Case Report

Acute abdominal pain and constipation due to lead poisoning

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Abstract

Although uncommon, lead poisoning should be considered as a differential diagnosis in cases of unexplained acute abdominal pain in both adults and children. We present the case of a 35-year-old Asian male who presented with abdominal pain and constipation secondary to lead poisoning. Initially, the source of lead exposure was not apparent; this was later found to be due to ingestion of an Ayurvedic herbal medicine for the treatment of infertility. Lead poisoning due to the ingestion of Ayurvedic remedies is well described. We discuss the diagnosis, pathophysiology and treatment of lead poisoning. This case illustrates one of the rarer medical causes of acute abdominal pain and emphasizes the need to take a thorough history (including specific questioning regarding the use of over-the-counter and traditional/herbal remedies) in cases of suspected poisoning or drug toxicity.

Keywords

Lead poisoning, poisoning, basophilic stippling, chelation therapy, ayurvedic medicine.

Key Learning Points

- *lead poisoning is an uncommon but important medical cause of acute abdominal pain in both children and adults.*
- *enquire specifically about the use of complementary/alternative medicines if an environmental source of lead exposure is not readily apparent.*
- *traditional or complementary remedies are not necessarily safe or 'natural'; Indian Ayurvedic herbal medicines may contain toxic amounts of heavy metals including lead, mercury and arsenic.*
- *specific chelation therapy is recommended if blood levels are ≥50µg/dL.*
- *involve the NPIS or regional toxicology unit in all cases of lead poisoning.*

Background

Case History

A 35 year-old male of Indian origin presented to the Emergency Department with acute abdominal pain and constipation. His past medical history included erectile dysfunction, tablet-controlled type 2 diabetes and asthma. He had recently attended an Endocrinology clinic for infertility associated with hypogonadotrophic hypogonadism. His only prescribed medication was metformin 500 mg TDS. On clinical examination he appeared pale, his temperature was 37.8°C, heart rate 70/min, BP 114/88mmHg, oxygen saturations 99% on air. He had tenderness in the lower abdomen but his abdomen was soft with normal bowel sounds. PR examination revealed an empty rectum. His initial blood tests revealed microcytic anaemia with an Hb concentration of 82g/L and MCV 75.8, CRP 2 mg/L, amylase 67iU/L and normal renal function. Liver function tests were mildly abnormal with an elevated bilirubin at 47µmol/L and ALT of 48iU/L. His Hb concentration three months prior to admission was normal at 147g/L. Chest and abdominal X-rays revealed no abnormality.

He was admitted initially to the Acute Surgical Unit and underwent a CT scan of his abdomen. This showed collapse of the distal sigmoid and rectum with high contrast material within the rectum, suspicious for blood. No obvious cause for his abdominal pain was found. He was discharged with arrangements for outpatient colonoscopy.

He re-presented one week later to the Acute Medical Unit with malaise, weight loss, constipation and worsening abdominal pain. His Hb concentration was 84g/L and peripheral blood film showed increased polychromasia, basophilic stippling and poikilocytosis with nucleated red cells. His serum lead level was elevated at 88.59µg/dL(normal range <10µg/dL).

On initial inquiry, he denied any history of lead ingestion and there was no obvious occupational exposure. Plain films of his abdomen did not show any radio-opaque material to suggest lead particles.On repeated questioning, he admitted to occasionally using lead pellets for his shooting hobby. He had recently ordered some lead pellets online and had to chew them between his teeth as they would otherwise not fit into his air rifle. His case was discussed with the regional toxicology centre and the UK National Poisons Information Service (NPIS). Chelation therapy was advised with oral DMSA (Succimer) at a dose of 30mg/kg/day for 5 days.

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His symptoms responded rapidly to this treatment. He was reviewed at the regional toxicology unit two weeks after discharge where it emerged that the likely source of lead poisoning was ingestion of an Ayurvedic herbal medicine for infertility which he had taken during a recent trip to India. A repeat serum lead level performed six months later had fallen to 31µg/dL. He continues to be followed up by the regional toxicology centre.

