result in resolution of the condition, these measures are not always feasible or successful. Conventional treatment for lymphoma in these patients poses unique problems as the combination of chemotherapy-induced neutropenia and immunosuppressive drug therapy leads to a degree of compromise of immune status that often results in a lethal opportunistic infection, as in our case.


Gastrointestinal haemorrhage associated with free-base (crack) cocaine

DA Fennell, SS Gandhi, BNC Prichard

Summary
We report a case of a crack user presenting with chronic gastrointestinal haemorrhage due to deep gastric ulceration; the putative aetiology being predictable from this agent's pharmacology.

Keywords: crack cocaine, haemorrhage, gastric ulceration

The spectrum of multisystem toxicity for cocaine and the alkaloid free base 'crack' includes gastropropic, duodenal ulceration, and perforation. Its heat stability, conferred by dissociation of the hydrochloride moiety during transformation from cocaine to free base, underlies its pharmacokinetic behaviour, providing (via inhalation) extremely efficient drug delivery. The fast (below 30 s) rise to peak plasma concentration of this potent sympathomimetic during smoking potentially predisposes the user to systemic ischaemic pathology.

Case report
A 34-year-old male primary crack user presented with a one-week history of melaena, haematemesis and epigastric pain. There was no recent history of non-steroidal anti-inflammatory use, and although a cigarette smoker (20 per day) there was no previous nor familial history of peptic ulcer disease. He had recently been smoking crack cocaine on a daily basis. On examination, he was afebrile, haemodynamically stable, but profoundly anaemic (haemoglobin 4.8 g/dl, mean corpuscular volume 90 fl), with generalised upper abdominal tenderness, and evidence of melaena. Urgent upper gastrointestinal endoscopy identified a very deep ulcer (with no evidence of scarring) localised at the incisura, which was injected with adrenaline. The patient discharged himself 48 hours after admission.

Clinical features of crack use

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<tr>
<th>Pharmacokinetics</th>
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<tr>
<td>rapid absorption via inhalation</td>
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<td>eliminated by plasma esterases; half-life 50 min</td>
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<th>Pharmacodynamics</th>
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<tr>
<td>euphoria</td>
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<td>sympathetic activation (ie, tachycardia, peripheral vasoconstriction)</td>
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<td>intense psychological dependence</td>
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<th>Toxicity</th>
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<td>convulsions</td>
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<td>psychotic symptoms</td>
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<tr>
<td>hyperthermia</td>
</tr>
<tr>
<td>multisystem pathology secondary to vasospasm (eg, cutaneous, myocardial, cerebral, bowel infarction)</td>
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Discussion

This demographically atypical presentation of haemorrhage from gastric ulceration shows consistencies with observations previously described in the US. To our knowledge this is the first report in the UK of gastric ulceration complicating inhaled crack cocaine; a plausible mechanism underlying the pathogenesis being predictable from this illicit drug’s pharmacology.

Gastric mucosal blood flow provides an important path for the disposal of influxing luminal acid, and thus an important barrier mechanism. Owing to cocaine’s pharmacodynamics, namely monoaminergic synaptic potentiation (by inhibition of the uptake I transporter), and consequent alpha-1 adrenoceptor mediated mucosal arteriolar vasoconstriction, this presumably disrupts mucosal ‘proton washout’, predisposing to gastric ulceration. Recurrent short ischaemic episodes associated with frequent crack smoking would provide a background for prolonged impaired mucosal defence. The slow absorption/low peak plasma kinetics of snorted cocaine hydrochloride probably explains the absence of such systemic toxic manifestations.

In view of the increasing use of this agent in Europe, similar presentations are likely to become more common. One scenario for example, being the recent upsurge in cocaine trafficking into the UK by ‘body packers’ (swallowers), whereby package rupture or leak may potentially expose these individuals to the risk of such life-threatening complications.

Summary points

- the pharmacokinetics of crack cocaine underly its specific complications
- gastrointestinal haemorrhage secondary to gastric peptic ulceration is a rare, potential complication of cocaine misuse
- peptic ulceration probably results from disruption of the gastric mucosal barrier mechanism through its sympathomimetic activity


Summary

A woman with an eight-year history of polycythemia vera presented with numbness and weakness of both legs. A large spinal haematoma was revealed on magnetic resonance imaging which was treated clinically and which subsequently resolved.

Keywords: polycythemia vera, spinal haematoma

Spontaneous, nontraumatic spinal Epidural or subdural haematomas are uncommon and are a rare cause of spinal cord compression. Approximately half of spinal haematomas are thought to be spontaneous. Spontaneous bleeding may occur in polycythemia vera. However, polycythemia vera has not been previously described as a cause of spontaneous spinal haematoma.

Case report

A 60-year-old woman with an eight-year history of polycythemia vera presented with numbness and weakness of both lower extremities lasting 30 minutes. Although she had been maintained on phlebotomy on a regular basis, her platelet count was 1.25 x 10^9/l. Emergency magnetic resonance imaging (MRI; figure) of the spine demonstrated a large subdural haematoma from T7–S2 with spinal cord compression. Due to the patient’s bleeding tendency, it was elected to follow her clinically rather than perform surgical decompression. Treatment also included hydroxyurea, interferon and platelet pheresis. Her motor strength continued to improve and she was discharged. Follow-up outpatient MRI demonstrated resolution of the spinal haematoma.
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