

Comparison of blood lead levels in 1 –7 year old children with and without seizure

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ABSTRACT

Objectives: Lead is a nonessential metal and is not a natural constituent in humans. Lead is very toxic for children, especially to the central nervous system. We studied and compared blood lead levels in children aged 1-7 years old with and without convulsions.

Methods: In this study we randomly measured blood lead levels in 206 children referred to Imam Reza Hospital, Pediatric Emergency Ward and Outpatient Clinic, Mashad, Iran from December 2001 to June 2003. There were 95 children with convulsions and 111 without convulsions.

Results: There were no significant differences in the

mean value of age, place of residence and economic status between the 2 groups (*P* values were 0.20 for age, 0.14 for place of residence and 0.76 for economic status). The mean blood lead level \pm SD in the convulsive group was 126.53 ± 35.91 $\mu\text{g/lit}$ and in the non-convulsive group was 118.03 ± 32.10 $\mu\text{g/lit}$ (*p* = 0.70).

Conclusion: This study showed that the blood lead levels in convulsive patients is not statistically significant compared to the non-convulsive group and routine measurement is not advised.

Neurosciences 2005; Vol. 10 (3): 210-212

Seizure is common in children and is seen in approximately 10% of children, and its most common etiology is extracranial.¹ In developed countries, the measurement of the lead level of children living in the urban area is recommended as lead is one of the heavy metals around us and in children with blood lead levels more than 100-150 $\mu\text{g/lit}$, it will damage many organs such as the brain.² In high-risk regions, public screening is of economic value and in low risk regions the measurement of lead level is carried out according to existence of risk factors.^{3,4} A constant lead level more than 100-150 $\mu\text{g/lit}$ indicates an unacceptable risk to cause constant central nervous system (CNS) damage, and with the lead level increasing, practical and developmental skills become lower, and lowering of the lead level will improve the cognitive condition and brain function in children

after 2 years. The clinical diagnosis of poisoning is difficult before severe CNS damage, therefore the diagnosis depends on laboratory results.⁵⁻⁹ The aim of this study is to measure the lead levels of children with seizure and to compare this with the lead levels of children without seizure, to decide whether lead level has the ability to affect seizure incidence.

Methods. This study was carried out on 206, 1-7 year old children referred to the Pediatric Emergency Ward and the Pediatric Clinic of Imam Reza Hospital, Mashhad, Iran. Ninety-five children with seizures were chosen, in addition to 111 children without seizures chosen as the control group. The seizures of the children were not due to brain infection or metabolic disorders. The children were classified into 3 groups according to the

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Received 20th October 2004. Accepted for publication in final form 26th March 2005.

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economic level of their families: group one (low economic condition): families with a monthly income of less than 1500000 Rials; group 2 (intermediate economic condition): families with a monthly income of more than 1500000 Rials and less than 5000000 Rials; and group 3 (high economic condition): families with a monthly income of more than 5000000 Rials. In this study, the children's blood was sent to the poisoning laboratory in heparinized tubes. The Perkin Elmer 3030 atomic absorption spectrophotometer was used with the heated graphite absorption system, and the standard addition calibration method. The method of obtaining data was interview with the mothers of the children, and physical examination of the patients with recording of the children's blood lead levels. The data were obtained through questionnaires prepared before the study. The obtained data included age, name, place of residence (such as city center, village, around the city) and economic condition. The obtained data were analyzed by SPSS version-11.5 software and variables were analyzed by T-Test and a $p < 0.05$ was considered significant.

Results. In this study, the lead levels were classified into 3 groups (<100, 100-200, >200) and each 100 µg/lit was classified into one group. According to T-test, there was no meaningful difference between case and control groups ($p=0.06$). This study showed that 52 (25%) children had blood lead levels less than 100 µg/lit and 154 (74.8%) of the referred children (case and control groups) had a lead level more than 100 µg/lit, namely 77 (81.1%) in the case group and 77 (69.4%) children in the control group with an odds ratio of 1.88%. The age of the referred children in the case group was 38.26 ± 17.6 months, and in the control group was 41.56 ± 9.46 months. In the case group with a standard error of 1.80 and in control group with a standard error of 1.84, and according to $p = 0.20$, there was no significant difference in the ages of these 2 groups. The ages of the children were classified into 6 groups, and each 12 months was classified in one group. As shown in **Table 1**, 60% of children in the case group and 50.4% of children in the control group were less than 3 years old, and according to T-test there was no meaningful difference between these 2 groups ($p = 0.34$).

