

Brief Communication

Lead levels in children with developmental delay. A hospital-based study

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Lead is a neurotoxin that has been shown to adversely affect cognitive functioning and development in children.¹ Infants and children are more vulnerable to lead exposure because of the more rapid airway and gastro-intestinal absorption, hand-to-mouth activities, and increased susceptibility of the developing brain to the neurotoxic effects of lead.¹ Moreover, children with developmental delay are at even greater risk of developing lead toxicity due to their habits such as chewing on objects or sucking their fingers, and due to the higher risk of iron deficiency anemia related to their nutritional habits.¹ Iron deficiency promotes lead absorption from the gut and leads to higher blood lead levels in children.¹ While no evidence-based threshold for the toxic effects of lead has been identified, the Center for Disease Control and Prevention (CDC) statement on childhood lead set 10 µg/dL as the toxic lead level.² The CDC recommended routine screening of all children 1-6 years if >12% of children in the community have toxic levels (>10 µg/dL).² Otherwise, targeted screening is advised only for those thought to be at high risk.²

This cross-sectional, case controlled study was conducted at Jordan University Hospital, Amman, Jordan over a period of 10 months (April 2005 - February 2006), and its aim is to determine whether children with developmental delay are at an increased risk of lead toxicity compared to the general child population in Amman, and to determine accordingly whether these children should be considered as a high risk group and thus, actively targeted for screening.

Seventy children with global developmental delay and 140 controls were included. In this study group, etiologies for developmental delay were as follows: cerebral palsy (n=24), epileptic encephalopathy (n=13), neurometabolic disorders (n=7: mitochondrial encephalopathy [5], metachromatic leukodystrophy [1], Neimann Pick disease [1]), genetic syndromes (n=4: Down's syndrome, Usher syndrome, Silver-Russel syndrome, trisomy 18), hypothyroidism (n=1), post meningitis (n=1), undetermined etiology (n=20).

The average age was 38.80 ± 18.52 months for the developmentally delayed group, and 42.07 ± 18.80

months for the control group. Table 1 shows the baseline characteristics of patients in each of the 2 groups. The patient group included more boys than the control group. Parents in the control group were more educated than parents of the patient group. Lead levels >10 µg/dL were reported in 4 of the delayed children (5.7%) and in 3 of the controls (2.1%), the difference between the 2 groups was not statistically significant ($p=0.17$). This is in contrast to results from previous studies, which showed significantly higher toxic lead levels in delayed children (12%), and advised routine lead screening for this high risk group.¹ The mean blood levels were 5.27 µg/dl in the patient group and 4.62 µg/dl in the control and the difference between them was not statistically significant ($p=0.12$).

Analysis using the chi-square test for the patient group showed no significant relation between toxic lead levels >10 µg/d in the patient group and iron status, fathers working as drivers, or the use of Kohl (traditional eyeliner).

The relatively low lead levels in our study may be attributed to the lower environmental contamination with lead in Jordan compared to many other places worldwide.³ Lower blood levels of lead (<10 µg/dL), while not typically associated with potentially fatal encephalopathy, are also neurotoxic in children and have lasting effects on neurobehavioral functioning. More than half of the delayed children in our study had lead levels between 5-9.9 µg/dL. These levels, although below the conventional toxic level, can still produce cognitive and behavioral deficits. Several risk factors have been reported to increase blood lead levels. Several studies have shown that children exposed to Kohl have higher levels of lead in their blood.¹ Although approximately 20% of the children in our study had been exposed to Kohl, there was no correlation between the use of Kohl and toxic lead levels. A possible explanation is that

Table 1 - Patient characteristics in developmentally delayed children and in controls.

Patient characteristics	Patients (n=70) n (%)	Controls (n=140) n (%)	P-value
Male to female ratio	2.2:1	1.2:1	0.06
Working mothers	12 (17.1)	43 (30.7)	0.03
<i>Mother's level of education</i>			0.00
University	6 (8.6)	41 (29.3)	
Diploma	18 (25.7)	40 (28.6)	
High school	30 (42.9)	41 (29.3)	
Less than high school	16 (22.9)	18 (12.9)	
<i>Father's level of education</i>			0.00
University	14 (20)	72 (51.4)	
Diploma	6 (8.6)	22 (15.7)	
High school	26 (37.1)	34 (24.3)	
Less than high school	24 (34.3)	12 (8.6)	

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the Kohl used by Jordanians is mostly lead-free. This was confirmed by the parents who reported using only natural plant oils to prepare Kohl.

Another risk factor reported to be strongly associated with elevated blood lead level is the manual job of the father.¹ This association has been previously reported and linked to the exposure of children to the contaminated clothes at home.^{1,4} Although 18.6% of fathers work as drivers has no significant association between this occupation and blood lead levels >10 µg/d, emphasizing the fact that environmental contamination with lead in Jordan is low. Although 32.9% of children with developmental delay had an iron deficiency (leading to increased lead absorption from the gut) compared to 23.6% of children in the control group, there was no significant relation between iron deficiency and toxic lead levels.

In conclusion, this study has several limitations that may impede the generalizability of its results to other settings. Firstly, the study is a hospital-based and includes only those children living in Amman. Further studies in varying settings will be needed in order to determine whether the results of this study are generalizable to other populations. The percentage of developmentally delayed children with toxic blood lead levels in this study did not warrant the inclusion of this group for targeted screening. However, this result must not be over-reassuring. Given that low blood lead levels can adversely affect cognition, and that children with developmental delay already have lower baseline levels of cognitive functioning, even minor additional impairment due to lead exposure can have detrimental effects on their functional capacity. Consequently, even in countries with low lead levels where screening is not indicated by CDC standards, lead exposure

prevention strategies are strongly advised for children with developmental delay.

At low levels of lead contamination, prevention strategies include simple environmental measures such as hand washing before meals, wet wiping of hard surfaces, frequent washing of soft toys, and avoiding sanding off old paint in the home. These environmental measures have been shown to substantially reduce the amount of lead ingested by children¹ and thus, should be encouraged, with special emphasis on children with developmental delay, because they can least afford to lose any of cognitive, motor, or behavioral strengths they possess.

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