recommended that all patients subjected to thyroidectomy in euthyroid state should be given maintenance dose of thyroidal drugs. During follow-up period a careful physical examination is the most important step. Thyroid function tests could be used to presage the occurrence of hypothyroidism or recurrence of hyperthyroidism.

## REFERENCES

- Bachrac LK, Daneman D, Daneman A, Martin DJ. Use of ultrasound in childhood thyroid disorder. J Pediatr 1983, 103: 547-552.
- 2. Hopwood NJ, Carroll RG, Kenny FM, Foley TP. Functioning thyroid masses in childhood and adolescence. J Pediatr 1976, 89: 710-717.
- 3. Hung W, August GP, Randolp JG, Schisgall RM, Chandra R. Solitary thyroid nodules in children and adolescents. J Pediatr Surg 1982, 17: 225-229.
- 4. Kirkland R, Kirkland JI, Rosenberg HS, et al. Solitary thyroid nodules in 30 children and report of a child with thyroid abscess. Pediatrics 1973, 51: 86-90.
- 5. Reiter EO, Roat AWB, Detting K, Vargus A. Childhood thyromegaly. Recent developments. J Pediatr 1981, 99: 507-519.
- Scott MD, Crawford JDX. Solitary thyroid nodules in childhood. Is the incidence of thyroid carcinoma declining? Pediatrics 1976, 58: 521-526.
- 7. Fisher DA. Thyroid nodules in childhood and their management. J Pediatr 1976, 89: 866-868.
- 8. Anderson A, Bergdahl L, Boquist L. Thyroid carcinoma in children. Am Surg 1977, 43: 159-163.
- 9. Mahoney CP. Differential diagnosis of goitre. Pediatr Clin North Am 1987, 34: 891-903.

# Lead Poisoning Due to Environmental Pollution

S. Bhatia S.K. Saxena R.L. Khatri A.K. Jain

Lead is one of the most ubiquitous elements present in man's immediate environment, i.e., food, water, dust, air and soil. Such usual exposure which is about less than 120  $\mu$ g/day leads to normal blood lead level of 5-25  $\mu$ g/dl and this level does not cause any side effects(1-5).

Due to inappropriate exposure of lead, lead enters the human physiological system and causes lead poisoning which varies from asymptomatic cases leading to increased risk for future neurobehavior and acute lead encephalopathy (1,3).

Lead poisoning is a well known occupational hazard and major health problem due to increase in small scale industries in the residential area without any engineering control measures. Children suffer more than the adults during such exposures (1,6).

We report a case of lead poisoning due to environmental pollution.

## Case Report

An 11-month-old muslim girl presented with generalized convulsions and unconsciousness for half an hour. One hour after

From the Department of Pediatrics, Kasturba Hospital, Delhi 110 006.

Reprint requests: Dr. Sunita Bhatia, C-180, Sarvodya Enclave, New Delhi 110 017.

Received for publication September 14, 1991; Accepted January 2, 1992 admission, the child developed acute ballooning of the anterior fontanelle. On examination she weighed 6.5 kg, was lying limp with face turned to one side and was afebrile. She had mild pallor; pulse was 180/min regular, good volume; respiratory rate was 16/min, shallow respiration; and blood pressure was 90/60 mm Hg. She was in Grade 3 coma, muscle tone was decreased, deep tendon reflexes were normal and superficial reflexes could not be elicited. Muscle power and sensory system could not be examined as the child was in coma. On admission, blood sugar was 90 mg/dl, and urine examination was normal. A lumbar tap could not be done at admission because of the serious condition of the child, the same was done later when the condition improved. Hemogram revealed a Hb of 8 g/dl, total leucocyte count of 15,200/cumm and differential count of P 65, L 32 and M 3.

The case was diagnosed as acute encephalitis and put on anti-convulsant therapy, intravenous fluids, injection crystalline penicillin and chloramphenicol (meningitic doses) and intravenous mannitol (20%, 2 g/kg, two doses four hours apart). Convulsions and ballooning of anterior fontanelle continued inspite of anticonvulsant therapy and osmotic diuresis over a period of 12 hours.

A detailed history from the mother at this stage revealed that they were living in this area for the last two years and there are about eight to ten factories in their surroundings. In these small factories they burn the battery casings for fuel purposes and resale of metallic lead. These factories emit a lot of fumes also. Local people had seen the death of a buffalo and two dogs after convulsions and unconsciousness. A diagnosis of acute lead encephalopathy was made. The blood lead level was found in

the toxic range of more than 160  $\mu$ g/dl by chemical Dithizine method. The CSF was normal but the X-ray long bones showed presence of dense white line in the metaphysis (Fig. 1).