In this study we found that in the case group, 57 (60%) children were living in the city and 21 (22.1%) children were living in the suburbs and 17 children (17.89%) were living in the village. In the control group, 81 children (73%) were living in the city and 17 children (15.3%) were living in the suburbs and 13 children (11.7%) were living in the village, and according to a $p = 0.14$, the place of residence did not have any meaningful difference

Table 1 - Distribution of age in the case (n = 96) and control (n = 111) groups.

Age in months	Groups			
	Convulsive n	(%)	Non-convulsive n	(%)
12-23	33	(34.7)	34	(30.6)
24-35	24	(25.3)	22	(19.8)
36-47	17	(17.9)	18	(16.2)
48-59	12	(12.6)	18	(16.2)
60-71	8	(8.4)	19	(17.1)
>72	1	(1.1)	0	(0)
$p = 0.34$				

Table 2 - Family economic status in the case (n = 96) and control (n = 111) groups.

Economic status	Groups			
	Convulsive n	(%)	Non-convulsive n	(%)
Good	7	(7.4)	17	(15.3)
Medium	25	(26.3)	36	(32.4)
Low	63	(66.3)	58	(52.3)
$p = 0.76$				

between these 2 groups. In the case group, 7.4% of children had a good family economic status and in the control group 15.3% had a good family economic status, according to a $p = 0.76$, this difference was not statistically meaningful (**Table 2**). The lead level in the case group was 126.53 ± 35.91 µg/lit (mean \pm SD). The minimum lead level was 13 µg/lit and the maximum lead level was 247 µg/lit. In control group the lead level was 118.03 ± 31.10 µg/lit (mean \pm SD). The minimum lead level was 53 µg/lit and the maximum lead level was 298 µg/lit; and according to a $p = 0.70$, there was no statistically significant difference between these 2 groups.

DISCUSSION. Lead existed in 4 conditions and is one of the dangerous heavy metals. A lead level more than 100 µg/lit can be harmful for many organs such as the brain, and an acute increase of lead levels to more than 1000 µg/lit can cause encephalopathy, which presents with some acute signs such as vomiting, altered level of consciousness and seizure before complicating to encephalopathy itself.⁴ In this study 206, 1-7 year old children were studied, 95 of whom suffered from seizure, and the cause of their seizure was not metabolic disorders nor infections. In addition, 111

healthy or sick children that did not have seizures were used as the control group. There were no meaningful differences between these 2 groups for age, economic level and place of residence. Although the mean lead level of the patient group was 6.72 µg/lit more than the control group, this difference was not statistically significant ($p = 0.70$). Therefore, lead level probably does not have any role in the children's seizures, and measurement of the lead level is not necessary in patients with seizure without other clinical manifestations to indicate lead poisoning. However, the important point is that 154 (74.8%) of the children, in both groups, had a lead level more than 100 µg/lit ($p = 0.54$ and odd ratio 1.88).

In a study carried out in California on 5115, 1-6 year old children, the lead level was more than 100 µg/lit in 371 children (7.25%).¹⁰ In a study carried out in Uruguay the mean lead level was 96 µg/lit and 36% of the studied cases had a lead level more than 100 µg/lit.¹¹ In a study in Waxi City, China the mean lead level was 82 µg/lit and 27.3% of the cases had a lead level more than 100 µg/lit.¹² In a study on 463, 6 months - 6 year old children in Massachusetts, 20.2% of children had a lead level more than 100 µg/lit.¹³ In a study in Vancouver, Canada on 177, 2-3 year old children, 8.1% of children had a lead level more than 100 µg/lit.¹⁴

Comparing this study with other studies, we found that the lead level of the children in this study was higher, and a higher percentage of children are at risk for lead poisoning. In these cases (lead more than 100 µg/lit), it is recommended that another laboratory test is carried out after 1-3 months,^{4,5} and parental education for decreasing the lead level is also recommended.¹⁵ Lead levels more than 100 µg/lit can be harmful to the intelligence quotient of the children,¹⁶ and the increase in the lead level may be due to an increase of the lead in air, water, dust and food.¹⁷

In conclusion, we found that the lead levels of children with seizures was not significantly higher than children without seizures, and its measurement for children with seizures is not routinely recommended. Also, according to the high lead level of the studied children, it is recommended to identify the source of contamination in other studies.

