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Lead Poisoning

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INTRODUCTION

Lead poisoning or plumbism in children can be traced to pica (eg, dirt eating), acute ingestion of lead-based paints or consumption of home remedies (1,2).

Lead poisoning in children presents with non-specific and vague symptomatology, however in serious conditions it may present with encephalopathic features (1,3).

Radiologically it is characterized by presence of dense transverse bands in the growing metaphyseal regions (2,3,4).

Here we report a case of chronic lead poisoning in a child of 18 months, which presented with encephalopathy.

CASE REPORT

A 18 months male child born in lower socio-economic strata presented with an episode of generalized tonic-clonic seizure lasting for half an hour not associated with fever, the history of projectile vomitings since 3 days, passage of watery stools 3-4 times a day and drowsiness. There was history of recurrent admissions in the past 4 months in some peripheral hospital for near similar complaints and empirically was on anti-tubercular therapy but no significant improvement was noted. His developmental milestones were normal. There was no history to suggest birth asphyxia and peri-natal history was non-contributory.

On examination child was drowsy and irritable but arousable. General examination revealed pallor of mucosal membranes and gums, otherwise normal. Detailed neurological examination revealed the following: child was drowsy and irritable, normal cranial nerves, motor system examination showed hypotonia, grade 3 power in all limbs with absent deep tendon jerks and equivocal planter reflexes, co-ordination could not be tested, was responding to prick. He had no signs of meningeal irritation. Fundus examination revealed no significant abnormality. Other systemic examination showed hepatomegaly of 4 cms. Investigations revealed: Hb-9.4 gm%, TLC-12650, DLC-N62L31M3E4B0, ESR-38 mm/hr, platelet count-3.79 lacs, random blood sugar-93 gm%, SGOT-310 IU/dl, serum calcium-8.7 mg%, routine urine examination- showed traces of albumin, blood urea- 39 mg%, serum creatinine-1.2mg%, CSF analysis revealed no significant abnormality.

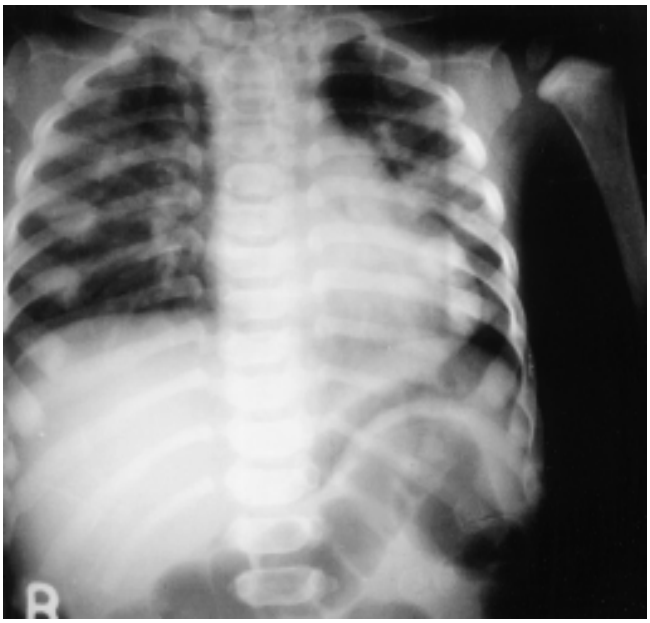

Fig.-1:- Chest PA view shows prominent costochondral junctions and dense proximal metaphysis of left humerus.

Ultrasound of the abdomen showed mild hepatosplenomegaly. Contrast enhanced CT of head showed mild prominence of lateral ventricles and subtle hypodensities in left hippocampal region, which did not explain patient's symptoms. Chest X-ray revealed prominent costochondral junctions and more strikingly dense proximal metaphysis of humerus on left side (Fig.-1). This prompted us to take radiographs of extremities for further evaluation. Radiographs of wrist with hands and both the lower extremities were taken which showed thick metaphyseal bands in the distil ends of radius and ulna,

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both femurs, bases of metacarpals and phalanges (Fig.-2&3). Considering the clinical presentation and radiological features a probable diagnosis of heavy metal poisoning was thought. On further interrogation of the parents, they came out with the history that child used to ingest the lead of the batteries which his father used to bring home for repairs. To confirm the diagnosis blood lead levels was done which showed very high lead levels of 97 gm/dl (Normal level-10 gm/dl). Erythrocyte protoporphyrin levels were raised to 144 gm/dl. Peripheral smear showed basophilic stippling and reticulocyte count was 12%.

