'retained memory' of the shunt tubing has also been proposed as the appearance of the coiling was similar to that in the packaging when supplied(1).

The treatment recommended for ventricular shunt migration is removal of the migrated shunt tube and replacement as though the patient may remain asymptomatic, visual field defects have been reported(4,5).

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**REFERENCES**

1. Dominguez CJ, Tyagi A, Hall G, Timothy J, Chumas PD. Subgalial coiling of the proximal and distal components of a ventriculoperitoneal shunt. An unusual complication and proposed mechanism. Childs Nerv Syst 2000; 16: 493-495.
2. Kim KJ, Wang KC, Cho BK. Proximal migration and subcutaneous coiling of a peritoneal catheter - report of two cases. Childs Nerv Syst 1995; 11: 428-431.
3. Eljamel MS, Sharif S, Pidgeon CN. Total intraventricular migration of unisystem ventriculo-peritoenal shunt. Acta Neurochir (Wien) 1995; 136: 217-218.
4. Gupta PK, Dev EJ, Lad SD. Total migration of a ventriculoperitoneal shunt into the ventricles. Br J Neurosurg 1999; 13:73-74.
5. Shimizu S, Mochizuki T, Nakayama K, Fujii K. Visual field defects due to shunt valve migrating into the cranium. Acta Neurochir (Wien) 2002; 144: 1055-1056.

## Acute Lead Encephalopathy with Optic Neuropathy

Lead encephalopathy and the resulting neurological sequelae are an entirely preventable problem with no coherent preventive strategies in India. We fail to manage many cases of lead encephalopathy due to lack of diagnostic facilities and poor availability of chelators such as calcium sodium versenate, dimercaprol, or succimer.

An 11-month-old girl was brought with a history of ingesting a metallic object used for fishing, 15 days prior to admission. She had fever, vomiting, constipation for 3 days, convulsion and absence of menigeal signs or neurological deficits. She had normal fundus, cerebrospinal fluid (CSF), total and differential leukocyte counts and normocytic hypochromic anemia (Hb 8 g/dL). Plain radiograph of the abdomen revealed a radio-opaque foreign body of size 0.5 × 1.5 cm in left hypochondrum, which was subsequently not observed in the stool (*Fig. 1*). On 4th day of hospitalization she developed signs of raised intracranial tension and then lapsed into shock. The serum electrolytes were normal and the CSF remained normal. Blood was withdrawn for lead levels that were 129 µg/dL by flameless atomic absorption spectrophotometry. She was treated with D-penicillamine (30 mg/kg/day), the only available chelator in the market. She was also given supportive treatment for raised

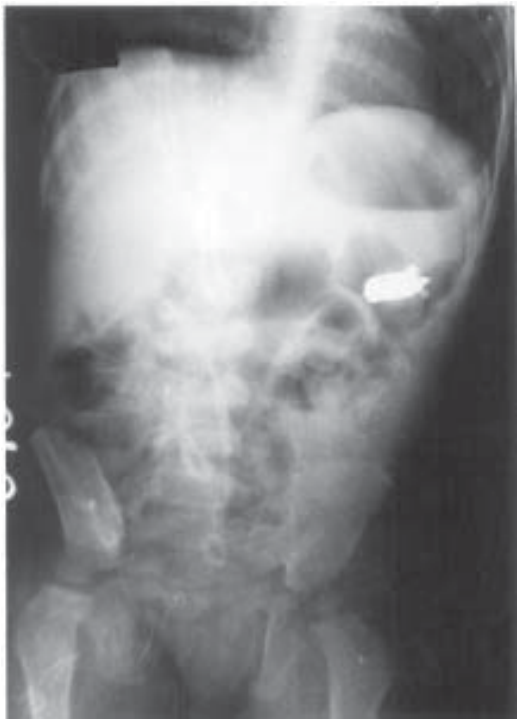

*Fig. 1. Plain radiograph of abdomen showing radio-opaque foreign body.*

intracranial tension with shock. Within 48 hours there was an improvement in her sensorium and her vital parameters stabilized. However, fundus examination on the 8th day revealed anterior ischemic optic neuropathy. Her blood lead a week later was 67 µg/dL.

This was a patient of acute lead poisoning encephalopathy resulting in ischemic optic neuropathy, rare neurological sequelae. The combination of raised intracranial pressure and anemia is responsible for ischemic optic neuropathy in lead encephalopathy(1). Existing high prevalence of iron deficiency anemia may increase the toxic effects of lead on children in our country(2). The prevalence of elevated blood lead level ( ≥10 µg/dL) in preschool children in the community has been reported to be 67.7% in this city, the main sources of exposure being house paint and eye cosmetic (surma)(3). Acute lead encephalopathy is an emergency usually resulting from ingestion of lead rich objects and associated with blood 1ead ≥100 µg/dL(4). Because of this high prevalence of elevated lead levels in the community, the possibility of lead encephalopathy should always be considered in the differential diagnosis of children presenting with coma and convulsions of unknown etiology, and, blood lead levels should be obtained even in absence of history of ingestion of lead rich objects. Even when identified and promptly treated, severe and permanent brain damage may result in 70-80% children(5). Prompt recognition of prodromal symptoms followed by early medical management with chelating agent can prevent the onset of life threatening complications.

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REFERENCES

1. Baghdassarian SA. Optic neuropathy due to lead poisoning. Report of a case. Arch Ophthalmol 1968; 80: 721-723.
2. Tandon SK, Khandelwal S, Jain VK, Mathur N. Influence of dietary iron deficiency on nickel, lead and cadmium intoxication. Sci Total Environ 1994; 148: 167-173.
3. Patel AB, Williams SV, Frumkin H, Kondawar VK, Glick H, Ganju AK. Blood lead in children and its determinants in Nagpur, India. International J Environ Occup 2001; 7: 119-126.
4. Piomelli S, Rosen JF, Chisolm JJ, Graef JW. Management of childhood lead poisoning. J Pediatr 1984; 105: 523-532.
5. Perlstein MA, Attala R. Neurologic sequelae of plumbism in children. Clin Pediatr 1966; 5: 292-298.