Blood Lead Levels in Children with Neurological Disorders

by Ashok Kumar, P.K. Dey, P.N. Singla, R.S. Ambasht, and S.K. Upadhyay

Department of Paediatrics, Institute of Medical Sciences and Department of Botany, Faculty of Science, Banaras Hindu University, Varanasi, India

Summary

Blood lead levels were measured by atomic absorption spectrometry in 82 children suffering from various neurological disorders (cerebral palsy 42, seizure disorders 35, acute encephalopathy of unknown origin 5) and in 28 healthy children, aged 1 to 12 years. Mean blood lead levels were 11.96 ± 10.97 μg/dl in control children and 19.30 ± 17.65 μg/dl in children with neurological disorders. A significant number of control children as well as those who had neurological disorders were found to have blood lead concentrations of ≥10 μg/dl and ≥20 μg/dl, the cut-off limits for lead poisoning and medical evaluation, respectively. Blood lead levels were, statistically, elevated in children with cerebral palsy compared to controls. Children with pica behaviour exhibited higher blood lead concentrations.

Introduction

The burden of lead toxicity is greatly underestimated because most cases of lead poisoning are clinically inapparent. Recent studies indicate that lead can cause cognitive and behavioural deficits in children even at low levels of exposure and some of these effects may be irreversible. Children with neurological disorders should be evaluated for lead poisoning, either because lead may cause these disorders or these conditions may be associated with increased lead ingestion and/or absorption. Exposure to lead of children with pre-existing neurological handicaps can have serious consequences for them, because it can further impair their residual cognitive, motor, or behavioural abilities. The environment of many developing countries is heavily contaminated due to unrestricted use of lead in industry and automobile fuel. This poses a significant health hazard for children. There is scarcity of information on blood lead levels in common neurological disorders in children. This prompted us to undertake the present study.

Materials and Methods

Eighty-two children with various neurological disorders, namely, cerebral palsy (n = 42), seizure disorders (n = 35), and encephalopathy (n = 5), aged 1 to 12 years, were selected randomly from the ‘Outdoor’ and ‘Indoor’ sections of the Department of Pediatrics, University Hospital, Banaras Hindu University, Varanasi, India. Twenty-eight age-matched healthy children served as controls. Cerebral palsy was defined as a non-progressive disorder of movement and posture. The diagnosis of seizure disorder was made on the basis of history and electroencephalographic findings. Only those children were included who had definite history of seizures. Encephalopathy was defined as generalized disturbance of cerebral function of acute onset, manifesting as coma with or without seizures, without any obvious cause. Children with the following conditions were excluded from the study: inflammatory brain disease, degenerative brain disease, intracranial space-occupying lesions, febrile seizures, and encephalopathy due to well defined cause.

Venous blood was collected in heparin-containing deionized vials using deionized syringes and needles. Blood lead was estimated by atomic absorption spectrometry. Care was taken to prevent contamination from the environment of blood specimens during sample preparation and analysis. The lower limit for detection of lead by this instrument was 1 μg/dl. Other investigations included electroencephalography and cranial computed tomography. The data were analysed statistically by Student’s t-test.

Results

Table 1 shows blood lead levels in various study groups. Blood lead levels were statistically elevated in children with cerebral palsy in comparison to control children. Blood lead levels of ≥10 μg/dl and ≥20 μg/dl were found in a significant number of control children as well as in those with neurological disorders. Children with partial and generalized seizures had comparable blood lead values.

Pica behaviour was reported in three control children and in 15 children with neurological disorders. Blood lead levels were higher in these children than in those who had no history of pica. However, the difference was
significant only in control children (23.33 ± 16.29 μg/dl vs. 10.66 ± 9.77 μg/dl) and not in those who had neurological disorders (26.00 ± 19.09 μg/dl vs. 17.81 ± 17.25 μg/dl).

**Discussion**

Lead has no known biological value. Thus, the ideal blood lead level is 0 μg/dl. However, due to constant exposure to lead in the environment, blood lead levels tend to increase. Until recently blood lead levels as high as 30 μg/dl were considered acceptable. It is now being recognized that impairment of cognitive function begins to occur at levels as low as 10 μg/dl, even though clinical symptoms are not observed. The current Centers for Disease Control (CDC) guidelines recommend a cut-off value of 10 μg/dl for diagnosing lead poisoning and a value of 20 μg/dl necessitating medical evaluation. The mean blood lead levels in both groups of children in the present study were above the cut-off value for lead poisoning. This is in contrast to US children where blood lead levels average between 4 and 6 μg/dl.

The major sources of lead in the environment are leaded petrol and lead-based paint. Ingestion and inhalation are the principal routes of lead absorption in children. The normal hand-to-mouth activity of young children is an effective way to transfer lead-laden dust from the environment into the body. Pica is an important risk factor for lead toxicity, although it need not be present. In the present study, the control children with a positive history of pica had higher blood lead levels than those without such a history. On the other hand, blood lead levels in children with neurological disorders were not influenced by the presence or absence of pica. It is likely that pica behaviour of neurologically handicapped children might go unnoticed as they are generally neglected and poorly supervised.

The results of the present study demonstrated significantly higher blood lead levels in children with cerebral palsy compared to control children. This could be due to excessive hand-to-mouth activity and pica behaviour, often present in these children, and their poor nutritional status consequent to feeding difficulties, poor appetite, and social neglect. Deficiencies of iron, zinc, and calcium are known to enhance lead absorption from the intestine. This is of grave concern since nutrient deficiencies, especially iron deficiency, are so common in many parts of the world. Approximately one-third of children with cerebral palsy in this study had blood lead levels of 20 μg/dl or more. A number of studies have found that for every 10 μg/dl increase in blood lead level, there is a lowering of the mean intelligence quotient by 4 to 7 points. This would produce catastrophic effects on children with pre-existing handicaps because they can least afford to lose whatever cognitive, motor, or behaviour strengths they possess. Although cerebral palsy has been recognized as a sequel of lead encephalopathy, we consider that elevated blood lead levels in the present study were the consequence rather than cause of cerebral palsy because the majority of them had well defined risk factors for this condition, such as birth asphyxia/trauma, prematurity, low birthweight, kernicterus, and meningitis/encephalitis, and so on.

Children with seizure disorders and encephalopathy also demonstrated elevated blood lead values. Seizures have been well described in survivors of lead encephalopathy. The seizures are recurrent, focal, or generalized tonic-clonic. None of our patients had a history of preceding encephalopathy before the onset of seizures. All children appeared to have primary seizure disorders as no cause was found despite investigations. Elevated blood lead levels in these children may simply be a reflection of exposure to a lead-contaminated environment. Of the five children with acute encephalopathy, three had blood lead levels above 20 μg/dl (23, 40, and 44 μg/dl). Blood lead levels in these children were not high enough to account for clinical manifestations. Generally, encephalopathy develops when blood lead levels exceed 100 μg/dl. However, occasionally the condition may develop at lower levels.
References


