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Neurological, Nutritional and Cognitive Effects of Environmental Lead Exposure in Infants and Children

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Introduction

Environmental lead exposure is a pertinent public health risk that affects our rural population in Wyandot County, Ohio. Children exposed to lead hazards through toys, home remodeling and contaminated soil and water are at a greater risk for developing neurological, cognitive and behavioral issues than are children not exposed to lead (Cecil et al., 2011). Wyandot County parents are not adequately educated on the risks and consequences of elevated blood lead levels in their children. As a child advocate and director of the Women, Infant and Children program for Wyandot County there is an obligation to educate and inform parents of the risk for cognitive delay and behavioral issues related to elevated blood lead levels in their children. Education on decreasing lead exposure, proper nutrition to decrease the absorption of lead, testing to identify their child's lead level and understanding of the long term risks of lead exposure are priority topics of counseling for parents. Protecting children from exposure to lead is important to lifelong good health. No safe blood lead level in children has been identified and even low levels of lead in the blood have been shown to affect IQ, ability to pay attention and academic achievement. Effects of lead exposure cannot be corrected ("Lead," 2015, p. 1).

Signs, Symptoms and Risk Factors

Lead is known to have acute and chronic effects including loss of appetite, constipation, abdominal colic, decreased IQ, behavioral problems (inattentive, hyperactive or disorganized behaviors), hearing and balance problems, encephalopathy, anemia, growth retardation, delayed sexual maturation, dental caries, cardiovascular disease and renal disease (Bennett, Lowry, & Newman, 2015, p. 19). As compared to decades past, it is rare for a child to demonstrate acutely symptomatic lead poisoning due to the overall reduction in blood lead levels. This reduction is attributed to the ban on lead in gasoline and paint that has been in effect for several years in the United States. Studies have shown that lead is neurotoxic at low blood lead levels, with harmful effects detectable at 2 ug/dL (Bennett et al., 2015).

A child's age is an important risk factor for elevated blood lead levels. The incidence of lead poisoning in children generally peaks around 18-30 months of age, mainly because of normal development and behaviors at that age (Schnur & John, 2014, p. 239).

Children are more likely to have elevated lead levels as compared to adults for several reasons. Children have increased hand-to-mouth behaviors, lead exposure from pica, an immature blood-brain barrier leading to greater neurotoxicity, increased lead absorption and concomitant iron deficiency anemia.

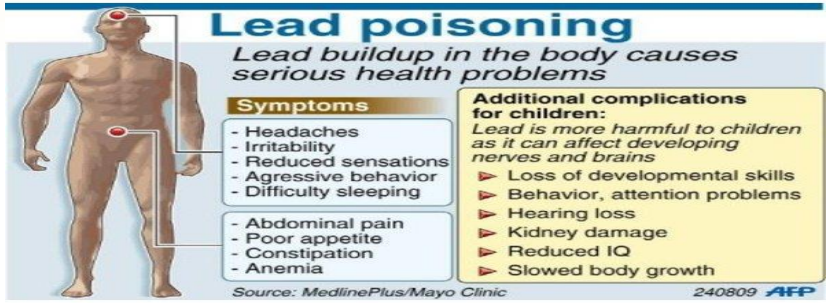
Children under three years of age appear to be particularly vulnerable to lead's neurotoxic effects, due to their rapidly developing neurological systems (Schnur & John, 2014, p. 240). Children from lower socio-economic backgrounds living at or below the poverty level have a more significant risk of lead poisoning than do children from wealthier neighborhoods. This is due to the condition and age of housing available. Other risk factors to consider are children whose parents use ethnic remedies for ailments and recent immigrants, refugees or international adoptees. Children with nutritional deficiencies such as iron, zinc, calcium, protein and vitamin C are at increased risk for enhanced absorption of ingested lead (Schnur & John, 2014, p. 240).

Pathophysiology of Lead

Lead absorption is dependent on several factors, including the physical form of lead, particle size ingested, gastro-intestinal tract transit time and the nutritional status of the person ingesting the lead. Lead absorption is augmented in the presence of iron, zinc and calcium deficiency as well as malnutrition, high-fat intake and calorie deficient diets (Holstege, Huff, Rowden, & O'Malley, 2013, p. 1). Lead absorption is decreased if phosphorus, riboflavin, vitamin C and vitamin E are in the diet.
Absorbed lead is exchanged primarily among blood, soft tissue, including the liver, kidneys, lungs, brain, spleen, muscles and heart, and mineralizing tissues such as bones and teeth. In the blood, lead is primarily found within the red blood cells. Lead is distributed throughout the body and made available to other tissues via the blood supply.
Lead has an effect on heme biosynthesis, causing anemia at high blood levels. Lower levels of lead in the system cause microcytosis (i.e. decreased mean corpuscular volume and mean corpuscular hemoglobin) and a compensatory increase in the number of red blood cells. Lead irreversibly binds to the sulfhydryl group of proteins causing impaired function without any discernible threshold (Holstege et al., 2013, p. 3). The enzymes delta-aminolevulinic acid dehydratase, which catalyzes the formation of the porphobilinogen ring, and ferrochelatase, which catalyzes the incorporation of iron into the protoporphyrin ring are both compromised by lead.

