocaine colitis is an uncommon and easily missed aetiology of the acute abdomen and requires a high clinical suspicion for diagnosis.
- Abdominal pain associated with lower gastrointestinal (GI) bleeding is an alarm symptom and requires urgent evaluation.
- The usual differential diagnosis of rectal bleeding includes infectious, inflammatory or ischaemic. Clinicians should be aware of usual aetiologies in at-risk populations and promptly recognise cocaine toxicity as a cause of GI bleeding.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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