CASE REPORT

Pain in abdomen - do not forget lead poisoning

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Lead toxicity has been recognized for thousands of years, and is still around. We encountered 11 patients with lead toxicity in the last two years. All patients had presented with diffuse pain in the abdomen, anemia and mild derangements of liver biochemistry. History of intake of indigenous or herbal medicine for diabetes mellitus or psychosexual disorders was present in eight patients. All of them had elevated blood lead levels. Abdominal pain responded promptly to treatment with chelating agents. [Indian J Gastroenterol 2002;21:225-226]

Key word: Plumbism

Lead occurs naturally in the earth's crust. When ingested, inhaled or absorbed through the skin, it is highly injurious to humans. Lead toxicity (plumbism), which has been recognized for thousands of years, is still one of the leading environmental diseases among children. Developed countries have various prevention programs, like a ban on lead-based house paints and lead soldering for food cans, and use of leaded gasoline. However, in developing countries like India, we still come across this silent disease. We report 11 patients with lead poisoning seen in the last two years.

Case Reports

All our patients presented with diffuse crampy pain in the abdomen, which was moderate to severe in intensity and associated with poor appetite, weakness and constipation. There was significant weight loss (>10% of original body weight) in 5 patients.

One patient presented with aggressive, abusive behavior, hallucinations and incontinence of urine with no motor weakness. CT scan of the head and cerebrospinal fluid examination were normal in him. The diagnosis was established on the basis of high blood lead levels (203 μg/dL). Another patient had twitching over both lower and upper limbs. Electromyography of both gastrocnemius muscles showed fasciculations that appeared as couplets and triplets. There were polyphasic long-duration motor units with decreased recruitment on voluntary contractions. Needle EMG of bilateral tibialis anterior, quadriceps, left adductor pollicis brevis and flexor digitorum indicus muscles showed similar changes, while that of the left deltoid was normal. H reflex and sural sensory nerve conduction velocities were normal bilaterally. Nerve conduction studies were also normal in bilateral peroneal, tibial, median and ulnar nerves. These electrophysiological findings favored continuous muscle fiber activity suggesting Isaac's syndrome.

General examination of these patients revealed pallor in eight and mild icterus in six. Abdominal examination revealed a soft abdomen with no palpable organomegaly and no peritoneal signs in all except in one patient who had tenderness in the epigastrium. This patient was found to have biochemical evidence of pancreatitis (serum amylase 1810 U/L, serum lipase 2120 U/L). He had no history of alcohol intake. USG and CT scan abdomen revealed no gallstones and serum calcium, triglycerides and arterial blood gases were normal. Serum lead levels were high and response of pain to D-penicillamine was prompt.

All these patients were interviewed in detail regarding drug intake, particularly herbal medicines, and occupational history. The details of clinical profile, possible source of lead exposure and blood lead levels are shown in the Table. Blood tests in all patients were remarkable for low hemoglobin and MCV (74-101 fL), and mild elevation of bilirubin and transaminases. Four patients had basophilic stippling on peripheral blood film. Blood lead levels were done by atomic absorption spectrometry.

All patients were treated with D-penicillamine for 3 months, as this was the only drug available to us. Symptomatic improvement occurred within 4-7 days. Repeat blood lead levels done after 3 months in 6 patients were <14 μg/dL. At 3 months, hemogram and liver function tests were normal in all patients.

Discussion

Worldwide, the major sources of lead exposure include home paint, airborne emissions from incinerators and industries, water (old lead pipes, plumbing fittings) and heavy industrial pollutants. Lead can also be found in herbal medicines, health food and cosmetics like eye liners.1-2 The major toxic effects of lead are on the bone marrow, and gastrointestinal and nervous systems.3-5 Symptoms of lead poisoning vary from non-specific symptoms like lethargy, appetite loss, headache and occasional vomiting to severe neurological symptoms like encephalopathy complicated by seizures and coma. Abdominal pain, which occurs in lead poisoning, is diffuse and crampy; its pathogenesis remains unknown.

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One of our patients had evidence of mild pancreatitis with high serum lead levels. No other etiological factor could be identified and he responded to D-penicillamine. We believe this to be the first postulated case of acute pancreatitis with lead toxicity.

In children chronic exposure to lead even in small amounts can result in learning or behavioral problem. Peripheral nervous system involvement with wrist and foot drop occurs more commonly in adults. Isaac’s syndrome has not been previously reported in relation to lead toxicity.

Lead interferes with a variety of red cell enzyme systems leading to anemia which is frequently microcytic but can be normochronic and normocytic. Basophilic stippling is not specific for lead poisoning and has also been described in B12 deficiency, pyrimidine nucleotide deficiency and thalassemia.

In adults, symptomatic lead poisoning usually develops when blood levels exceed 80 μg/dL, but symptoms can develop at lower levels and with brief period of exposure also. Lead toxicity is treated with chelating agents like disodium acetate calcium, dimercaprol or D-penicillamine. Response to D-penicillamine is prompt. Abdominal pain disappeared in all patients and none had recurrence of pain. One patient who had presented with psychosis also responded completely in 5 days, though he needed anti-psychotic drugs in addition. We could withdraw anti-psychotic drugs after 2 months. The patient with Isaac’s syndrome also showed gradual improvement in muscle twichings. As symptoms may recur due to mobilization of lead from bones, treatment with chelating agents should be continued for a few months.

These cases highlight that lead poisoning should be considered in patients with recurrent abdominal pain but no abdominal physical signs. History of herbal medicines should raise suspicion. Early diagnosis can save patients from unnecessary investigations and permits start of treatment on time.

References


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