

Environmental and public health repercussions of the heavy metal lead (Pb) in the pediatric population

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ABSTRACT

The clinicopathologic, public and environmental health effects of the heavy metal, lead (Pb) due to its exposure in children are of global implications and concerns. Inordinate numbers of children are exposed to lead substances and are subjected to diverse forms of morbidity and consequent mortality. Infants and children under age-5 years old are at increased risk of lead poisoning because their brains are susceptible to insults prior to full development culminating in prolonged or lifelong cognitive, neurological and physiologic aberrations. Speculations are rife as to the concentration of lead severally or jointly with other metals in organs and systems that could exacerbate concomitant behavioral, cultural, economic, developmental, and social variables and determinants in children. It is intricately complex to detect, diagnose or correlate the health consequences due to incessant exposure of low lead levels invariably encountered in both the internal and external milieu regarding the fetus and children, respectively. The toxic attributes of lead encompass biologic, economic, environmental, familial and social factors, which by themselves are well-nigh impossible to be explicated or resolved solely via epidemiological trajectories. Studies are pertinent to replenish knowledge and information of emerging criteria and issues to elucidate extant and future threats associated with lead in children, particularly in low- and middle-income countries.

Introduction

The heavy metal lead (Pb) that is a potent neurotoxin, albeit with a minimal level exposure correlates to diminutive IQ scores, reduced attention span, irritability, potential violence, anti-social disposition and criminal behavior of the children in later life. Lead has no beneficial, but untoward effects inside human organs and systems when ingested via water or food and inhaled via air, aerosol or any other conceivable form. Lead poisoning or plumbism generates a condition termed saturnism that clinically mimicks AIP, and ostensibly, dysfunctionality of heme synthesis with delta-aminolevulinic acid accumulation [1]. Despite expansive warnings regarding the devastating impacts of lead, it continues to be introduced in diverse industrial and consumer products to the detrimental effects of fetuses and children. There are inadequate infrastructures to prevent or impede lead impairment as frequently encountered in the internal and external milieu [2]. Research on lead exposure may contain flaws because of imprecise methodologies, confounding and inaccurate covariate adjustment [3].

Etiopathogenetic Mechanisms, Predictive or Diagnostic Parameters and Remedies

The precise pathophysiological regulatory or control mechanisms of lead toxicity or poisoning are not clearly elucidated; however, it is established that this heavy metal competes with other associated heavy metals or minerals, such as zinc, cadmium [4] and calcium within the internal and external milieu. Blood lead and manganese concentrations are elevated in iron-deficient infants and are associated with extended breast feeding [5]; in which iron deficiency may culminate in cognitive derangements due to the deficiency or concomitant or exclusive elevated metal levels induced by iron deficiency. Within cellular systems, lead interacts with other trace elements resulting in the disruption of the functionalities of viable and beneficial elements [6]. The environmental and public health issues associated with lead are exposure of children to decadent lead fragments, exhaust and petrol fumes, lead contaminated and polluted food, natural or potable water [7].

Lead poisoning constitutes one of the most significant chronic public and environmental health disorders in the pediatric outpatient and emergency departments. The predominant mechanism of lead poisoning is due to augmented production of reactive oxygen species, ROS and the obstruction of antioxidant formation [8]. Lead generates ROS, such as hydrogen peroxide, hydroperoxide and singlet oxygen which become stabilized and sustained by glutathione, and following its oxidized conversion to glutathione disulfide, it is reduced and reverted by glutathione reductase to GSH. Lead deactivates glutathione via its binding to the GSH sulfhydryl group resulting in the attenuation GSH potential and increased oxidative stress. In addition, lead impedes the functionality of certain antioxidant enzymes, such as catalase and superoxide dismutase. The augmentation in oxidative stress results in cell membrane excoriation via lipid peroxidation and culminating in erythrocyte hemolysis [9].

