CASE REPORT

Chronic lead poisoning in an Iranian opium smoker resident in London

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SUMMARY

A 46-year-old man of Iranian origin presented with a 4-day history of colicky abdominal pain and absolute constipation on a background of several weeks of irritability and malaise. He had smoked 10 g of opium per week for a year and a half. On examination, he had diffuse abdominal tenderness and faecal loading. This was cleared, but the abdominal pain, nausea and vomiting persisted. He had extravascular haemolytic anaemia with punctate basophilic stippling on blood film. The patient's serum lead concentration was substantially elevated and he perhaps demonstrated Burton's line. The patient underwent chelation therapy and has recovered clinically and biochemically. Public health experts were notified and conducted an assessment of the risk to the patient and others; their lead exposure questionnaire was subsequently amended. This is an important case report of a UK resident describing lead toxicity secondary to the inhalation of opium.

BACKGROUND

Abdominal pain of unknown aetiology is a common presentation encountered by emergency physicians, surgeons, medics and general practitioners. The differential diagnosis is broad. Rare diagnoses must always be considered. This is a case report of a UK resident with lead toxicity secondary to the inhalation of opium.

CASE PRESENTATION

A 46-year-old man presented as an emergency to a general surgical team of a London district general hospital with a 4-day history of worsening severe colicky abdominal pain and absolute constipation. He also had a 3-day history of nausea, vomiting and anorexia. The pain was migratory in nature initially from his right iliac fossa to the suprapubic region and would not change with food, antacids or body position.

The patient is of Iranian origin and works in haulage. He had smoked 10 g of opium per week for a year and a half, and smoked 15 cigarettes a day. He took no medications regularly and had no past surgical or relevant family history. He had successful eradication of *Helicobacter pylori* in 2011. He emigrated from Iran 20 years ago and has not returned in the past 3 years.

On examination, the patient had diffuse abdominal tenderness and fullness on the right side of the abdomen. His bowel sounds were present and normal in character. His per rectal examination revealed an empty rectum with no masses, blood or

mucus. The CT scan showed marked faecal loading within the right colon. The patient was admitted and laxatives successfully cleared the faecal loading, but the abdominal pain, nausea and vomiting persisted.

On further assessment, he had felt fatigued and easily irritated for several weeks, which had strained his personal relationships. He had also experienced frequent, widespread 'pins and needle sensations' in his arms and legs. His abdominal pain and tenderness continued to migrate around the abdomen and appeared to worsen when interacting with medical staff, suggesting the possibility of drug-seeking behaviour. His cardiovascular, respiratory, abdominal and neurological examinations were normal. On day 5 after admission, initial biochemical and haematological abnormalities were further investigated (table 1)—his notable findings were an elevated alanine transaminase of 486 IU/L, bilirubin 28 µmM, conjugated bilirubin 12 µmM, Hb 101 g/L, corpuscular volume 87.9 fL, reticulocyte percentage 5.7 and haptoglobin 0.84 g/L. His blood film showed anisocytosis, polychromasia and punctate basophilic stippling. A single disease process would therefore suggest a non-inflammatory disease causing extravascular haemolytic, normocytic anaemia with gastrointestinal, hepatic, neurological and constitutional involvement.

DIFFERENTIAL DIAGNOSIS

The unusual and changing nature of the patient's abdominal pain prompted us to ask for review by the hospital's Drug and Psychiatric Team querying narcotic bowel syndrome, opiate withdrawal, somatoform disorders and factitious disorders. His pain worsened with tramadol but not with other opiates. He had experienced opiate withdrawal before; his pains appeared to be too severe for withdrawal. He had indeed suffered severe trauma during his life and was diagnosed with post traumatic stress disorder. He was very forthcoming with details on the stressors in his life and his drug habits. In addition, he was keen to reduce his in-hospital analgesic intake and to engage with drug and alcohol services. Their assessment was that his principal problem was more likely to have an organic rather than a psychiatric origin, although psychological factors (including secondary gain) were likely to be contributory, and that his sensory disturbances could be explained by intermittent symptoms of withdrawal.