Since whole of the family was exposed to lead X-rays of other sibling were also done which too showed evidence of lead poisoning.

After confirmation of diagnosis anti tubercular treatment was stopped and chelation therapy was started. Patient responded to treatment and regained consciousnesses, muscle power, and there was no episode of convulsion.

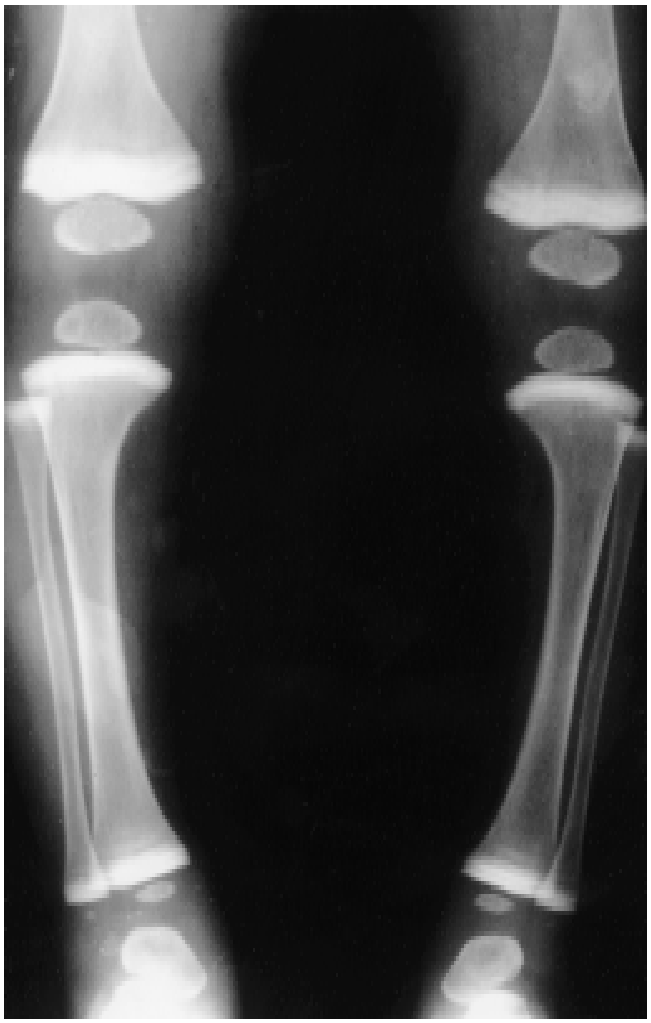

Fig.-2:- X-ray of both lower limbs shows dense metaphysis of femur, tibia, fibula and bones of feet.

