Unmet need to screen for lead toxicity in the prevention of ADHD

Dear Editor,

In children, attention deficit hyperactivity disorder (ADHD) is one of the most prevalent psychiatric disorder.[1] Though a number of genetic loci have been identified, there is still a lot of uncertainty, thus is often labelled idiopathic. However, the very likely aetiology, are the various environmental pollutants, especially heavy metals. As reported from earlier studies, among the environmental toxicants, lead (Pb) and mercury (Hg), contribute the most to the risk of ADHD.[2] It has been established through many previous studies that elevated lead levels led to several neuro-behavioural deficits in children. One possible mechanism is the substitution of certain polyvalent ions like calcium and zinc by Pb. The membrane ion channels and signalling molecules are the most commonly associated target molecules. The substitution affects cellular pathways like metal ion transport, energetics and catabolism, protein assembly and intra- and/or inter-cellular signalling.[3]

Though seemingly inconspicuous lead can get easily accumulated in the body of a child through multiple sources. Lead-based paints used to be the mainstay of building as late as till the later half of 1970s. The cracking and peeling off of the paints can be absorbed through ingestion or inhalation by the child. Drinking water may get contaminated with lead, either at the source through surface run-off, or, through lead-lined water pipes or during storage. Some amount of lead is found in confectionaries, as well as home remedies practised across the cultures. Toys and some items of jewellery also contain lead in minute quantities. Parents also become a major source of lead assimilation by a child, if they work in factories actively involved in lead processing like smelting, battery manufacturing, etc., Households near airports, heavy industries or metallurgical factories have an increased propensity to contaminate soil and air with lead pollutants.[4]

Lead once absorbed is exchanged primarily between three compartments: blood, soft tissues, bones and teeth. The route of administration affects absorption of lead, and it is inversely proportional to particle size; Hence absorption through the respiratory route in the form of lead dust is more toxic than that through the gastrointestinal tract (GIT). The most common form of lead is the inorganic form which is not metabolized by the liver, but the organic form is nearly totally absorbed and metabolized by the liver. About 99% of the lead in blood is carried by red blood cells. Its half-life in adult blood is around 28–36 days. The estimation of lead levels in blood and urine provides an assessment of contemporary exposure, whereas hair and nail levels reflect metabolic changes over long periods of time. Lead and other heavy metals in these biological matrices are not affected by rapid variations in diet, air, or water. In case of children, 73% of the lead burden is in the bones and teeth. In this too there is no uniform distribution as there is a high tendency to accumulate in bones undergoing rapid calcification. There is exacerbated bone-to-blood mobilization in conditions like iron deficiency anaemia, hyperthyroidism, physiological stress, fractures, kidney diseases, and calcium deficiency. The absorbed and stored lead can disturb several generations of somatic cells. Because there is maximum neuronal development in the first year of life, prevention is better than cure which is rare in ADHD. Attention has also been drawn to the increased bone to blood mobilization of lead during pregnancy due to the physiological stress and the association of harmful effects on foetal neurodevelopment.[5]

Having been enriched by these evidences, it is of paramount importance for undertaking steps to screen for risk levels in populations and areas likely to have lead and other heavy metals in toxic levels so as to cause ADHD. Prevention of exposure to such elements at a prenatal stage and their chelation and removal may go a long way in providing quality life to such affected individuals and more so for their family members.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Suchanda Sahu[1], Saurav Nayak[1], Joseph John[2]

Departments of [1]Biochemistry, and [2]Paediatrics, All India Institute of Medical Sciences, Bhubaneswar, Odisha, India

Address for correspondence: Dr. Suchanda Sahu, Department of Biochemistry, All India Institute of Medical Sciences, Bhubaneswar- 751019, Odisha, India.
E-mail: biochem_suchanda@aiimsbhubaneswar.edu.in

References

1. Zhou R, Xia Q, Shen H, Yang X, Zhang Y, Xu J. Diagnosis of children's attention deficit hyperactivity disorder (ADHD) and its association with cytomegalovirus infection with ADHD: A historical review. Int J Clin Exp Med 2015;8:13969-75.
2. Huang S, Hu H, Sánchez BN, Peterson KE, Ettinger AS,

Lamadrid-Figueroa H, *et al.* Childhood blood lead levels and symptoms of attention deficit hyperactivity disorder (ADHD): A cross-sectional study of mexican children. Environ Health Perspect 2016;124:868-74.

3. Mason LH, Harp JP, Han DY. Pb Neurotoxicity: Neuropsychological effects of lead toxicity. Biomed Res Int 2014;2014:1-8. doi: 10.1155/2014/840547.
4. Centers for Disease Control and Prevention (CDC). Lead poisoning in pregnant women who used Ayurvedic medications from India--New York City, 2011-2012. MMWR Morb Mortal Wkly Rep 2012;61:641-6.
5. Miranda ML, Edwards SE, Swamy GK, Paul CJ, Neelon B. Blood lead levels among pregnant women: Historical versus contemporaneous exposures. Int J Environ Res Public Health 2010;7:1508-19.

Received: 06-10-2020
Accepted: 02-12-2020 **Published:** 30-01-2021

Access this article online

Quick Response Code:

Website: www.jfmpc.com

DOI: 10.4103/jfmpc.jfmpc_2065_20

How to cite this article: Sahu S, Nayak S, John J. Unmet need to screen for lead toxicity in the prevention of ADHD. J Family Med Prim Care 2021;10:586-7.