EDITORIAL

Blood Lead Level in Children with Encephalopathy

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No other heavy metal toxicity has gained as much public attention as lead – due to its impact on the developing brain of young children. Lead is ubiquitous in our environment but has no physiologic role in our biological systems. Lead became part of our daily life due to its easy workability, low melting point and corrosion-resistance. Later in our industrial society, it became part of house paint due its shining and lasting function and also used in gasoline for its anti-knock properties.

The toxicity of lead comes from its ability to mimic other biologically important metals, most notably calcium, iron and zinc, which act as cofactors in many enzymatic reactions. Lead is able to bind to enzymes delta-aminolevulinic acid dehydratase, and ferrochelatase, affecting the biosynthesis of heme causing anemia.

Lead also interferes with excitatory neuro-transmission by glutamate, which is the transmitter at more than half the synapses in the brain and is critical for learning. N-methyl-D-aspartate (NMDA) receptor, thought to be associated with neuronal development and plasticity, is blocked selectively by lead. This disrupts long-term potentiation, which compromises the permanent retention of newly learned information(1). Lead exposure also decreases the amount of NMDA receptor gene and protein in hippocampus(2).

The main sources of lead poisoning in USA is ingestion of lead contaminated house dust and leaded paint chips in older housing. In developing countries contaminated soil due to burning of leaded gasoline is one of the major source of lead poisoning. Lead smelting/mining and automobile battery recycle factories can also contaminate the environment. Other potential sources of lead poisoning in young children are from ayurvedic preparations and cosmetics such as kohl and *surma*. Other sources are imported toys, such as many made in China. Lead can leach from leaded china, dishes, and mugs. Lead can also migrate into wells and waterways from nearby rifle ranges, if the right conditions exist.

Children are more at risk than adults for lead absorption. Lead absorption is augmented in the presence of iron, zinc, and calcium deficiency. Lead absorption is also augmented by malnutrition. Lead absorption decreases if phosphorus, riboflavin, vitamin C, and vitamin E are in the diet. In general, approximately 30-50% of lead ingested by children is absorbed compared with approximately 10% in adults. Once absorbed, lead is distributed widely throughout the body, where it exists in three major compartments – blood, bone, and soft tissues.

Lead encephalopathy is currently uncommon, and usually requires blood lead levels exceeding 150 µg/dL in adults; children are at much greater risk for developing lead encephalopathy at lower lead levels (>70-90 µg/dL)(3). Clinically, lead encephalopathy can present in acute or chronic forms. In acute lead encephalopathy the patient presents with headache, vomiting, ataxia, convulsions, paralysis, stupor and coma(4).

In this issue of *Indian Pediatrics*, Patel and colleagues(5) have presented a case control study of young children between the ages of 4 and 10 years, where they determine and compare the prevalence of elevated blood lead level (EBLL) among children

with encephalopathy from those with no encephalopathy. Researches found significantly elevated EBLL in patient with encephalopathy compared to non encephalopathic group. Their analysis found a strong correlation between EBLL with predictors such as physical wasting, anemia, use of *surma*, recent house paint removal and repainting.

This is an interesting intuition of evaluating lead level among children who were admitted to the hospital with encephalopathy. It would be arduous to constitute an association between EBLL and non lead encephalopathy. One argument is that if EBLL is linked to increased risk of infection. There are only 16 cases of infectious encephalopathy in the study, which included bacterial, mycobacterium and viral etiology. These 3 subclasses of infections have different pathogenesis, it would be laborous to detemine how EBLL affect the body's defense mechanism to promote infection. And what is the threshold of EBLL to promote infection. Also there were 26 cases of non-infectious encephalopathy, the same infection promoting hypothesis may not apply there?

The paper points out an important epidemiological issue that underlying health conditions such as anemia, malnutrition can enhance the absorption and can increase the deleterious effect of lead poisoning. The same coexisting conditions may have contributed in their susceptibility to encephalopathy?

I am still intrigued with the fact that the group with encephalopathy had high EBLL. This study points out some of the predictors of EBLL which were more common in encephalopathic group like use of surma, still it does not explain the 8 cases who have CDC class III lead poisoning. This obscurity may be uncovered by analyzing the baseline characteristics of the study population.

Children in encephalopathic group are lot younger than those with no encephalopathy. Non occupational lead poisoning is more common in young children. Young children get exposed to environmental hazards more often which is due to their mental immaturity, or natural curiosity of exploration. Their personal hygiene skills are not that sophisticated. They put their hands in dirt and then into their mouth. They are small and closer to ground, so they inhale more dust than adults.

The study did not find intake of ayurvedic medicines as a predictor of EBLL but it has potential of lead contamination and there were 7 children in encephalopathic group and none in non encephalopathic group who used ayurvedic medicines.

The study provides a preliminary insight of prevalence of lead toxicity in the young population. This study does not disclose any new knowledge about lead toxicity or support any association of EBLL to infection. It sure does bring up the fact that chidren are at high risk to preventable environmental health hazards and these hazards can add to their total outcome of the health and illness.

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