Diverse Neurological Manifestations of Lead Encephalopathy

I. SEGAL, D. SAFFER, F. SEGAL

SUMMARY

Three patients with lead encephalopathy due to industrial poisoning are presented. They all showed a wide spectrum of neurological manifestations, which mimic other neurological presentations. It is emphasised that lead poisoning still occurs in industry, despite efforts at prevention.

S. Afr. Med. J., **48**, 1721 (1974).

Lead is one of the oldest metals to be used by mankind. Lead pipes were used even before Roman times, and in 1285 the first cistern of lead was constructed in the City of London; King Henry III 'in the twenty-first year of his reign' granted permission to the citizens of London to convey water from the Town of Teybourne by pipes of lead into their city.[1,2]

Lead is a soft, bluish-grey metal, heavy, malleable and ductile. Metallic lead is not dangerous to handle, but when it is exposed to air it readily becomes coated with a film of oxide. This creates dust which is inhaled by workers who shovel scrap lead. Lead readily volatilises at high temperatures, and all lead-smelting, melting and burning jobs are hazardous.

Lead has many industrial uses, for example: the manufacture of pipes, cisterns, roof coverings; the manufacture of lead accumulator plates and storage batteries; ship-building and ship-breaking; plumbing, painting and soldering. It is used in the chemical industry in containers for sulphuric acid, and for evaporation pans. In the pottery industry it is used as a low-solubility lead glaze. In the printing industry it is important in the manufacture of linotype metals.

It is the impression of industrial physicians that lead intoxication is now infrequent. Whitfield *et al.*[3] found only one recorded case of lead encephalopathy in the USA since 1960 and stated, 'Obviously this does not mean that this does not occur more often'. Nevertheless, cases of industrial lead poisoning do occur, despite modern public health directives. The following 3 case reports reflect its occurrence in South African adults and illustrate the hazards of specific occupations, and the profound and dramatic clinical presentation, which may mimic many other neurological disturbances.

Department of Medicine, Baragwanath Hospital and University of the Witwatersrand, Johannesburg

I. SEGAL, M.B. B.CH., M.R.C.P.
D. SAFFER, M.B. B.CH., M.R.C.P.
F. SEGAL, M.D.

Date received: 17 October 1973.

CASE REPORTS

Case 1

A 46-year-old man, employed in a factory manufacturing batteries, was admitted to hospital complaining of tremor of the arms and legs, and cramp-like abdominal pains, of 4 weeks' duration. He had been in his present employment for 4 years, but often neglected to wear his face mask when handling white lead oxide.

Mild pallor and a gingival lead line were present. Central nervous system examination showed a coarse tremor of his tongue, and marked intention tremor of his arms and legs. Dysdiadokokinesis and a wide-based ataxic gait were observed. There was global weakness of his muscles, with no obvious wasting or fasciculation. The rest of the physical examination was essentially negative.

Laboratory investigations: The cerebrospinal fluid pressure was normal; with neutrophils 6/mm^3; lymphocytes 3/mm^3; protein, 88 mg/100 ml; sugar 74 mg/100 ml; and chlorides 124 mEq/L.

The haemoglobin concentration was 11,8 g/100 ml, the haematocrit 39%, and the MCHC was 30%. The leucocyte count was 5 500/mm^3, with 47% neutrophils, 2% monocytes, 46% lymphocytes, and 5% eosinophils, and a normal platelet count. Blood urea was 62 mg/100 ml. serum sodium was 142 mEq/L; potassium 5,1 mEq/L; CO_2 content 24,4 mEq/L; and chlorides 102 mEq/L.

The urinary ALA was found to be 16,4 mg/L (maximum normal 10 mg/L), while the blood lead level was 90 μg/100 ml (maximum normal 70 μg/100 ml).

The patient was given penicillamine in a dosage of 0,5 g every 6 hours, and his blood lead level dropped to 87 μg/100 ml after the first 24 hours, and after 48 hours to 72 μg/100 ml. The urinary ALA was 13,2 mg/L. The urinary lead level before therapy was 16 μg/L, and after 24 hours' therapy, it was 1 005 μg/L.

The patient was discharged from hospital two months after admission, much improved, but still with mild cerebellar signs.

Case 2

A 22-year-old woman, employed in the printing industry, had a fit and was admitted to hospital. There was no previous history of epilepsy or of head injury, and the family history was normal. She was drowsy, but well-orientated for time and place. Except for a gingival lead line, nothing abnormal was found on examination. On direct questioning, there had been no other contact with lead, other than at her place of employment.

Laboratory investigations: The haemoglobin concentration was 8,2 g/100 ml. The leucocyte count was 21 100/mm³, with 85% neutrophils, 1% monocytes, and 14% lymphocytes. Punctate basophilia was observed in the red blood cells.

Blood urea was 45 mg/100 ml; serum potassium was 4,2 mEq/L; sodium 137 mEq/L; CO_2 content 14 mEq/L; and chlorides 110 mEq/L.

Lumbar puncture yielded no cells in the cerebrospinal fluid, which contained protein 80 mg/100 ml; sugar 47 mg/100 ml; and chlorides 730 mg/100 ml (as NaCl).

