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Lead induced anaemia due to traditional Indian medicine: a case report

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Abstract

Lead intoxication in adults without occupational exposure is a rare and unexpected event. The case of a western European is reported who had severe anaemia after ingestion of several avurvedic drugs, obtained during a trip to India. Laboratory findings showed high blood lead concentrations, an increased urinary lead concentration, and an increased urinary excretion of δ -aminolaevulinic acid. Also, slightly increased urinary concentrations of arsenic and silver were found. Physicians should be aware that with growing international travel and rising self medication with drugs from uncontrolled sources the risk of drug induced poisoning could increase in the future.

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The sources of lead intoxication in adults without occupational exposure are often insidious and the diagnosis can be missed in the absence of a typical history. Here we report the case of a patient who presented with severe anaemia (haemoglobin 78 g/l) after returning from a trip to India and south east Asia. The cause of the anaemia was found to be lead intoxication due to intake of traditional Indian medicine (ayurveda). The patient responded readily to treatment with D-penicillamine.

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Case report

A 37 year old western European man was admitted to our hospital because of weakness, dizziness, nausea, and diffuse muscle pain that had developed over the past few weeks. Two months before he had travelled to India for about 4 weeks, returning through south east Asia. The physical examination was unrevealing. The blood test on admission showed a normochromic anaemia with a haemoglobin concentration of 78 g/l (normal 13–17 g/l). The reticulocyte count was 5.7% (normal 0.5%-1.5%) and a slight but inconstant basophilic stippling was noted. Further laboratory tests showed an increase of aspartate aminotransferase (AST) to 44 U/l (normal 1-18 U/l), of alanine aminotransferase (ALT) to 107 U/l

(normal 1–22 U/l), and of γ -glutamyl transpeptidase (γ -GT) to 34 U/l (normal 6–28 U/l). Serum concentrations of vitamin B12 and folate were normal. Serum iron was increased to 40 µmol/l (normal 10.7-28.7 µol/l), and serum ferritin was in the upper normal range. Reduced haptoglobin of 78.9 mg/dl (normal 123-320 mg/dl) indicated a mild haemolysis; however, lactate dehydrogenase and bilirubin were not increased. There was no indication of gastrointestinal bleeding by testing the stool for occult blood and by endoscopic investigation. The bone marrow biopsy showed a slight erythropoietic hyperplasia with siderosis of reticulum cells in an otherwise normal bone marrow.

As the patient reported a febrile illness during his travels in south east Asia, we first assumed an infection as the cause of the anaemia.

A diagnostic investigation for various infectious diseases showed hepatitis C infection with detection of anti-HCV-antibodies and of HCV-RNA in the serum. Otherwise there was no evidence of further viral, bacterial, or parasitic infections, including HIV, parvovirus B19, and malaria. A liver biopsy, obtained 8 weeks later, was consistent with a chronic hepatitis C of only marginal inflammatory activity, which could not explain the anaemia and the rather severe symptoms.

As no apparent cause of the anaemia could be established a new history was taken. The patient reported that during his stay in India he visited a traditional ayurvedic medical centre where he received herbal medicine and several bowel washings with oil. He had continued most of the medication until a few days before admission to our hospital and he was still taking a paste, both pure and as spread, every day. The metallic appearance of the paste and the detection of basophilic stippling in the red blood cells raised the suspicion of heavy metal poisoning. The diagnosis of lead intoxication was confirmed by a blood lead concentration of 580 µg/l (normal <100 µg/l), an increased urinary lead concentration of 385 µg/l (normal <15 µg/l). The increased urinary excretion of δ-aminolaevulinic acid of 200 mg/l (normal <6 mg/l) indicated an earlier much higher blood lead concentration. Also, slightly increased urinary concentrations of arsenic 24.1 µg/l (normal < 20 μ g/l) and silver 1.3 μ g/l (normal

<0.5 µg/l) was found, whereas the urinary concentrations of mercury, chromium, manganese, antimony, and cadmium were within normal limits. The lead content of the metallic paste was 238 µg/g. The amount of lead in this paste alone does not seem to be sufficient for the high blood concentrations found in this patient. Therefore we assumed that the patient additionally ingested lead with the other ayurvedic drugs which he had taken up to a few days before admission. Unfortunately, these drugs were used up and could not be recovered for toxicological investigations. There was no indication of an exposure to occupational or other known sources of lead, including drugs.

After the diagnosis was established, chelation treatment with D-penicillamine was given. Urinary excretion of δ-aminolaevulinic acid dropped after 4 weeks to the normal range and the haemoglobin increased to 119 g/l. After 4 months of follow up, the blood lead concentration was still slightly increased at 180 µg/l.

The patient was treated on an outpatient basis and did not require any blood transfusions. As an interesting side effect, there was a stable reduction of the liver enzymes AST and y-GT to the normal range and of ALT to 34 U/l during chelation treatment.

Discussion

This case shows the risk of lead intoxication through ingestion of ayurvedic drugs. The classic clinical symptoms of lead intoxication in adults are abdominal pain, anaemia, renal disease, headache, ataxia, memory loss, and peripheral neuropathy. The predominant symptoms in this patient were anaemia together with non-specific constitutional symptoms. As well as a detailed history, the basophilic stippling of red blood cells was the leading sign toward the correct diagnosis.

Although basophilic stippling has been described as a non-specific finding in various diseases,¹ lead intoxication should always be suspected.

So far, there have only been a few reports published about lead or other heavy metal intoxication by ingesting health food or herbal drugs.²⁻⁸ Most of these cases have been diagnosed in members of Asian ethnic groups, but intoxication in Mexican-Americans has also been reported.3 However, even where lead or other heavy metals are not a usual component of the traditional medicine, accidental contamination or illegal criminal production of faked drugs could be a potential cause of high heavy metal concentrations in these drugs.8 Therefore, physicians should be aware that with growing international travel and rising self medication with drugs from uncontrolled sources the risk of drug induced poisoning could increase in the future, especially in patients who are normally not expected to consume traditional remedies.

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