Lead Poisoning – ACC Review Issue 16 (March 2005)

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ACC - Prevention, Care, Recovery

For Providers

Clinical best practice

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General points

Lead is a bio-accumulative toxin and prolonged and/or heavy exposure can give rise to a wide range of adverse health effects

Diagnostic criteria include clinical features and/or evidence of end organ damage in association with elevated blood lead level…

Risks to the foetus may occur at lower blood levels than those associated with other adverse effects. Care is required to minimise exposure during pregnancy

Treatment includes removing the source of excess exposure, plus chelating agents for certain specific toxic effects and/or particularly high blood levels

Lead poisoning is a serious-harm illness of occupation and is notifiable under the Health and Safety in Employment Act 1992.

Background

Lead is bio-accumulative, with a half-life of about 30 days in blood and soft tissues, but is released only very slowly from bone. It causes dose-related dysfunction of the nervous, hematopoietic, gastro-intestinal, renal, endocrine and musculoskeletal systems. In New Zealand, normal levels in the general population can range up to 0.50 umol/L [0.50 micromoles of lead per litre of blood is equivalent to 10.36 micrograms/decilitre or 10.36 ug/dL] in men, 0.35 umol/L [7.25 ug/dL] in women and 0.55 umol/L [11.40 ug/dL] in children.

Blood levels of 0.72 umol/L (15 ug/dL) or more are notifiable to the Ministry of Health under Section B, Second Schedule of the Health Act 1956. Occupational lead poisoning is notifiable under section 25 of the Health and Safety in Employment Act 1992, at whole blood levels of 2.6 umol/L (53ug/dL).

Prevalence and risk

In New Zealand in 2003, 119 cases of lead poisoning were notified, of which 5 were hospitalised. Children (≤ 14 years) accounted for 21 cases. The next highest age-specific rate was 40–49 year olds. Most of the occupational exposures were for painters, builders, plasterers, sanders and foundry workers.1 Inhalation, especially of fumes from burning lead based paint, is the key mode of occupational exposure.

Adults absorb around 10% through ingestion and practically none through skin. … Other sources include lead dust (e.g. brought home on clothes), drinking from unglazed vessels or lead pipes.

Diagnosis and clinical features of poisoning

Multi-system signs and symptoms, albeit subtle, are a key diagnostic feature(2). Blood level is the most informative biomarker but not the sole diagnostic consideration given individual differences in susceptibility at different blood levels. In some adults, symptoms may manifest at blood lead levels of 1.95 umol/L (40 ug/dL), but in others at ≥ 3.40 umol/L (≥ 70 ug/dL)(3). The rate and duration of blood lead elevation can influence effects at any given level. Blood levels may indicate current exposure as well as long term release from bones. Hair analysis and post-chelation urinary levels may not be reliable tests for toxicity….

In adults, common symptoms include abdominal pain, fatigue, arthralgia, decreased libido, headache, irritability, impotence, depression, anorexia, muscle pain and/or weakness, change in bowel habits, weight loss and paresthesiae(5). Impaired short-term memory, concentration, reaction time, mood, verbal concept formation and visuospatial functions may appear at ≥ 1.95–2.45 umol/L (≥ 40–50 ug/dL). Slowed nerve conduction velocities (e.g. small motor fibres of the ulnar nerve) can occur around ~1.45–3.4 umol/L (~30–70 ug/dL) but peripheral neuropathy (muscle weakness with minimal sensory loss) is rare below 2.90 umol/L (<60 ug/dL). Overt neurological signs are not usual until levels exceed 2.90–3.90 umol/L (60-80 ug/dL) for several months. Neurological and gastrointestinal effects are often less marked in chronic poisoning.

Severe encephalopathy is rare under 4.85 umol/L (<100 ug/dL) but has been described in children at ~3.4 umol/L (~70 ug/dL). Mild to moderate anaemia has been found in 5% of adults with levels of 1.95–2.85 umol/L (40–59 ug/dL)(6).