The blood lead level was 180 μg/100 ml, and the urinary ALA 8 mg/L. After therapy urinary lead was 1 004 μg/L.

Four days after admission the patient became confused and restless and developed bilateral VIth nerve palsies and papilloedema. A course of EDTA therapy was commenced, and her confusion and restlessness abated in a few days, the VIth nerve palsies disappearing by the tenth day. The papilloedema, however, persisted, and progressed to optic atrophy.

Four courses of EDTA therapy were given during the patient's stay in hospital. The haemoglobin and blood urea returned to normal levels before she was discharged from hospital.

Case 3

A 29-year-old man, employed in a battery factory, was admitted to hospital with the history that during that day he had had four attacks of epilepsy. There was no previous history of epilepsy, nor of head injury.

He was confused and restless, with nystagmus to the left and tonic conjugate deviation of the eyes to the left. There was no neck stiffness. A small retinal haemorrhage was present on the right side. No other abnormal central nervous system signs were observed. The blood pressure was 220/130 mmHg, the pulse 96/min and regular, and the respiratory rate 18/min and regular. The temperature was normal. There was no clinical cardiomegaly and the heart sounds were normal, as was the rest of the physical examination.

Treatment with antihypertensive agents was commenced.

Laboratory investigations: Lumbar puncture showed normal pressure; neutrophils were 3/mm³; lymphocytes 1/mm³; protein 54 mg/100 ml; and chlorides 119 mEq/L.

The haemoglobin concentration was 13,9 g/100 ml. The leucocyte count was 14 400/mm³, with 82% neutrophils, 4% monocytes and 14% lymphocytes. Marked punctate basophilia was observed in the red cells, and also moderate anisocytosis, polychromasia, elliptocytosis and fragmentation.

Blood urea was 60 mg/100 ml; serum potassium 4,3 mEq/L; serum sodium 148 mEq/L; and serum chloride 102 mEq/L.

The blood lead level was 316 μg/100 ml, while urinary lead was 152 μg/L (maximum acceptable is 150 μg/L). Urinary ALA was 85 mg/L.

The blood pressure dropped to 130/80 mmHg 2 days after admission and antihypertensive therapy was stopped. A course of intravenous EDTA therapy was commenced, and the patient improved dramatically during the next few days. His mental state returned to normal, and his blood lead level dropped to 106 μg/100 ml. The urinary lead level was 280 μg/L and urinary ALA level 73 mg/L. A further course of EDTA therapy was given, and he was discharged from hospital.

DISCUSSION

The cardinal features of lead poisoning include the lead or Burtonian line on the gingiva, abdominal colic, peripheral neuritis, basophilic stippling of the erythrocytes, anaemia, pronounced urinary excretion of coproporphyrin III and of delta-aminolaevulinic acid (ALA).

In children, a common form of exposure to plumbism is the ingestion of lead-containing paint. The nervous system involvement includes a peripheral neuropathy and, more commonly, an encephalopathy.

In adults the main form of exposure is to white lead oxide powder, which may be inhaled, for example, during the manufacture of batteries. Encephalopathy is not a common manifestation of plumbism in the adult. Abdominal colic and a neuropathy involving the most frequently used muscles is more usual.

Encephalopathy is the most dramatic and life-threatening development of lead poisoning, with a mortality rate of 25% or more. The onset of cerebral symptoms is usually acute, with seizures (which may be focal or generalised), delirium and coma, often associated with papilloedema. Any type of neurological sign may develop, including cerebellar ataxia, cranial nerve palsies, optic atrophy, hemiplegia, and decerebrate rigidity. Chronic cases may show mental dullness, poor memory, headache, trembling, head retraction, deafness, hemianopia and amaurosis without fundal changes.

All our patients showed an elevated cerebrospinal fluid protein level, and case 1 showed an increased cerebrospinal fluid cell count. Cases 2 and 3 had an acute onset of encephalopathy, with epilepsy as the presenting feature; while case 1 developed his seizures over 4 weeks and his encephalopathic picture took the form of a cerebellar ataxia.

All 3 patients had elevated blood lead levels, therapy resulting in both biochemical and clinical improvement.

The diversity of symptoms and signs that may be present in lead poisoning is demonstrated in our cases. It is interesting that case 3 presented with an elevated blood pressure, which rapidly fell to normal, an effect previously described.[4]

It should be kept in mind that in an industrial community, despite efforts at prevention, lead poisoning is probably more prevalent than is thought, and that lead encephalopathy is a great simulator of other neurological disorders.

REFERENCES

1. Browning, Ethel (1969): *Toxicity of Industrial Metals*, 2nd ed. London: Butterworths.
2. Stow, J. (1945): *The Survey of London*. London: J. M. Dent & Sons.
3. Whitfield, C., Li Ch'ien, L. T. and Whithead, J. D. (1972): Amer. J. Med., **52**, 289.
4. Cantarow, A. and Trumper, M. (1944): *Lead Poisoning*. Baltimore: Williams & Wilkins.