Clinical Toxicology

Lead encephalopathy from an imported Toby mug

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Summary: Encephalopathy is an unusual manifestation of lead poisoning in an adult, the more common presentation being abdominal colic, anaemia and limb palsy.

We report a case of adult lead encephalopathy and describe the use of a simple screening test for lead poisoning together with the increasing number of cases associated with imported ceramics.

Introduction

In 1975, fifty cases of adult lead encephalopathy were reported, mostly secondary to drinking illicit whisky in the USA.¹ We report a case of lead encephalopathy which was associated with the use of a partially glazed drinking vessel.

Case report

A 33 year old female was admitted as an emergency after a single fit. In the 3 days prior to admission her behaviour had become abnormal, her speech slurred and she complained of headaches. She had been seen two months previously with a 10 day history of intermittent lower abdominal pain. A diagnosis of irritable bowel syndrome was made. A hypochromic microcytic anaemia was also noted and interpreted as indicating iron deficiency attributed to heavy periods. She was treated with iron tablets and discharged.

On examination she was stuporose but the central nervous system, including fundoscopy and pupillary reactions, was otherwise intact. The only abnormal finding was a distinct 'lead line' on both upper and lower gingival margins together with poor dental hygiene.

Haemoglobin concentration was 8.1 g/dl; MCV 75 fl; MCH 24 pg; MCHC 31 g/dl; reticulocytes 21% and the blood film showed marked basophilic stippling. Blood urea, electrolytes, glucose, liver function tests, paracetamol and salicylate levels, electrocardiogram, chest and skull X-ray were all normal.

Blood lead and urinary delta-amino laevulinic acid (ALA) were found to be grossly elevated: blood lead 12.7 $\mu\text{mol/l}$ (reference range: $<1.4 \mu\text{mol/l}$) and urine ALA 407.4 $\mu\text{mol/24 h}$ (reference range: 11.4–57.2 $\mu\text{mol/24 h}$). Blood and urinary lead were measured on a Perkin Elmer 107 atomic absorption spectrophotometer with a Delves cup micro-sampling assembly.² Urinary ALA was measured spectrophotometrically.³

The analytical performance of the blood lead method was monitored by participation in the UK External Quality Assessment Scheme for General Clinical Chemistry (UKEQAS). Samples received from the scheme were stored deep frozen after assay. These were re-used as assayed material after statistical analysis, as commercial controls were not readily available.

The linear regression equation between our results (y) and the overall national mean (x) was $y = (0.926x + 0.22) \mu\text{mol/l}$ ($n = 26$, $r = 0.97$, $\text{s.e.} = 0.16$). The inter-batch coefficient of variation (CV) was 4.8%; this value was derived using a s.d. from pairs formula from 15 UKEQAS samples, each of which was analysed on two separate occasions.

The clinical diagnosis of lead encephalopathy was supported by the very high blood lead levels. On the day of admission chelation treatment was commenced using calcium disodium edetate 40 mg/kg intravenously every 12 hours for 7 days and dimercaprol 150 mg intramuscularly 4-hourly for 3 days.⁴ Treatment was monitored by measuring blood lead levels and urinary ALA and lead concentration (Figure 1).

Within the first 24 hours there was a sharp fall in the blood lead level with a steady decline thereafter. After 48 hours of treatment the patient recovered consciousness but was confused and by the sixth day she

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Figure 1 Clinical and biochemical response in a patient with lead poisoning.

was fully orientated (Figure 1). She was discharged 15 days after admission on oral penicillamine maintenance treatment at 750 mg/day in divided doses. This had to be stopped and recommenced at a lower dose after the patient developed a skin rash.

The lead source was investigated before discharge and the environmental health team were contacted. For four months prior to hospital admission the patient had been regularly drinking cider from a partially lead glazed mug made in Italy in 1976 (Figure 2). On later testing a lead level of 130 mg/l was recorded after leaving cider in the mug for a 12 hour period, the European Economic Community permitted maximum level being 50 μg/l.

Discussion

Lead encephalopathy is a rare presentation of lead poisoning in adults. Between 1959 and 1972 there were 31 well documented cases in the English language literature.⁵ Most cases seen in the USA were secondary to gasoline sniffing and consumption of 'moonshine' whisky. The most prominent symptoms reported were reduced alertness, memory deficits, mood changes and altered perception. Mania, delirium and, in 80% of patients, fits also occurred. Acute encephalopathy is a more common presentation in children and can occur at blood lead concentrations lower than those required to cause encephalopathy in adults.⁶

In the USA an increasing number of cases of lead poisoning are being described associated with imported ceramic pottery particularly from Mexico, China and Italy.^{7,8}

Although in the USA, as in this country, regulations apply to the manufacture of pottery as well as to

importing pottery, there are no rules concerning personal use.⁹ The Consumer Protection Act 1987 and General Safety Requirements both include lead glaze recommendations but the onus is on the supplier to ensure the goods are safe.