## Family Studies

X-rays of the parents, 3 sisters (Figs. 2 & 3) and the neighbors showed a positive lead line in the metaphysis of long bones. Except the parents, whose blood lead levels were raised, other family members refused to give blood for testing.

### Treatment

Therapy with BAL (Dimercaprol) 500 mg/m<sup>2</sup> per 24 hours in 6 divided doses intramuscularly was started. Calcium EDTA could not be procured inspite of the best efforts. BAL was given for seven days



Fig. 1. Patient's lower limb X-ray showing white lines in the metaphysis (Arrow).



Fig. 2. X-ray of patient's mother's upper limb showing white line in metaphysis (Arrow).

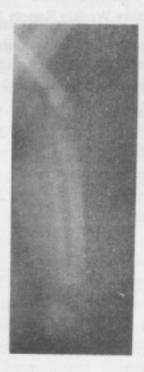


Fig. 3. X-ray of neighborhood child showing white line.

and then D-Penicillamine 500 mg/kg/day was given orally. Restricted intravenous fluids were given according to urine output.

The patient started improving. The convulsions stopped, 36 hours after starting BAL therapy and the conciousness level improved. Oral feeds were started after 48 hours alongwith continuation of intravenous fluids as the child was vomiting due to BAL therapy. As the child showed marked improvement with no apparent neurological involvement, she was discharged on penicillamine, phenobarbitone, iron, zinc, copper and calcium. At the time of discharge, blood lead level was 43 µg/dl. The family was advised to leave that area.

## Discussion

Lead poisoning occurs when the blood lead level exceeds 25 µg/dl. The CDC(5) guidelines list four risk categories depending on the blood lead level (PbB): Group I-Normal PbB (5-24 µg/dl); Group II-Moderate risk (25-49 µg/dl); Group III-High risk (50-69 µg/dl); and Group IV-Urgent risk (>70 µg/dl). The present case falls in Group IV as the blood lead level was more than 70 µg/dl. The family members also showed evidence of lead toxicity in form of changes in the X-ray. The radiological changes appear only when there is persistent increase in blood lead level of more than 50 µg/dl(3) meaning thereby that most family members were under high risk of lead toxicity. The children show denser white lines than adults as they absorb 40-50% of lead as compared to adults who absorb only 10-20%. Also, children retain about 10-20% of it while adults retain very little of it(1,6).

Acute lead encephalopathy is a very rare and serious disease and 50% and more of the survivors treated after the symptoms of encephalopathy sustain permanent brain injury(1).

Lumbar puncture is not essential for diagnosis but may be dangerous. If it is a must for differential diagnosis it should be done carefully and only few drops should be withdrawn. Mild pleocytosis, mild to moderate increase in protein and increased pressure(1,2) may be seen. In the present case CSF did not show any abnormality. The treatment of acute encephalopathy should be started as soon as the diagnosis is established and in all potential cases with persistent vomiting, ataxia, seizures and altered consciousness. Adequate urine flow is a must before starting chelation therapy. The convulsions are controlled by diazepam and/or paraldehyde. Chelation therapy is given with BAL and CaEDTA. The latter drug was not available and so only BAL was given followed by penicillamine. The lead levels should be checked regularly. In the present case the lead levels reached 43 µg/dl but the follow up after fifteen days was not possible as the patient did not return.

The most important aspect of this human made misery is prevention by public health education, a better administrative control in not allowing such pollution of atmosphere in residential areas and earliest detection and treatment of the cases and their families.

#### REFERENCES

- 1. Julian Chisolm J. Jr. Increased lead absorption and lead poisoning. *In:* Nelson's Textbook of Pediatrics, XIII edn. Eds Behrman RE, Vaughan VC, Nelson WE. Philadelphia, WB Saunders Company, 1987, pp 1507-1510.
- 2. Julian Chisolm J Jr. Increased lead absorption and acute lead poisoning. *In:* Current Pediatric Therapy 12th edn. Eds Gullis S, Kagan BM. 1986, pp 667-670.
- 3. Julian Chisolm J Jr, Bangtrap D. Recognition and management of children with increased lead absorption. Arch Dis Child 1979, 4: 249-262.
- 4. Centre for Disease Control. Lead Poisoning Associated Death from Asian, Indian Fulk Remedies. Florida Mobility, Mortality Weekly Report 1984, 33: 638.
- 5. Centre for Disease Control. Preventing lead poisoning in young children—Statement, 1985.
- 6. Rothschild EO. Lead Poisoning—the silent epidemic. N Eng J Med 1970, 283: 704-705.