With passage into the intracellular space, lead rapidly binds to the erythrocytes, and permeates soft tissues including bone marrow, brain, kidney and liver. The impacts of lead toxicity in brain and nervous system activities are multifarious with resultant retarded or reversed development, sustained learning disabilities, seizures, coma and ultimately death. The long-run complexities and sequelae of lead exposure are extremely pronounced within the initial two to three years of life, as the critical formative stage of the developing brain is realised. Lead is stored in bone during a prolonged half-life, and jointly or severally increases bone marrow turnover with simultaneous immobilization of lactation, menopause or pregnancy, and elevated blood lead concentrations. Anemia is a crucial aspect of the clinical syndrome emanating from lead toxicity in children due to essentially diminished and compromised red cell viability with concomitant hemolysis.

Lead excretion is via bile and urine with variations in elimination rates regarding the tissue involved in the lead absorption and accumulation. Blood lead content estimations are diagnostic or predictive of acute lead toxicity, whereas the magnitude of erstwhile lead exposure may be estimated based on the body burden of lead as depicted from EDTA, CaNa₂EDTA lead mobilization test [9]. Lead poisoning in children presents as a sentinel for assessment of the magnitude of public and domestic environmental contamination and pollution as well as human health-related issues. The health problems could be remedied through diminished accessibility to lead sources, selective food and water intake devoid of lead and lead-related substances, as well as identification of the consumption and improper disposal of lead-poisoned products. Lead levels in contaminated/polluted precincts are frequently exacerbated in preponderance to the availability and concentrations of other heavy metals [10] and worsen lead-induced Attention Deficit Hyperactivity Disorder, ADHD [11].

Prenatal and Infancy Presentations

Prenatal or in utero exposure to a specific metal could influence fetal growth and development, but anecdotal information exists on the combined effects of metals [12]. It is suggested that in utero combined/joint metal exposures may affect birth outcome; and these associations are liable to vary by the gender of the infant. Also, optimization of body iron status and restriction of lead exposure in early childhood can enhance child health, growth and development as well as proper cognitive assessments [13]. The placenta constitutes a conduit for hazardous environmental lead and an ineffectual barricade to the foetus. During hormonal alterations in pregnancy, lead extrusion into the bloodstream of the mother from deposits in bones and dental structures occur due to prolonged accumulation in a contaminated/polluted precinct or environment. As a neurotoxic element, lead exposure in prenatal and postnatal growth and development may precipitate severe neurocognitive degeneration with resultant Attention Deficit Hyperactivity Disorder, ADHD [14].

Gestational exposure to lead is precarious and deleterious to the welfare, well-being and health of the offspring via diverse mechanisms, such as epigenome modification and DNA methylation, and these are specifically associated with trimester pregnancy [15]. Lead exposure and prenatal stress are comorbid risk factors during development and share biologic substrates [16]. Prenatal stress correlates with transgenerational transfer of behavioral phenotypic changes. Conversely, the transgenerational behavioral or biochemical resultant effect of lead exposure, modifications and alterations of such impacts due to prenatal stress are not adequately explicated. Understanding the long-run and multi-generational consequences of lead exposure is a pertinent measure to elucidate both the geotypic changes underlying the presenting behaviors and the veritable mechanism of heritability [17]. Pathway analysis has depicted altered genes in mechanisms, such as synaptic functionality and plasticity, endocrine homeostasis, epigenetic alterations and neurogenesis. These are indicted in lead-enhanced neurobehavioral impairments and/or inheritances. The etiological factors and pathways to neuropsychiatric disorders are poorly explicated. In that instance, the correlation of neuropsychiatric morbidity to lead exposure and feasible interactions with genetic susceptibility are poorly explained [18].

Neurotoxicity and Neurobehavioral Deficits

Lead is a heavy metal that is a ubiquitous environmental toxicant, and neurotoxic particularly to children. Lead retards nervous system development with substantive neural and cognitive dysfunctionalities. Lead toxicity has been shown to present deleterious impacts on the neurobehavioral development and intelligence disposition of children aged two to four years old

[19]. Children are susceptible and sensitive to lead toxicity due to their unique metabolism, growth and developmental attributes. As a result of the immature feature of the blood-brain-barrier in children, lead easily penetrates the brain and causes extreme and persistent damage to organs and systems [2,6,7,20]. Increasing evidence provides that the CDCP screening guideline of 10ug/dL for the blood lead concentration for children must not be envisaged as the level within which deleterious impacts are not tenable. It was demonstrated that the neurodevelopment of children is inversely correlated to their blood lead contents at <10ug/dL [21].

