

Lead in Neurological Practice: A Mini Review

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Abstract

Lead (Pb) had a wide range of use in industry due to its properties like ease of casting and fabrication, resistance to corrosion and opacity to x rays and a low melting point. It has gradually being replaced by other material. Can be absorbed in body from almost any route. Lead is distributed to the following 3 pools blood and soft tissue. We briefly review the manifestations of lead in neurology.

Keywords: Lead; Neurological; Casting; Fabrication

Introduction and History

Lead (Pb) had a wide range of use in industry due to its properties like ease of casting and fabrication, resistance to corrosion and opacity to x rays and a low melting point [1,2]. It has gradually being replaced by other material.

Discussion

Lead can be absorbed in body from almost any route. Absorption in to the body has been known via respiratory tract as well as the gastrointestinal tract and sometimes even skin. Depending in the route, both elemental or inorganic lead and organic lead can be absorbed. It's distributed all over the body, especially erythrocytes.

Lead is distributed to the following 3 pools blood and soft tissue (this is the exchangeable pool, therefore most crucial toxicologically), soft tissues, and a skeletal pool. It's excreted in urine and faeces (unabsorbed and from bile). Small amounts of lead are also eliminated in other fluids e.g. saliva, sweat, breast milk, etc [2].

Robert Kehoe suggested that the blood lead concentrations below 80 µg/dl may not cause clinical lead poisoning. Maximum blood concentration considered safe was therefore reduced to 80 µg/dl. Currently considered safe levels are below 10 µg/dl. Nephrotoxicity (seen as proteinuria and low GFR) occur with blood lead levels more than 50 µg/dl. Sperm abnormalities occur over 40 µg/dl. Endocrine effects are evident beyond 60 µg/dl [2]. Delayed puberty occurs in girls [3].

International Agency for Research on Cancer determined that inorganic lead compounds are carcinogenic to humans (group 2A), and that organic lead compounds are not classifiable regarding carcinogenicity to humans (group 3) [4].

A prolonged elimination half life (30 days in blood and 27 years in bone) results in increases in body levels with time. Lead accumulates till critical body burdens are reached and then sudden onset, rapidly progressive symptoms develop. However, subclinical presentations may occur early.

Lead can affect central, peripheral and autonomic nervous system.

Centrally, mild symptoms are characterized by fatigue and lethargy that can disturb the routine activities. Severe conditions like encephalopathy, impaired consciousness, and bizarre neurological signs are unusual in adults but frequent in children due to pica [5]. The severity of symptoms range from confusion and disorientation to repeated resistant seizures, coma and death. Lateralizing signs like focal seizures, hemiparesis and Babinski sign on one side may be seen as well [6]. Encephalopathy with headaches, excess salivation, vomiting, irritability, insomnia, delusions and hallucinations may be seen [7]. Chronic exposure to lead causes psychiatric symptoms and mild cognitive impairment [5].

Peripherally, neuropathy is frequently seen. This neuropathy is usually asymmetrical. The symptoms are commonly motor and rarely sensory. The weakness is more common in upper limbs than in lower limbs affecting finger extensors followed by wrist extensors leading to “wrist drop”. Weakness may also be seen in other muscles and even more proximal muscles. Similar weakness in lower limbs causing “foot drop” may be seen in children [5].

Conclusion

Lead poisoning is not a common condition seen any more. However, one needs to be aware of the neurological symptoms that can be progressive if left unattended.

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