FETAL LEAD EXPOSURE: ENCEPHALOPATHY IN A CHILD

Herman S. Dsouza, Geraldine Menezes & Venkatesh. T

Department of Biochemistry and Biophysics, St. John's Medical College
St. John's National Academy of Health Sciences,
Koramangala, Bangalore, Karnataka- 560034, India

ABSTRACT

It is well known that lead exposure in the early period of pre pregnancy and antenatal life leads to serious health complications. In this case report, a five month old child who was suffering from encephalopathy was finally confirmed a victim of lead exposure, the source being the environment and the family. We report this case with complete clinical investigation including blood lead analysis. This case report highlights the various ways in which lead may accumulate in the body. It is also aimed at increasing awareness regarding the pathogenesis and clinical manifestations of lead toxicity. The literature survey done reveals that this is the youngest age at which lead toxicity has been documented in India.

KEY WORDS:

Blood lead, Zinc Protoporphyrin, Encephalopathy

INTRODUCTION

Lead is the number one environmental pollutant all over the world causing health hazards (1,2). The major source of lead is from occupations where lead and lead based components are used, resulting in high prevalence of lead toxicity in the population exposed to such activities (3). The most sensitive population for lead exposure are pregnant women, infants and young children (4,5). Most of the environmental exposure occurs through inhaling air containing lead dust, drinking water supplied through leaded pipe lines and consuming processed, preserved and stored food (6,7). Occupational exposure to workers is seen in lead based industries such as lead acid battery manufacturing, cable and wire products industries, rubber and plastic industries, soldering activities, in-foundry work such as casting, forging and grinding activities. Apart from this, construction workers involved in painting or paint removal, plumbing, welding and cutting also get exposed to lead (8,9). Children especially get exposed to lead through various hobbies such as playing with painted toys, repeated ingestion of non food substances etc. (10). A pregnant lady can

transfer her body burden of lead to the growing fetus as there is no placental barrier for a heavy metal like lead (11). Lead exposure is also seen due to the usage of certain traditional medicines and folk remedial procedures (12).

CASE REPORT

A five month old female child was referred to our "National Referral Center for Lead Poisoning in India" (NRCLPI) with a presumptive diagnosis of lead poisoning. The child was delivered at home to an 18 year old mother (para 1) with an uneventful antenatal history. The child cried immediately after birth, was put on sugar water as pre lacteal feed for a day and breastfed subsequently. Patient's history revealed that she was admitted to a local Children's Hospital with complaints of vomiting of four days duration and one episode of generalized tonic convulsion of five minutes duration on the day of admission. There was no fever and other precipitating factors. On examination, the finding per abdomen: liver was palpable 2 cms below the right costal margin, Central Nervous System: patient was drowsy but arousable. Rest of the examination showed no abnormality. The results of the relevant investigations were : RBS 4.5 mmol/l (81 mg/dl), serum calcium 2.00 mmol/l (8.0 mg/dl), Hemoglobin 7.5 g/dl, Peripheral Blood

Author for correspondence:

Dr. T. Venkatesh M.Sc., Ph.D., FACBI., FINASS.

Professor at above address

e-mail address: venky_tv@hotmail.com

Tel: (O): 0091-080-2065058

(R):0091-080-5532146 Fax: 0091-080- 5520777

Smear - dimorphic anaemia. Rickets was suspected.

X-ray of the wrist showed metaphyseal dense bands. The child was put on anticonvulsant phenytoin. The child did not have convulsions during the eight day stay in the Hospital and was discharged. After six days of discharge, the child was readmitted with complaints of convulsions and refusal of feeds. On examination, the anterior fontanelle was bulging and the child drowsy. Meningitis was suspected. A lumbar puncture done showed normal results. During this period, the child had two more attacks of convulsions and was restarted with anticonvulsant phenytoin. Skeletal survey done to check for heavy metal poisoning showed metaphyseal dense bands at all sites. The child was discharged after convulsions were under control and at this point was referred to our NRCLPI. Screening child's blood for lead poisoning in our laboratory involved estimation of lead levels and zinc protoporphyrin (ZPP), an abnormal heme compound found elevated in lead toxicity / iron deficiency anemia. ZPP was estimated by front face fluorometry using AVIV haematofluorometer and lead by anodic stripping voltammetry (ASV) using ESA lead analyzer. Results showed elevated ZPP level of $3.24\mu\text{mol/l}$ ($182\mu\text{g/dl}$) and lead level of $4.68\mu\text{mol/l}$ ($97\mu\text{g/dl}$). [Blood lead acceptable upto : $0.48\mu\text{mol/l}$ ($10\mu\text{g/dl}$); ZPP : $0.62\mu\text{mol/l}$ ($35\mu\text{g/dl}$)]

Parents history revealed that they had lived in a lead based industrial area. The father's occupational career involved removal of lead from battery, grinding it into powder and making pipes. The parents were subsequently checked for lead. The father was found to have ZPP level of $1.83\mu\text{mol/l}$ ($103\mu\text{g/dl}$) with a lead level of $4.72\mu\text{mol/l}$ ($98\mu\text{g/dl}$) and mother a ZPP level of $1.74\mu\text{mol/l}$ ($98\mu\text{g/dl}$) with a lead level of $2.31\mu\text{mol/l}$ ($48\mu\text{g/dl}$). The mother remained asymptomatic for lead poisoning during pregnancy, though the father had been experiencing generalized body ache, abdominal pain and occasional giddiness.