However, frank anaemia does not usually develop until levels exceed 3.85 umol/L (>80 ug/dL) for a prolonged period.

Renal changes are not uncommon(7). Exposure for years, especially at levels ≥ 3.85 umol/L (≥ 80 ug/dL) increases the risk of chronic insufficiency but rarely progresses to renal failure. Decreased uric acid clearance occurs with the risk of 'saturnine' gout. There is some evidence to suggest that levels of 0.95–1.95 umol/L (20–40 ug/dL) may be associated with a rise in systolic blood pressure (0.5-3.0 mmHg).

Decreased sperm counts have been observed at 1.95 umol/L (40 ug/dL) and abnormal morphology and motility at mean levels of ~2.55 or 2.95 umol/L (~53 or 61 ug/dL). Decreased female fertility has been described mainly in the context of high exposure.

Significant associations exist between lead levels and pre-term birth, lower birth weight, reduced post-natal growth, increased incidence of minor congenital abnormalities,(8) and early deficits in post-natal neurological or neurobehavioral status. Mild foetal impairment may occur at maternal levels of 0.75–0.95 umol/L (15–20 ug/dL)(9)(10). There is little evidence of major congenital malformations.

Management

The key to managing chronic lead toxicity is to identify and remove the source of exposure. Clinical management involves treatment of life-threatening effects, minimising absorption, and enhanced elimination.

Activated charcoal does not bind lead. However, absorption may be reduced in some situations by timely gastric emptying procedures or whole bowel irrigation.

Chelation is recommended when blood levels reach 2.15 umol/L (45 ug/dL) in children or 3.40 umol/L (70 ug/dL) in adults, and/or where there is encephalopathy, neuropathy, anaemia, nephropathy, severe abdominal colic, arthralgia or myalgia…. In severe acute cases, hospitalisation and dimercaprol followed four hours later by calcium disodium edetate, for five days is favoured. Oral DMSA (dimercaptosuccinic acid) is the preferred oral therapy….

In the workplace, three successive levels of ≥ 2.6 umol/L (≥ 54 ug/dL) require suspension from work. A slightly lower suspension level of ≥ 2.4 umol/L (≥ 50 ug/dL) applies in Australia, with a return to work level of 1.93 umol/L (~40 ug/dL), with lower levels for females of reproductive capacity.

Issues relevant for ACC

Patients with a raised blood lead level and evidence of personal injury (anatomical derangement or functional impairment) as a result of their employment are eligible for cover. In the absence of personal injury, weekly-earnings compensation for time off work to reduce blood lead levels is unlikely to be payable, as there is no incapacity secondary to personal injury.

References

ESR. Notifiable and other diseases in New Zealand. Annual Report 2003. 2004; 24-25.

Allcott JV, et al. Acute lead poisoning in two users of illicit methamphetamine.J Am Med Assoc 1987; 258: 510-1.

Rempel D. The lead-exposed worker. J Am Med Assoc 1989;262:532

Baker NJ. A 13 year review of childhood lead poisoning in Christchurch and Nelson. N Z Med J 1995;108:249-51.

Cullen MR. Adult lead intoxication: presentation of 31 new cases and a review of recent advances in the literature. Medicine 1983;62:221-47.

Baker EL Jr, et al. Occupational lead poisoning in the United States: clinical and biochemical findings related to blood lead levels. Br J Ind Med 1979;36:314-22

Wedeen RP, et al. Occupational lead nephropathy. Am J Med 1975;59:630-41.

Needleman HL, et al. The relationship between prenatal exposure to lead and congenital anomalies. J Am Med Assoc 1984;251:2956-9.

Andrews KW, et al. Prenatal lead exposure in relation to gestational age and birth weight. A review of epidemiologic studies. Am J Ind Med 1994;26:13-32.

Bellinger D, et al. Longitudinal analyses of prenatal lead exposure and early cognitive development. N Eng J Med 1987;316:1037-43.

The environmental case management of lead exposed persons. Guidelines for Public Health Services. Ministry of Health; 1998.