References

1. Michael VJ. Seizures in childhood. In: Behrman RE, Kliegman RM, Jenson HB, editors. Nelson textbook of pediatrics. 17th ed. Philadelphia (PA): WB Saunders; 2004. p. 1993-2009.
2. Rolnick SJ, Nordin J, Charnary LM. A comparison of costs of universal versus targeted lead screening for young children. *Environ Res* 1999; 80: 84-90.
3. Kember AR, Bordley WC. Cost effectiveness analysis of lead poisoning strategies following the 1997 guidelines of the center for disease control and prevention. *Arch Pediatr Adolesc Med* 1998; 152: 1202-1208.
4. Markowitz M. Lead poisoning. In: Behrman RE, Kliegman RM, Jenson HB, editors. Nelson textbook of pediatrics. 17th ed. Philadelphia (PA): WB Saunders; 2004. p. 2358-2362.
5. Jolian J, Chislom JR. Lead poisoning. In: McMillan JA, Deangelis CD, Feigin RD, Warshaw JB, editors. Oskis Pediatrics. 3th ed. Philadelphia (PA): Lippincott Williams & Wilkins; 1999. p. 629-635.
6. Sibert J, Fleming PJ. Poisoning, accidents and sudden infant death syndrome. In: Campbell AGM, McIntosh N, editors. Forfar, Arneil's Textbook of Pediatrics. 5th ed. New York (NY): Churchill Livingstone; 1998. p. 1708.
7. Binshy MS. What do parents know about lead poisoning? *Arch Pediatr Adolesc Med* 1998; 152: 1213-1218.
8. Kumar A, Dey PK, Singela PN. Blood lead levels in children with neurological disorders. *J Trop Pediatr* 1998; 44: 320-322.
9. Johnston MV, Goldstain GW. Selective vulnerability of the developing brain to lead. *Curr Opin Neurol* 1998; 11: 689-693.
10. Gallert GA, Wagner GA, Maxwell RM, Moore D, Foster L. Lead poisoning among low income children in Orange County, California. A need for regionally differentiated policy. *JAMA* 1993; 270: 69-71.
11. Schutz A, Schotz A, Barregard L, Sallsten G, Wilske J, Manay N, et al. Blood lead in Uruguayan children and possible sources of exposure. *Environ Res* 1997; 74: 17-23.
12. Goa W, Li Z, Kaufmann RB, Jones RL, Wang Z, Chen Y, et al. Blood lead levels among children aged 1 to 5 years in Waxi City, China. *Environ Res* 2001; 87: 11-19.
13. Dalton MA, Sargent JD, Stukel TA. Utility of a risk assessment in identifying children with lead exposure. *Arch Pediatr Adolesc Med* 1996; 150: 197-202.
14. Jin A, Hertzman C, Peck SH, Lockitch G. Blood lead level in children aged 24 to 36 month in Vancouver. *CMAJ* 1995; 152: 1077-1086.
15. Kimbrough RD, Levois M, Webb DR. Managment of children with slightly elevated blood levels. *Pediatrics* 1994; 93: 188-191.
16. Center disease control and prevention (CDC). Blood lead level United States 1999-2002. *MMWR Morb Mortal Wkly Rep* 2005; 54: 513-516.
17. Presidents Task Force on Enviroment Health Risks and Safety risks to children, US Department of Housing and Urban Development. Eliminating childhood lead poisoning: a federal strategy targeting lead paint hazard, 2000. Washington DC: US Department of housing and urban development. Available at URL: http://hud.gov/offices/lead/reports/fedstrategy_2000.pdf.