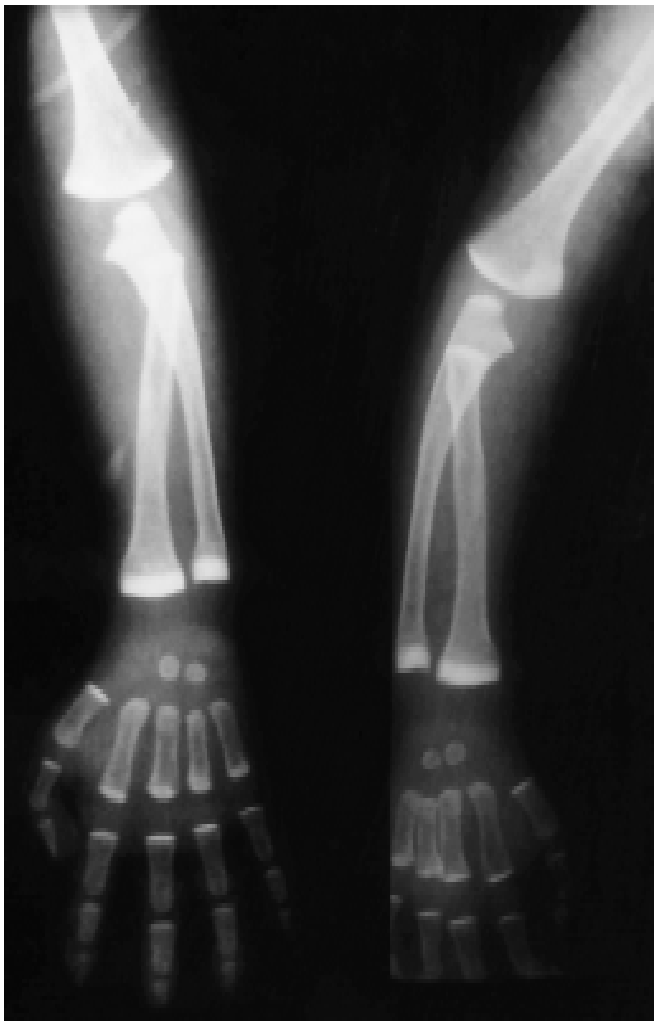

Fig.-3:- X-ray of both upper limbs shows prominent dense metaphysis of all bones.

This treatment was continued for 2 weeks and on request of parents patient was discharged (probably parents could not afford the cost of treatment), there after patient is untraceable.

DISCUSSION

The natural blood level of lead is 0 g/dl but practically levels upto 10 g/dl are considered normal. Any level above this is considered abnormal and lead lines (metaphyseal band sign) appear at levels of 50 g/dl while encephalopathic features are usually seen when lead levels are more than 100 g/dl (1,5).

The toxicity of lead results from its avidity for the sulfhydryl (SH) group of proteins and various enzymes which leads to jeopardization of their function (1,3). In bones lead inhibits osteoclastic remodeling preferentially in the zone of provisional calcification which results in an increase in the thickness and number of trabeculae at the metaphysis. And exuberant calcium deposition in the zone of provisional calcification yields dense metaphyseal bands (2,6).

The most serious manifestation of lead poisoning is acute encephalopathy. This is usually preceded by behavioral changes and/or vomitings, intermittent abdominal pain and constipation. Encephalopathy includes persistent vomtings, ataxia, seizures, altered behavior, impaired consciousness and coma. In absence of encephalopathy symptomatology is non specific and vague, and difficult to diagnose (1,3).

Diagnosis is established by definitive history of exposure of lead in any form, raised blood levels of lead and erythrocyte protoporphyrin levels. Radiological examination is supportive and can give a clue to clinch the diagnostic possibility. Features to suggest are dense transverse metaphyseal bands at ends of growing bones particularly around the knee (2,4). On review of literature in one study presence of dense metaphyseal band especially in proximal fibula was highly suggestive of plumbism (5). With treatment or cessation of lead exposure, the lead band will demonstrate an apparent migration into the metadiaphysis because of normal new bone growth. Accordingly, when the lead level returns to normal, the metaphyseal band will gradually decrease in radiopacity and disappear in approximately 4 years (2). On imaging calcifications in basal ganglia region can be appreciated in some cases (7).

The dense metaphyseal band sign enables a lengthy differential diagnosis. In order of decreasing frequency, the causes of a dense metaphysis include normal variance, plumbism, treated leukemia, healing rickets, other heavy metal (arsenic, bismuth, mercury) poisoning, recovery from scurvy, vitamin D hypervitaminosis, congenital hypothyroidism, hypoparathyroidism, and transplacental infections (eg, toxoplasmosis, rubella, cytomegalovirus, and herpes) (7-10).

In conclusion, as laboratory analysis of lead levels cannot always be readily performed in infants with unexplained encephalopathy, in such cases radiography of the knees can be of great help. The presence of dense metaphyseal bands strongly supports the diagnosis of lead toxicity which can be confirmed by relevant investigations (11).

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