This correlates with a supralinear association between lead concentrations and neurobehavioral prognosis. Another finding indicated that blood lead level is inchoately sensitive to recognise or detect lead-induced toxicity early [22]; and suggests erythrocyte AChE as a marker for early detection of veritable environmental lead exposure and lead-induced neurotoxicity in children. Universal cognitive potential or general intelligence comprises diverse correlated attributes. Stressors recognized to be predominantly effective in childhood cognitive prowess are not sustained, and are categorically generalized as activities/behaviors which are modifiable for childhood cognitive/cognition enhancement with a potential of complex interplay between the overall ambient and early cognitive development of the child [23]. Merely anecdotal data for persistent neurobiological effect of early lead exposure in later life are available, with lead exposure propagating exacerbated risk of long-term intractable development, health, psychological and social dilemma [24].

Socioeconomic Factors

In combination with lead exposure, low socioeconomic status and environment adversely govern and impair development, especially in children. There is pronounced negative correlation of residential area in elevated lead risk census tracts in children of lower, in contradistinction, to those of higher income brackets. Following increased exposure risk, children of low-income status exhibited less cognitive test scores, reduced cortical volume and constricted cortical surface area [25]. It is suggested that the diminution of environmental perturbations connected with lead exposure risk ameliorates the susceptibility to psychometric deficits or other deleterious impacts in children [2]. Also, blood lead and manganese contents are elevated in iron-deficient infants, and correlates with long-term breastfeeding [5], whereby iron deficiency may culminate in cognitive derangements due to combined or exclusive elevated metal levels. Numerous children, particularly those in less developed countries are subjected to iron deficiency anemia and other nutritional deficiencies which constitute sources of impaired development both neurologically and socioeconomically [2].

Discussion

Globally, lead poisoning or toxicity is principally a pediatric burden. Lead is ubiquitous in the environment, and its prevention is not fool-proof despite measures to prevent the exposure of children to lead, especially in low- and middle-income ambients where the cost of not preventing same may be exorbitant [26]. Lead exposure can severely deteriorate the health of a child, impair the central and peripheral nervous system structures, retard growth and development as well as induce auditory and verbal debilities, trigger attention deficit disorders, assessment, learning and neurobehavioral excoriation. There is no extant safe blood lead level established for children; so, it necessitates prompt and veritable identification and regulation to inhibit or eradicate the lead emission or source [2]. Elevated lead concentrations in the environment are due to both natural processes and anthropogenic activities. The anthropogenic commercial activities include mining, industrial and other environmental polluting processes which may be inimical to the health, well-being, welfare and full potential of the children [27].

It becomes necessary not to relent in the monitoring and evaluation of lead and other toxic trace elements which may constitute environmental and public health risks and hazard as well as clinicopathological factors or determinants in children [28]. The major objective in this instance involves the prevention and/or restriction of trajectories and modalities driving the dissemination and consumption of toxic heavy metals to curb the dissipation of the lives and potentialities of children [2]. These are achievable via the implementation of effective, efficient and efficacious lead exposure programs and remedies [2,29]. These will promote methodologies for the determination of inter alia blood lead levels in a pediatric population, the interactions at low blood levels, monitoring and evaluation of spatiotemporal variations, changing trends and targets in comparison to other precincts and populations of similar [30] and disparate characteristics.

Conclusion

Lead poisoning or toxicity is more debilitating in infancy and childhood because the organs and systems are still precarious entities and not well-developed or still developing and, therefore, more susceptible to adverse effects of the internal and external milieu. The heavy metals or trace elements such as lead can easily penetrate tissues during early childhood growth and development causing excessive excoriation of vital organs and systems. Remedies to lead poisoning are inter alia chelation, abatement and avoidance of further exposure of lead and lead complexes to persons of pediatric age.

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