Lead poisoning—an aetiology not to be missed

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Lead is ubiquitous in the environment. Workers in industries such as plumbing, mining, and manufacturing of metals, plastics, or batteries are at risk of exposure through inhalation of lead-containing dust or fumes. Lead has also been used in many consumer products, such as gasoline, paints, cosmetics, crayons, ceramic glaze, cookware, and toys. Children with the eating disorder pica are at risk of excessive lead ingestion.

In Hong Kong, historical reports of lead poisoning include an outbreak among Gurkha soldiers exposed to chili powder contaminated with lead chromate, fisherman and their families who ingested excessive lead while repairing nets contaminated by lead weights, consumers of traditional Chinese medicine or processed herbs contaminated by lead grinders and pans used in the pulverisation process.[1,2] There has also been a case of poisoning due to chronic consumption of ashes from a Chinese talisman bearing writing in lead tetroxide.[3] Although lead poisoning is uncommon in Hong Kong, clinicians should still have a high index of suspicion, as lead poisoning has a wide range of clinical manifestations and may present to different specialities.

Our neurology team recently encountered a 60-year-old man who presented with generalised weakness in all four limbs and anaemia who later found to have severe lead poisoning. The patient enjoyed good past health until January 2017, when he reported experiencing dizziness and a tingling sensation in his extremities. When admitted to North District Hospital, Hong Kong, on 29 July 2017, the patient was pale and his speech was confused. His symptoms included bilateral facial palsy, weak voice dysphonia, dysphagia, dysarthria, generalised muscle wasting, hypotonia, and areflexia without muscle fasciculation. There was no apparent sensory-level or sphincter disturbance.

The patient's signs and symptoms were suggestive of progressive generalised polyneuropathies with bulbar involvement. Comprehensive investigations were performed to elucidate the aetiology. On 9 August 2017, the patient's blood lead level (BLL) was recorded as markedly elevated to 189 μg/dL, confirming severe lead poisoning presenting with anaemia, significant sensorimotor axonal polyneuropathy, and encephalopathy.

The patient was transferred to Toxicology Centre of Prince of Wales Hospital, Hong Kong, where treatment with 2,3-dimercaptosuccinic acid (DMSA) were initiated. Treatment was changed to calcium disodium edetate ($CaNa_2EDTA$) with dimercaprol later for suspected lead encephalopathy. After treatment, the patient's BLL dropped to 26.25 μg/dL. Haemoglobin improved to around 10 g/dL. The patient's facial weakness, speech, and limb power improved. The patient has regular follow-ups and repeated courses of DMSA; at his latest review, in February 2018, his BLL was still elevated to 48.4 μg/dL.

Because of increasing migration to and from mainland China, clinicians in Hong Kong should be aware that patients may acquire lead poisoning in mainland. According to a large case series of childhood lead poisoning in China published in 2017, industrial lead pollution (43.1%) and folk medicines (41.4%) account for the main source of lead exposure.[4] Our patient denied using traditional Chinese medicine, over-the-counter medication, or recreational drugs. He had worked as renovation worker in mainland China for decades. Therefore, we postulate that the patient's lead poisoning was due to contact with lead-containing paint.

Acute high-level lead exposure can lead to encephalopathy, coma, ataxia, seizure, colicky abdominal pain, nausea, and vomiting. Acute renal tubular dysfunction presenting with Fanconi-type syndrome (such as glucosuria, aminoaciduria, phosphaturia) has been observed in children.[5]

Chronic lead poisoning causes decline in cognitive function and neurobehavioural problems such as depression and irritability. Children are particularly susceptible, owing to an incomplete blood brain barrier. Chronic lead poisoning in children can result in lower intelligence quotient, attention deficit hyperactivity disorder, and impaired learning, memory, speech, and hearing development.[6] Mothers may transfer lead to their developing fetus through the placenta, or to breastfeeding infants through breast milk. There is no absolute safe BLL. The Centers for Disease Control and Prevention has lowered the toxic BLL from >25 μg/dL in 1985 to >5 μg/dL in 2010, which correspond to 97.5th percentile of BLL in children of United States.[7]

Chronic lead poisoning also causes neuropathies. Classical lead neuropathy predominantly involves wrist and finger extensors, resembling radial nerve palsy. This type of motor neuropathy is more likely to develop following relatively short-term exposure to high lead concentrations and evolves in a subacute fashion. The mechanism proposed is lead-induced porphyria rather than its direct neurotoxic effect. Shobha et al[8] described five patients with lead-related radial neuropathy. All patients had normal motor conduction velocities, with some demonstrating reduced compound muscle action potential or mild reduction of sensory nerve action potential. There is another type of neuropathy with distally accentuated sensorimotor and autonomic involvement, which evolves more slowly after many years of exposure. Nerve conduction studies in these patients have demonstrated normal motor conduction velocities and compound muscle action potential amplitudes, but prolonged distal motor and sensory latencies.[8-10] Lead poisoning can also cause electromyographic abnormalities such as denervation activity and polyphasic motor unit potentials, mimicking motor neuron disease.[11]

Chronic lead poisoning also causes anaemia through increased haemolysis and inhibition of enzymes, such as delta-aminolevulinic acid dehydratase and ferrochelatase, that are necessary for haem biosynthesis. The metabolic block results in increased urinary delta-aminolevulinic acid, coproporphyrin, and erythrocyte zinc protoporphyrin. Basophilic stippling (caused by inhibition of pyrimidine 5'-nucleotidase and ribosomes aggregates) and ring sideroblasts may be found.[12]

Finally, chronic lead exposure causes chronic interstitial nephritis, increased risk of hypertension, cardiovascular disease, and cancer.

Acute lead toxicity is confirmed by elevated BLL. Chronic lead toxicity can be established by measuring red cell zinc porphobilinogen and by X-ray fluoroscopy of bone.[13] Abdominal radiography may demonstrate lead chips in the bowels of children with pica.

Management of lead poisoning includes identification of source of exposure, screening of household members, and determination on the need for chelation therapy. Chelation agents include DMSA, CaNa2EDTA, and dimercaprol. Adverse effects of these treatments include rash, neutropenia, liver derangement, gastrointestinal upset, and haemolysis in glucose-6-phosphate-dehydrogenase deficiency, essential mineral loss, renal failure, and lead encephalopathy. Whole bowel irrigation is needed if abdominal radiography demonstrates lead-containing foreign bodies. Treatment of underlying nutritional deficiencies, such as iron and calcium deficiencies, helps to reduce lead absorption.[14]

In conclusion, although legislation controlling environmental lead exposure has resulted in declining numbers of lead poisoning cases in most developed countries, clinicians should remain aware of this diagnosis in patients with both haematological and neurological manifestations.

Author contributions

All authors contributed to the concept and design of the study, acquisition of data, and interpretation of data, and critical revision of the manuscript for important intellectual content. MF Ip drafted the manuscript.

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Declaration

All authors have disclosed no conflicts of interest. All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

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Answers to CME Programme
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I. Systemic lupus erythematosus: what should family physicians know in 2018?

A	1. True	2. False	3. False	4. False	5. True
B	1. False	2. True	3. False	4. True	5. False

Hong Kong Med J 2018;24:521–6

II. Recommendations on prevention and screening for colorectal cancer in Hong Kong

A	1. False	2. False	3. False	4. True	5. True
B	1. True	2. True	3. True	4. True	5. True