LEAD : THE INSIDIOUS ENEMY OF CHILDREN

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SUMMARY : *Recent studies have shown that any level of lead in blood higher than 10 µg/dL could have long term harmful effects on young children's health. They can easily absorb enough lead to reach this level if society does not stop using lead in gasoline, paint, water pipes and other products which still contain lead. Considering the sources of lead, childhood lead poisoning is a preventable illness if all the nations at risk pay enough attention to the subject.*

Key Words : *Lead Poisoning, Blood Lead Level, Children.*

INTRODUCTION

Childhood lead poisoning is one of the common paediatric health problems particularly in industrialized nations throughout the world. The primary route of exposure to lead is the gastrointestinal tract and secondarily the respiratory tract. Children in the oral stage are particularly susceptible to lead's toxic effects because they have more hand to mouth behaviour than adults, such as thumb sucking, nail biting and eating with dirty hands. They absorb more lead than adults, around 50 % as compared to 8 % for adults (14). As most poisoned children show no symptoms, the vast majority of cases still go undiagnosed and untreated. If present, early symptoms may include stomach ache and cramps, fatigue, frequent vomiting, poor appetite, constipation, headache, irritability and sleep disorders. Physicians may diagnose some of these early symptoms as a virus or other mild illness and delay treatment. The CDC statement (8) recommends universal blood lead screening "unless it can be shown that the community in which these children live does not have a childhood lead poisoning problem ".CDC in the USA recommends that blood lead levels of all children at twelve months of age should be checked and the tests repeated at twentyfour months of age if resources are available.

Working in the paediatric outpatient clinic of St.Louis University (U.S.A), I noticed that all the children were tested for lead poisoning when they were about one year old. Although there was no significant symptom or sign observed, results higher than 10 µg/dL were checked after a few months. If the repeated results were again high, the child was referred to St.Louis Lead Clinic for environmental check-up or medical therapy to avoid later harmful effects of lead on the central nervous system.

Lead poisoning is widespread throughout the world. Although some studies have shown that there is a relationship between lead absorption in childhood and unemployment (30), low social class (33), or inadequate care (19), these problems require

further examination under different circumstances. No geographical area or racial or ethnic population is found to be particularly susceptible to lead poisoning (8).

It is not possible to select a single number to define lead poisoning. A number of studies have shown that low blood lead levels between 0.48 µmol/Lt (10 µg/dL) and 0.97 µmol/Lt (20 µg/dL) are harmful (8, 16, 24). The CDC in the USA lowered its definition of an elevated blood lead level from 40 µg/dL to 30 µg/dL or higher in 1975, from 30 µg/dL to 25 µg/dL or higher in 1985 and from 25 µg/dL to 10 µg/dL or higher in 1991 (8). However, it is not clear how to best treat children who have blood lead levels between 10 µg/dL and 25 µg/dL.

Different lead indicators used in studies (blood, teeth or hair) represent different exposure periods. Although most studies have relied on blood lead levels to classify subjects, it is not an adequate marker of exposure to lead. As a marker of recent exposure, blood lead may return to normal levels even after excessive exposure. venous blood is usually preferred for the measurement of lead in blood because capillary blood may include skin lead contamination that falsely elevates blood lead. It is reported that only 31.9 % of 47 children with capillary blood lead higher than 10 µg/dl had a confirmed venous blood lead higher than 10 µg/dL (4).

SOURCES

For decades, lead poisoning was known simply as an occupational disease of adults. But today we know that the environment in which children live is full of lead. Lead based paint, leaded gasoline and solder are the major sources and air, food, water, dust and soil are the pathways.

In the USA, lead based paint and broken lead impregnated plaster, when ingested, are the most significant sources of high dose lead exposure and symptomatic lead poisoning for children (8, 9). Lead based paint was used in about 74 % of housing units before 1980. Since 1977, lead concentrations in paint for household use, interior and exterior residential surfaces, toys and furniture have been restricted by law to 0.06 % of dry weight (8). There is no uniform standard for a safe amount of lead in painted surfaces. "State and federal goverment in the USA use values ranging from 0,7-1.2mg/cm^2 of wall,when lead is measured using a portable X-ray fluorescence analyser or a standard of 0,5 % lead by weight when tests are performed using laboratory analysis" (8). However, according to St.Louis Lead Clinic, lead based paint remoued during the renovation of old housing units is still the most important source of lead.

