

Lead poisoning from an Asian Indian folk remedy

A. Hugh Pontifex, BA, MD, FRCPC
Arun K. Garg, MD, PhD, FRCPC

Lead poisoning, resulting chiefly from the ingestion of lead salts, has been a problem throughout history.¹ In spite of our awareness of the danger, sporadic cases of lead poisoning still occur, most as a result of the ingestion of lead salts.

Children absorb a higher proportion of ingested lead than do adults. Some common sources include paint, putty, dirt and newsprint. Metallic lead, as in fishing weights and bullets, can also induce poisoning. Other sources are food and drink stored in improperly glazed pottery and alcohol cooled in lead condensers, such as car radiators. Inhalation of vapourized lead is chiefly an occupational hazard in welders cutting lead-painted metals, workers in battery reclamation plants and painters burning paint off old houses.

A recently reported additional source is lead-containing folk remedies.³⁻⁵ We report a recent case of lead poisoning resulting from ingestion of such a remedy.

Case report

A 35-year-old Asian Indian man presented with weakness, malaise, nausea and abdominal pain. He was known to have diabetes and had no known industrial exposure to lead. The patient had recently returned from a trip to India that had lasted several months. For 6 weeks before admission he had been taking a nonprescription medication that he had acquired in India.

A physical examination gave unremarkable results. The following abnormal laboratory results were found: hemoglobin concentration 103 g/L, reticulocyte count $172 \times 10^9/L$, coarse basophilic stippling of the erythrocytes and presence of circulating nucleated erythrocytes. Bone marrow aspiration showed numerous sideroblasts and erythroid hyperplasia.

Specific tests for lead poisoning gave the following results: free-erythrocyte protoporphyrin level 317 (normally 22 to 87) $\mu\text{g}/\text{dL}$ (5.61 [normally

0.39 to 1.54] $\mu\text{mol}/\text{L}$), blood lead level 151 (normally less than 40) $\mu\text{g}/\text{dL}$ (7.29 [normally less than 1.93] $\mu\text{mol}/\text{L}$) and lead level in a 24-hour collection of urine 1925 μg (normally less than 80 μg in nonindustrial populations and less than 120 μg in those with industrial exposure) (9.29 [normally less than 0.39 and 0.58 respectively] μmol).

The nonprescription medication used by the patient was found to contain 8 mg of lead per gram of substance. It was derived from ground seeds and roots and is a traditional remedy for diabetes in ayurvedic medicine. The patient was taking 1 teaspoon per day, or approximately 40 mg/d of elemental lead;⁶ accumulation and toxic effects occur when more than 0.5 mg/d is absorbed.

The patient was treated with penicillamine, and his blood lead level, hemoglobin concentration and other levels returned to normal. At the time of writing he was well.

Comments

In addition to this case, we have recently seen a second Indian immigrant with lead poisoning. The patient, a housewife, was admitted with cramping abdominal pain and was found to have a hemoglobin concentration of 80 g/L and coarse basophilic stippling of the erythrocytes. Her blood lead level was 164 $\mu\text{g}/\text{dL}$ (7.91 $\mu\text{mol}/\text{L}$). She had no history of exposure to lead but had been taking a nonprescription medication for hemorrhoids that her husband had obtained in India. Unfortunately the medication had been discarded, so proof of the source of lead was not possible. No chelating agent was prescribed. Although her symptoms vanished, her blood lead level declined slowly; at last report it was 60 $\mu\text{g}/\text{dL}$ (2.90 $\mu\text{mol}/\text{L}$).

Lead poisoning resulting from ingestion of folk medicines has been reported in several immigrant groups, mostly in children. In Mexicans it has resulted from the use of *azarcón*,^{3,4,7} which contains lead tetroxide. Hmong refugees from northern Laos have acquired lead poisoning from *pay-loo-ah*.⁵ A fatal case of lead intoxication was reported in a 9-month-old Asian Indian boy in Florida.⁸ The confirmed case and the probable case that we report are, to our knowledge, the first in adult Asian Indian immigrants to Canada. They are probably not isolated incidents, and physicians

From the Department of Pathology, Surrey Memorial Hospital, Surrey, BC

Reprint requests to: Dr. A. Hugh Pontifex, PO Box 90, New Westminster, BC V3L 4X9

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caring for immigrants should be aware of this source of lead poisoning.

Perhaps more important is that, in children, lead poisoning may be asymptomatic or the symptoms may be very subtle. They may include behaviour disorders such as distractibility, impulsiveness and hyperkinetic behaviour. Garrettson² has suggested that screening for lead toxicity be undertaken in all children with pica, anemia, hyperkinetic behaviour or neurologic disease, including seizures, and in all children living in houses built before 1940. To these categories should possibly be added children from immigrant groups known to use folk medicines, which may be either prepared at home or obtained outside Canada without prescription from a physician.

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Q-fever endocarditis

Raphael Saginur, MD
Stephen S. Silver, MD
Richard Bonin, MD
Maureen Carlier, MD
Manuel Orizaga, MD

Q-fever, caused by *Coxiella burnetii*, is an unusual cause of culture-negative endocarditis in Canada. The following is the first reported case in this country in which hepatomegaly occurred.

Case report

Clinical course

A 57-year-old man was admitted to the Ottawa Civic Hospital May 2, 1983 with a 2-month history of intermittent fever. He had been given oral ampicillin therapy for the 4 days preceding admission.

He had a remote history of tuberculosis of the spine and a heart murmur. While in Saudi Arabia from 1978 to 1980 he had suffered a myocardial infarction and several brief episodes of fever.

From the departments of Medicine, Microbiology and Pathology, Ottawa Civic Hospital, University of Ottawa

Reprint requests to: Dr. Raphael Saginur, Infectious Disease Service, Ottawa Civic Hospital, 1053 Carling Ave., Ottawa, Ont. K1Y 4E9

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Goats and sheep abounded in his neighbourhood in Riyadh. Nine months before this admission he had had an influenza-like illness, treated with orally administered penicillin. Five months later he was admitted to hospital with congestive heart failure and florid aortic regurgitation. Surgery revealed that the aortic valve was bicuspid and the anterior cusp perforated, consistent with endocarditis; microscopic examination was not done. An Ionescu-Shiley valve was inserted. The postoperative course was complicated by cardiac tamponade and gastrointestinal hemorrhage.

At the time of the current admission the patient's temperature was 38°C, blood pressure 130/90 mm Hg, pulse rate 100 beats/min and respiratory rate 20/min. The jugular venous pressure was 6 to 7 cm of blood. The point of maximum impulse of the left ventricle was diffuse, and a systolic ejection murmur was noted at the left sternal border. The liver span was 16 cm. Mild pitting edema of the ankles, clubbing of all fingers and a splinter hemorrhage in the left forefinger were noted. An electrocardiogram revealed first-degree atrioventricular block and left bundle branch block. Ten blood samples were obtained for culture; all gave negative results.

A week after admission a faint diastolic blow-