A few reports implicate goods purchased on the retail market but an increasing number of reports have been concerned with items bought by visitors rather than commercial imports.¹⁰ One case report demonstrated that the pottery became progressively more hazardous as it aged and was subject to repeated washings and scourings.¹¹ Coffee with only mild acidity (pH 5.1) has been shown to leach large amounts of lead from a partially glazed cup.¹²

The majority of the cases associated with ceramics present with a variety of non-specific symptoms including abdominal cramps, arthralgia, mild anaemia, fatigue, anorexia and nausea. Our patient was unusual in that encephalopathy resulted from pottery related lead poisoning.

In all patients presenting with abdominal colic and anaemia a diagnosis of lead poisoning should be

Figure 2 The partially glazed Toby mug.

considered. The presence of basophilic stippling on the blood film should be sought although this is not diagnostic and lead levels will confirm the diagnosis. Urinary delta-amino laevulinic acid (ALA), which can be measured spectrophotometrically, may serve as a simple screening test. If urinary ALA is raised a blood lead level would be mandatory and other causes of elevated ALA including the porphyrias, hereditary coproporphyrinuria and excess alcohol should be excluded.

ALA is a precursor in haemosynthesis and urinary ALA increases as the haem synthetic chain is interrupted by lead which successfully competes with zinc for binding to the sulphhydryl group near the active site of the enzyme ALA dehydratase. The urine ALA levels rapidly return to normal after lead exposure discontinues.

The recommended treatment for lead encephalopathy involved chelation with intramuscular dimercaprol and intravenous calcium disodium edetate followed by oral penicillamine at 500–750 mg/day for a period of 1–6 months until the blood lead concentration is $<1.4 \mu\text{mol/l}$.⁴ Recent reports suggest

that dimercaptosuccinic acid, a water-soluble derivative of dimercaprol first evaluated in China 20 years ago, might be preferable as it can be given orally. Additional advantages include a higher chelating power, enhanced renal excretion of lead, fewer side effects and little or no effect on elimination of iron, magnesium or calcium compared with the other treatments.^{13,14}

In conclusion, the diagnosis of lead poisoning can easily be overlooked in view of the non-specific symptoms and signs. With travel making available previously inaccessible areas, and the lack of definitive warnings concerning some personal items, the risks of toxicity are increased. The diagnosis should be considered carefully and testing the urine can be used as a simple screen.

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References

1. Zenz, C. *Occupational Medicine*. Year Book Medical Publication, Chicago, 1975.
2. Delves, H.T. A micro-sampling method for the rapid determination of lead in blood by atomic absorption spectrophotometry. *Analyst* 1970, **95**: 431–438.
3. Tomokini, K. & Ogata, M. Simple method for determination of urinary aminolaevulinic acid as an index of lead exposure. *Clin Chem* 1972, **18**: 1534–1536.
4. Ibels, L.S. & Pollock, C.A. Lead intoxication. *Medical Toxicology*. 1986; Nov–Dec 1: 387–410 ADIS Press Limited.
5. Whitfield, C.L., Chien, L.T. & Whitehead, J.D. Lead encephalopathy in adults. *Am J Med* 1972, **52**: 289–298.
6. Chisolm, J.J. The use of chelating agents in the treatment of acute and chronic lead intoxication in children. *J Pediatr* 1968, **73**: 1–38.
7. de Rosa, E., Brighenti, F., Rossi, A., Curoidi, S., Gori, G.P. & Chiesura, P. The ceramics industry and lead poisoning. *Scand J Work Environment Health* 1980, **6**: 306–311.
8. Perrelli, G., Capellaro, E., Pira, E., Maina, G. & Vergnano, P. Further cases of lead poisoning from wine. *Am J Ind Med* 1984, **5**: 377–381.
9. Johnson, B.L. & Mason, R.W. A review of public health regulations on lead. *Neurotoxicology* 1984, **5**: 1–22.
10. Dunea, G. Letter from Chicago. Neuromyoclonic. *Br Med J* 1987, **295**: 1469–1470.
11. Wallace, D.M., Kalman, D.A. & Bird, T.D. Hazardous lead release from glazed dinnerware: a cautionary role. In: *The Science of the Total Environment*, Vol. 44: Elsevier Science Publishers B.B., Amsterdam, 1985, pp. 289–292.
12. U.S. Department of Health, Education & Welfare FDA Consumer Memo 'Glazes & Decals on Dinnerware', 1979.
13. Aposhian, H.V. DMSA and DMPS – Water soluble antidotes for heavy metal poisoning. *Am Rev Pharm Toxicol* 1983, **23**: 193–215.
14. Bentur, Y., Brook, J.G., Behar, R. & Taitelman, U. Meso-2, 3-Dimercaptosuccinic acid in the diagnosis and treatment of lead poisoning. *Clin Toxicol* 1987, **25**: 39–51.