Organic causes for his presenting complaint of severe colicky abdominal pain, absolute constipation and vomiting were considered. Bowel obstruction (with or without herniation) and volvulus

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Table 1 Characteristic blood results from day 5 after admission

Measure	Value	Reference range
Sodium	141 mmol/L	136–145 mmol/L
Potassium	4.2 mmol/L	3.5–5.1 mmol/L
Urea	7.2 mmol/L	3.2–7.4 mmol/L
Creatinine	91 µmol/L	64–111 µmol/L
eGFR	L 77 mL/min	>90 mL/min
Bilirubin	H 28 µmol/L	3–21 µmol/L
Conjugated bilirubin	H 12 µmol/L	<10 µmol/L
ALP	63 IU/L	40–150 IU/L
ALT	H 486 IU/L	<56 IU/L
Total protein	70 g/L	64–83 g/L
Albumin	42 g/L	35–50 g/L
Calcium	2.36 mmol/L	2.15–2.61 mmol/L
Calcium—corrected	2.34 mmol/L	2.15–2.61 mmol/L
Magnesium	0.91 mmol/L	0.66–1.07 mmol/L
Phosphate	0.92 mmol/L	0.74–1.52 mmol/L
Amylase	43 IU/L	25–125 IU/L
CRP	<1 mg/L	<6 mg/L
ESR	H 10 mm/hour	1–7 mm/hour
Haemoglobin	L 101 g/L	130–180 g/L
White cell count	8.8×10^9 /L	$3.5\text{--}11.0 \times 10^9$ /L
Platelets	360×10^9 /L	$130\text{--}450 \times 10^9$ /L
Mean corpuscular volume	87.9 fL	78.0–100.0 fL
Red blood cell count	$L\ 3.38 \times 10^{12}$ /L	$4.50\text{--}5.50 \times 10^{12}$ /L
Haemocrit	L 0.297 L/L	0.450–0.500 L/L
Ferritin	H 429 µg/L	22–275 µg/L
Folate	3.6 µg/L	3.1–20.5 µg/L
Vitamin B ₁₂	347 ng/L	187–883 ng/L
Reticulocyte percentage	H 5.7%	0.5–2.0%
Reticulocyte count	$H\ 194 \times 10^9$ /L	$30\text{--}100 \times 10^9$ /L
Film comment	Anisocytosis, polychromasia Basophilic stippling Suggestive of haemolytic anaemia	
Haptoglobin	0.84 g/L	0.14–2.58 g/L

The changes in liver function tests and cell counts were similar on admission. Any abnormal values are marked with 'H' or 'L' for high or low, respectively, before the value. ALP, alkaline phosphatase; ALT, alanine transaminase; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; ESR, erythrocyte sedimentation rate; HbSC, haemoglobin SC disease; HbS, sickle cell trait; MCV, mean corpuscular volume; PTSD, post traumatic stress disorder.

formation were high on the initial differential, although discounted by no past surgical history, normal examination and bowel sounds, and excluded on CT of the abdomen and pelvis. The CT also showed no evidence of kidney stones or hydronephrosis and in conjunction with his normal inflammatory markers excluded acute appendicitis, colitis, cholecystitis and diverticulitis. Haemodynamic stability, no recent abdominal insult and the normal CT scan excluded splenic injury. His normal serum electrolytes and normal bowel sounds excluded metabolic ileus. His normal serum lactate, amylase and urinalysis also discounted ischaemic bowel, pancreatitis and urinary tract infections. The acute hepatitis screen, including paracetamol levels, was negative; he had no history of alcohol use or hypotension. Testicular examination was normal with no masses, signs of inflammation or marked tenderness. It was thought that the significant faecal loading seen on CT scan secondary to his opiate use was the cause of his abdominal pain, but the pain persisted even after the faeces had been cleared with laxatives and the patient was passing soft stool regularly. Therefore, further causes for abdominal pain and anaemia were considered. His oesophago-gastro duodenoscopy showed minor

gastritis and a small hiatus hernia—it excluded *H. pylori* and peptic ulceration. Familial Mediterranean fever and paroxysmal nocturnal haemoglobinuria were unlikely due to his normal inflammatory markers, normal lactate and urine. Haemolytic anaemias (such as haemoglobin SC disease and sickle cell trait) and porphyria were considered for further evaluation of haemoglobinopathy and urinary metabolites. Heavy metal toxicities, including lead toxicity, were investigated as it could cause diverse symptoms, anaemia and abdominal pain. Furthermore, we knew that his accommodation had recently been of poor quality, suggesting a possibility of environmental exposure to lead.