Discussion

Lead poisoning is a well-established cause of abdominal pain and should be considered in the differential diagnosis when other common causes have been excluded.[1] Lead poisoning can present with non-specific symptoms and signs such as abdominal pain, constipation (or diarrhoea), anorexia, headaches, irritability, difficulty concentrating, fatigue and insomnia. Other features include microcytic anaemia with basophilic stippling, blue lines on the gums, nephropathy, hypertension, peripheral neuropathy (predominantly motor e.g. wrist drop) and abnormal LFTs. Encephalopathy with confusion, drowsiness, coma or seizures due to cerebral oedema may occur in cases of severe toxicity. Approximately 95% of lead poisoning among adults in the US results from occupational exposure.[2] This may occur in battery manufacturing, lead and zinc ore mining, painting and paper-hanging industries. Lead poisoning can also occur in those taking certain alternative/traditional medical remedies and in those cooking or eating off lead-glazed tableware or cookware.

Inorganic lead is absorbed from the lungs or the gastrointestinal tract. Once absorbed, it is then distributed to the blood, skeleton and soft tissues. 99% of lead in blood is bound to the erythrocyte and the remaining 1% is free in the plasma to exchange with soft tissues (kidney, brain, liver and bone marrow). Lead is excreted by the kidneys and the mean half-life is about 30 days if renal function is normal.[5]

Being an electro-positive metal, lead has high affinity for negatively charged sulfhydryl groups leading to the inhibition of sulfhydryl dependent enzymes involved in heme synthesis such as gamma-aminolevulinic acid dehydratase (ALA-D) and ferrochetalase. Anaemia can therefore develop with high blood lead levels (usually>80µg/L). The characteristic appearance of basophilic stippling on the peripheral blood smear is due to degradation of ribosomal RNA in red blood cells due to inhibition of pyrimidine 5' nucleotidase by lead.[6]

A careful medical, occupational and environmental history should be obtained in all suspected cases of lead poisoning to identify potential sources of exposure.[7] Drug histories in any case of poisoning or suspected drug toxicity should include specific questioning regarding the use of over-the-counter (OTC), traditional or herbal remedies which are not prescribed. Ayurvedic medicine is native to the Indian subcontinent and is one of the oldest systems of alternative or traditional medicine. There have been numerous case reports of lead poisoning associated with Ayurvedic medicines over the last 25 years. In one study, toxic concentrations of heavy metals (lead, mercury, arsenic) were found in 20% of Ayurvedic medicines, including those that were manufactured in the US.[8] Contamination of these traditional remedies with lead and other heavy metals may occur during the manufacturing process but often, these metals are intentionally added to the medicine (*rasa shastra*). It is believed that these metals are essential components of vital molecules within the human body and that their toxicity is reduced by a purification process (*samskara*) of prayer and pharmacological methods.

Diagnosis of lead poisoning is confirmed by directly measuring the blood lead level. Other tests such aselevated free erythrocyte protoporphyrin (FEP) or zinc protoporphyrin (ZPP) levels are rarely used as an indirect measure of the effects of lead on heme synthesis. A plain abdominal X-ray may be used to confirm ingestion of elemental lead or radio-opaque salts.

Management

Management of lead posioning consists of removal of the source of exposure and chelation therapy. Removal from source of lead exposure is recommended when blood levels rise to 50µg/dL.The current acceptable blood lead levels in healthy individuals without excessive environmental exposure to lead is <10µg/dL in children and <25µg/dL in adults.[8] There are no randomised controlled clinical trials demonstrating the efficacy of chelation therapy in terms of clinical outcomes. However, chelation is generally recommended for all individuals with blood lead concentrations ≥50µg/dL. In the UK, chelation treatment is with IV Sodium calcium edetate 75-80 mg/kg/day or oral DMSA (Succimer) 30 mg/kg/day for 5 days. Monitoring of LFTs (DMSA) or renal function (sodium calcium edetate) is required. Further courses of chelation may be required if lead concentrations are still ≥50µg/dL 1 week after the end of the initial course of treatment.

In adults with features of encephalopathy, calcium edetate 75mg/kg once daily or 40mg/kg twice daily by infusion in normal saline over 1-2 hours for 5 days is recommended.[9] IV fluids may be required to ensure adequate urine output during chelation and 24h urinary lead excretion may be used to adjust the duration of therapy.All cases of significant lead poisoning should be discussed with the NPIS and/or a regional toxicology unit.

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Conflict of interest

This study was funded entirely by the authors and they have no conflict of interest with any third party.

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