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Lead Exposure in Pregnancy

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Lead has long been suspected to be a teratogen. The main sources of lead are lead-based paint, contaminated soil, and occupational exposure. Lead is transferred freely through the placenta as early as 12 weeks gestation and a maternal-fetal barrier to lead is not known. (O'Halloran et. al., 1992).

Recent data indicate that lead is toxic at levels which were previously thought to be safe. Today, levels of 10-15 ug/dL are of concern when just a few years ago, a blood lead level of 25 ug/dL was the lowest level for concern (O'Halloran et. al., 1992). According to the Center for Disease Control (CDC), the acceptable blood lead level is <10 ug/dL. However, even low level of lead in the blood is thought to carry a risk for subtle CNS damage (West et. al., 1992). In 1984 it was estimated that half a million pregnant women were at increased risk due to lead exposure (Landrigan, 1990). Although these numbers seem to be decreasing, lead exposure during pregnancy continues to be a serious problem. This RISK||NEWSLETTER will address the issue of lead exposure in pregnancy.

MOBILIZATION OF LEAD

Lead is stored in bone where it accumulates throughout an individual's lifetime. Mobilization of these lead stores may occur during pregnancy and lactation due to bone demineralization. Theoretically, a significant history of childhood lead poisoning could lead to adverse pregnancy outcome later in life. Animal studies support this theory, lead mobilization, however has not been directly studied in human pregnancies. The possibility of childhood lead poisoning leading to adverse pregnancy outcome is based on secondary data. However, there are studies that have shown no increase in lead levels during pregnancy (O'Halloran et. al., 1992).

CONGENITAL MALFORMATIONS

A variety of minor malformations associated with an increased maternal blood lead level during pregnancy. These malformations include skin tags and papillae, hydroceles, hemangiomas, lymphangiomas, and undescended testicles in males. There is, however, no particular pattern of malformations that is associated with lead exposure. Nor have major malformations been associated with an increased maternal lead level (Needleman et. al., 1984; Wong et. al., 1992). Needleman et. al. (1994) did not find an association between lead levels and decreased birth weight, shortened gestation, apgar scores, respiratory distress, or jaundice. Wong et. al. (1992), however, reviewed two studies in which shortened gestation and low birth weight were associated with maternal lead levels. One study of 192 pregnancies showed a 1/2 week reduction in gestation for every 10 ug/dL increase in maternal blood lead levels. Also, low-level lead exposure was associated with a 114 g decrease in birth weight for every 10 ug/dL increase in maternal blood lead levels. A second study of infants with increased cord

blood lead levels found that their gestational age was significantly decreased when compared with infants whose cord blood levels were not elevated. Even though the studies of Needleman (1984) and Wong (1992) do not agree as to whether lead levels are associated with a decrease in gestational age or birth weight, all the studies consistently find an increased incidence of miscarriage and stillbirth among women with increased lead levels.

NEUROLOGICAL IMPAIRMENT

Wong et. al. (1992) reviewed four prospective studies on the neurologic effects of low-level in-utero exposure to lead. The studies used the Bayley Mental Development Index scores (MDI) as measurement of neurobehavioral development in infants. The first study followed 249 children from birth until two years old. Prenatal lead exposure was estimated at birth from cord blood. These children were categorized into two groups: low lead levels with a mean level of 1.8 ug/dL and high lead levels with a mean level of 14.6 ug/dL. When the MDI was given at 12, 18, and 24 months of age, the high level group consistently showed a deficit ranging from 4 to 8 points when compared to the low level group. This was statistically significant at the 95% confidence interval with a probability of $p=.0001$.

The second study followed 132 infants for the first year of life. Lead levels were estimated by maternal blood lead levels and cord blood levels. The mean maternal blood level was 6.5 ug/dL, with the highest lead level being 14.7 ug/dL. The mean umbilical cord blood level was slightly lower, 5.8 ug/dL. Even at this seemingly low exposure, the infants showed abnormal reflexes, decreased muscle tone, and abnormal neurological soft signs at birth. At 12 months of age, the lead exposed infants had a statistically significant lower MDI score when compared to non-exposed infants.

The MDI scores were also lower in lead exposed infants in the third study. In 192 pregnancies that were exposed to low lead levels, infants had an average decrease of 2.25 in their MDI scores for each 10 ug/dL increase in in-utero lead levels. The fourth study evaluated 2 year old children and showed similar results. Every 10 ug/dL increase in lead levels produced a 2.0 point decrease in MDI scores.

Children whose cord blood lead levels were increased (10-25 ug/dL) and who also showed lower MDI scores until 24 months of age showed no difference in the McCarthy Scales of Children's Abilities when compared to non-exposed children at 57 months of age. One possibility is that the two scales (MDI and McCarthy) evaluate different abilities and lead exposure does not affect those abilities measured on the second test (McCarthy). Another possibility is that these children recovered and compensated for their deficits sometime between 24 and 57 months of age (Bellinger et. al., 1990). Over 2,600 children were followed prospectively. Their cord lead levels were taken at birth to determine prenatal lead exposure and lead levels were measured in shed deciduous teeth to determine postnatal lead exposure. Any behavior problems identified when these children were 8 years old was associated with the postnatal lead exposure rather than the prenatal exposure (Bellinger et. al., 1994). This indicated that behavior problems in school-aged children are more likely due to postnatal lead exposure and not low-level prenatal lead exposure.

OCCUPATIONAL EXPOSURES

Lead is the third most common occupational exposure in women (Bentur et. al., 1991). As stated earlier, past studies have shown that women who have occupational exposure to lead tend to have more preterm deliveries, miscarriages, and stillbirths. There is also an increased risk for minor anomalies. The World Health Organization (WHO) recommends that the expressed limit for women who work with lead should not exceed 30 ug/dL (O'Halloran et. al., 1992). However, if a woman does have an occupational exposure, her mean lead levels should not exceed other women's levels in the same geographic location.

There has been no evidence of adverse fetal outcome among pregnancies with paternal lead exposure.

Among males who work with lead, there have been chromosome alterations and abnormalities in sperm count, vigor, and morphologic features observed. These effects, however, tend to cause infertility rather than adverse fetal effects (Bentur et. al., 1991). Previous reports have show a higher frequency of stillbirths and miscarriages in pregnancies with paternal exposure to lead. It is now believed that lead brought into the home on contaminated work clothes, thereby elevating maternal lead levels, was responsible.

RECOMMENDATIONS

Women who are at risk for having increased lead levels during pregnancy should have their blood levels monitored. If their levels are elevated, every attempt should be made to reduce their exposure. Chelating agents which reduce lead levels are not recommended for use during pregnancy because their teratogenic effects are unknown.

Three hundred forty-nine African American women who took prenatal vitamins generally had lower lead levels than women not taking prenatal vitamins. Specifically, an inverse association between lead levels and antioxidants such as vitamin E and ascorbic acid was observed (West et. al., 1994). Deficiencies in iron, calcium, and zinc can lead to increased lead absorption. Therefore it is important that at-risk women have good nutrition (O'Halloran et. al., 1992).