INVESTIGATIONS

On day 9 of his admission, we were notified by the National Poisons Information Service that the patient's serum lead level was 5.5 µmol/L, which is 11 times the nominally desirable level. A diagnosis of lead poisoning could explain nearly all of his pathology. Constipation and colicky, migratory abdominal pain are part of the classical presentation of moderate to severe chronic lead toxicity—as is psychological dysfunction and basophilic stippling on blood film (figure 1). He was then noted to possibly demonstrate Burton's line (figure 2).

TREATMENT

The National Poisons Information Service recommended a sodium calcium edetate infusion regimen to chelate and renally excrete the serum lead (figure 3). The infusion substantially reduced his serum lead concentration and the patient experienced no side effects.

OUTCOME AND FOLLOW-UP

The local health protection team at Public Health England (PHE) was notified and assessed the patient, in collaboration with the Centre for Radiation, Chemical and Environmental Hazards to determine the likely source of lead exposure and any risk to others. After PHE's assessment, they concluded that the likely pathway of lead exposure was smoking opium.

Figure 1 A red blood cell displaying basophilic stippling from the patient's film. A sign of chronic lead poisoning.

Figure 2 Blue discolouration of the patient's gums tracking adjacent to his teeth (Burton's line). A sign of chronic lead poisoning.

Figure 3 Urinary lead excretion and serum lead concentration against time. Day 0 is before the EDTA chelation infusion began.

The patient has returned to his home, is engaged with drug services and has not smoked opium since hospital discharge. He has no symptoms and all of his biochemical abnormalities are returning to normal. PHE's lead action card, the guide for lead toxicity investigation for local public health teams, will be changed in the new iteration to prompt investigating opium or other drugs as a potential exposure source. Local healthcare providers and drug services were alerted to the disease aetiology.

DISCUSSION

Exposure to inorganic lead occurs primarily through food and drinking water, although exposure via soil, dust, air and paint chips significantly contribute to the total number of cases of lead toxicity. In adults, lead poisoning is usually a result of occupational exposure.¹ The patient's history did not contain a recent dietary, environmental or occupational source of lead exposure; therefore, it was questioned whether his opium consumption might be the potential source. This phenomenon is well documented in Iranian medical literature. In Iran, biochemically verified case reports of lead poisoning due to opium exposure have

been published. One review concluded that 25 of 240 cases of lead poisoning diagnosed in Tehran over 1 year were caused by smoking contaminated opium.^{2–4} It is believed that lead is added to increase opium's weight for sale or is a contaminant incorporated during processing; lead levels can be considerable.^{5,6} Similar cases of lead toxicity caused by contaminated marijuana and heroin have been published.^{7,8} This is a case report of a UK resident who developed lead toxicity secondary to the inhalation of opium. Unfortunately, the patient has as yet been unable to provide a sample of opium for analysis.

Patient's perspective

I am back at work and I feel like I have the power to fight in my life again. I hope my story could alert doctors around the world to other patients, especially other Iranians (where opium smoking is prevalent), who might be suffering like I was.

Learning points

- ▶ Heavy metal toxicity is still an important element of the differential of abdominal pain.
- ▶ In patients with heavy metal toxicity, illicit drug use is a risk for exposure.
- ▶ This case should alert doctors encountering a similar clinical presentation to the possibility of lead toxicity in people who smoke opium.
- ▶ This case demonstrated the value of multidisciplinary clinical input to guide the diagnostic approach in patients with complex psychological and physical morbidity.
- ▶ This case demonstrates the value of multiagency input for rare case management.

Contributors AA was lead for writing the manuscript and coordinating the manuscript submission. KF was involved in conducting the Public Health England assessment and was the lead for the section describing that assessment. AA, SD, TH and MK were involved in investigating the differential diagnosis, the data interpretation and the management of the patient. AA, KF, SD, TH and MK were all involved in searching the literature, editing and refining the manuscript.

Competing interests None declared.

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