The inhibition of these enzymes may begin with a lead level as low as 5 ug/dL. If ferrochelatase is inhibited or inadequate iron is present, zinc is substituted for iron and zinc protoporphyrin concentrations increase. The major consequences of this effect is the reduction of circulating levels of hemoglobin. Basophilic stippling of erythrocytes may be present (Holstege et al., 2013, p. 3). Lead toxicity leads to damage in kidney function associated with albuminuria, reduced glomerular filtration rate and decreased creatinine clearance in lead exposed populations.
Histopathologically, renal impairment associated with lead poisoning is characterized by proximal tubular nephropathy, glomerular sclerosis, and fibrosis in peritubular and interstitial lesions (Kwon et al., 2015, p. 120). There is increasing evidence that the kidney may play a role in the clearance of erythrocytes. Infiltration of erythrocytes and iron deposition has been observed in proximal tubules and tubular lumens of patients with acute glomerulonephritis and hematuria as well as acute renal failure. Proximal tubular epithelial cells are capable of phagocytizing erythrocytes, a process known as erythrophagocytosis. Erythrophagocytosis is primarily carried out by macrophages in the spleen and liver and occurs when aged or damaged erythrocytes are phagocytized and cleared from systemic circulation (Kwon et al., 2015, p. 120).

This process is mediated by externalized phosphatidylserine (PS) on the outer membrane and by PS-bearing microvesicles. More than 99% of blood lead accumulates in erythrocytes. Lead exposure can lead to the externalization of phosphatidylserine and the generation of microvesicles in erythrocytes, which appears to be associated with increased erythrophagocytosis by renal tubular cells. Erythrophagocytosis seems to be associated with increased reactive oxygen species generation, induction of nephrotoxicity biomarkers, transforming growth factor B up-regulation and decreased cell viability of renal tubular cells (Kwon et al., 2015, p. 126).
Lead affects many biological activities at different levels of control, including the voltage-gated channels and the first, second and third messenger systems. Lead also affects the postnatal reorganization of the brain through the following mechanisms: decreased oligodendrite density, myelin deposition, cortical synaptogenesis, induction of precocious glial cell differentiation, blockage of voltage-sensitive calcium channels, interference with neurotransmitters, disorganized synaptic pruning and interference with protein kinases (Holstege et al., 2013, p. 2). Lead also impacts the auditory nervous system by affecting the conduction in the distal auditory nerve and the auditory pathway in the lower brainstem.



Significance

Lead's impact on the auditory nervous system may contribute to the learning delays seen in children with elevated lead levels. Subtle impairments of auditory processing could have profound effects on learning. Knowledge of lead's detrimental effects on the hearing process may trigger a clinician to refer for a hearing evaluation in a child with an elevated lead level.
Lead's inhibition of ferrochelatase and delta-aminolevulinic acid dehydratase in the blood leads to a decrease in circulating hemoglobin, which results in a decreased ability of the blood to deliver oxygen to vital organs and tissues (Holstege et al., 2013). Decreases in hemoglobin may lead to fatigue, irritability and decreased cognitive function.
More than 99% of blood lead accumulates in erythrocytes. Iron deposition in the kidneys were found in patients with various renal diseases suggesting that the retention of iron rich erythrocytes in the kidney may play a role in the pathogenesis of kidney diseases (Kwon et al., 2015, p. 120). Most of the lead absorbed in the body is excreted either through renal clearance or through biliary clearance in the feces (Holstege et al., 2013, p. 2). Knowledge of lead clearance would prompt a clinician to screen renal function in a child with an elevated lead level.
Leads detrimental effects on synapses, myelin deposition, neurotransmitters and calcium channels are attributed to cognitive delays, behavioral issues, developmental delays and learning deficits in children with elevated lead levels (Holstege et al., 2013, p. 2). A clinicians understanding of the long term effects of these deficits would warrant periodic follow up with behavioral and cognitive screenings to assess the degree of developmental delays.

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Nursing Implications

- Screen all children for lead
- Nutrition assessment
- Dietician referral if needed
- Lead source education
- Risks of lead exposure
- Proper referrals to State agencys/Health Depts.

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