Conclusion

Studies in the past have clearly shown the ill effects of lead on the central nervous system of a child, when the blood lead level is as low as $0.48\mu\text{mol/l}$ ($10\mu\text{g/dl}$) (13). Lead induced encephalopathy being a serious and an end stage disorder of lead poisoning happens, when the blood lead level is around $3.86\mu\text{mol/l}$ ($80\mu\text{g/dl}$). Our case report

highlights the various ways through which the child could have got exposed to lead. The child's father working with lead in a lead based industry, must have carried lead dust home thus contaminating his living environment. Since the family had been residing close to the lead based industry, inhaling and ingestion of lead dust had become an additional source of lead exposure which was evident in our report, with elevated blood lead levels being noticed in all the members of the family. Chronic lead poisoning in this family was also confirmed by elevated levels of ZPP, a good indicator of such exposure. During the pregnancy, the mother had become a source of lead to the growing fetus since maternal bone lead stores can be mobilized to a significant degree during pregnancy with consequent exposure to fetus. In addition to the lead passed on to the fetus, after the birth, the child continued to have lead exposure through the mother's milk which further alleviated the condition (4).

When the child attained the age of five months with episodes of vomiting and convulsion, it received only a symptomatic treatment without knowing the root cause of the problem. It was only when the child's X-ray showed metaphyseal dense bands was further screening done (14). The child was confirmed as lead poisoned with high ZPP and blood lead levels. The entire problem revolved around the occupational and environmental lead which was causing a major disaster in their lives.

With very low awareness levels regarding the harmful effects of lead in our environment, irreversible damage has already been caused to the only child of the family. Currently the child is on treatment and the parents are following the guidelines provided to them which has resulted in the shifting of their residence from a lead based industrial area. It is also heartening to know that the father of the child took a bold step in changing his profession to a skilled laborer in an unleaded industry. In a developing country like India, there are probably innumerable unidentified / undiagnosed cases of lead poisoning, similar to the one mentioned in this report.

Acknowledgement

The authors wish to thank "The George Foundation", a non-government organization, Bangalore, Karnataka, India. for sponsoring this study and Dr. Pranita for referring the above case.

REFERENCES

1. Lockitch, G. (1990) Perspectives of lead toxicity. Clin Biochem. 26(5), 371-381.
2. Olaiz, G., Fortoul, T.I., Rojas, R., Doyer, M., Palazuelos, E. and Tapia, C.R. (1996) Risk factors of high levels of lead in blood of school children in Mexico city. Arch Environ Health 51(2), 122-126.
3. Fanning, D.(1988) A mortality study of lead workers.1926-1985. Arch Environ Health 43(3), 247-251.
4. Silbergeld, E.K. (1991) Lead in bone: Implication for toxicology during pregnancy and lactation. Environmental Health Perspectives 91, 63-70.
5. Rahman, H.A., Alkhayat and Menon, N. (1986) Lead poisoning in infancy-unusual cases in the UAE. Annals of Tropical Paediatrics 6, 213-217.
6. Adgate, J.L., Weisel, C., Wang, Y., Rhoads, G.G.and Lioy, P.J. (1995)Lead in house dust. Relationships between exposure metrics. Environ Res 70(2), 134-147.
7. Pocock, S.J. (1980) Factors influencing household water lead: A British National Survey. Arch Environ Health 35,45-51.
8. Awad el Karim, M.A.,Hamed, A.S., Elhaimi, Y.A.A.and Osman,Y. (1986) Effects of exposure to lead among lead acid battery factory workers in Sudan. Arch Environ Health 41,261-265.
9. Gittleman, J.L., Engelgau, M.M., Shaw, J., Wille, K.K. and Seligman, P.J. (1994) Lead poisoning among battery reclamation workers in Alabama. J Occup Med 36, 526-532.
10. Schwartz, J. and Levin, R. (1991) The risk of lead toxicity in homes with lead paint hazard. Environ Res 54(1), 1-7.
11. Bellinger, D., Leviton, A., Rabinowitz, M., Alfred, E.,Needleman , H.and Schoenbaum, S.(1991b) Weight gain and maturity in fetuses exposed to low levels of lead. Environ Res. 54,151-158.
12. Peter, M.T. and Alan, M.B. (2000) Alternative medicines elevate blood leads in Omani children referred for extensive investigation. J.Trop.Ped. 46, 241-242.
13. Baghurst, P.A.,McMichael, A.J.,Wigg, N., Vimpani, G., Robertson, E.F., Roberts, R.J. and Tong, S.L.(1992) Life long exposure to environmental lead and children's intelligence at age seven: The Port Pirie cohort study. N. Engl. J. Med. 327,1269-1284.
14. Woolf, D.A., Riach, C.F., Derweesh, A. and Vyas, H. (1990) Lead lines in young infants with acute lead encephalopathy: a reliable diagnostic test. J. Trop. Ped. 36,90-93.