High leaded ink from printed material,especially from colour sections of newspapers and magazines is also frequently ingested by toddlers. For the primary prevention of childhood lead poisoning, the family should be educated about peeling paint and chewable materials that may contain lead based paint and this kind of paint must be removed from housing, if possible, by a qualified professional. The burning of waste oil,coloured newsprint, battery casings or lead painted wood can cause serious poisoning.

The greatest effect of leaded gasoline is to contaminate dust, soil and food which children ingest. Contaminating the air they inhale is a relatively less important effect. Street dirt and surface soil in many countries where leaded gasoline is still used,have extremely high lead content (34). Air lead levels measured at fixed monitoring stations can only approximate the amounts of lead children inhale, they do not measure the exposure from lead deposited on soil,dust and then ingested. The American Public Health Association has encouraged efforts by the Environmental Protection Agency to reduce and rapidly eliminate lead added to gasoline (34). Between 1976 and 1980, the mean blood lead level in the USA population decreased from 15.8 to 10 µg/dL, parellelling the reduced use of leaded gasoline (1) (Fig 1).

In some studies,blood lead levels were significantly higher in summer (6, 25) and fall (18).In the United States through the 1980's, both gasoline usage and lead concentration were found to be higher in warmer months (26). The other reason for higher blood lead levels in summer might be the increased exposure of children to outdoor lead sources while playing outside.

In an uncompleted screening study conducted in the paediatric outpatient clinic of Ankara Dışkapı SSK Hospital, among the first 40 children aged between 9-24 months, six had blood lead levels higher than 10 µg/dL. All these children were considered to be in a low risk group considering sources of lead except gasoline. Even the very early part of this study indicates that 15 % of the children aged between 9-24 months, an important period for CNS development, were already included were in the CDC

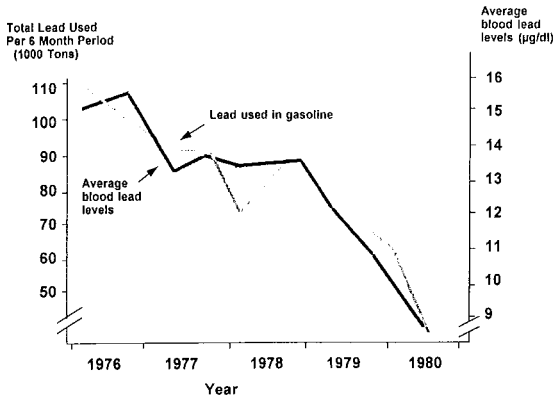

Fig - 1: Change in blood lead levels in relation to a decline in use of leaded gasoline, 1976-1980 (8).

definition for "childhood lead poisoning" although none showed any specific symptom. What is the source then? What are the insidious harmful effects awaiting these children?

As it is not easy to measure the lead concentration of painted walls in Turkey because of the absence of portable analysers for this work and as most children do not often come in contact with painted materials, newspapers or magazines at home for economic and educational reasons, leaded gasoline should be considered the main widespread source of childhood "chronic" lead poisoning in Turkey.

Although some countries have reduced the use of leaded gasoline, the benefit to children's health is not yet known in many parts of the world including Turkey where leaded gasoline is still used extensively (32). The control of airborne lead emissions should be an international health priority in these countries.

Particles of airborne lead deposited in soil and dust are often important lead sources for children who can easily take lead from dust and soil by their very often exploratory hand to mouth behaviour. It is suggested that infants who do not receive adequate physical and social stimulation may engage in more mouthing behaviour (5) and ingest more leaded paint, soil, and dust. Excess lead absorption is reported to be a greater problem in cities than in suburbs (25).

A more recent source of lead is community water systems. The source of lead in tap water is the material of the distribution system such as lead pipes. Lead glazed pottery can release large amounts of lead into food and drink, particularly if the glaze is chipped. In addition lead dust can cling to the hair, skin, clothes, shoes and be carried home by parents with lead related hobbies or work (7).

EFFECTS

It is already known that very severe lead exposure in children (higher than 55 µg/dL) can cause encephalopathy with permanent damage, coma, convulsions, and even death. Lower levels between 20 µg/dL and 55 µg/dL have toxic effects on major body systems; blood, kidneys, and brain (8) (Fig 2). Anaemia from lead toxicity has a microcytic morphology as in iron deficiency and often goes with it. It causes a decrease of red blood cell survival, impairment of globulin synthesis and inhibition of haem synthesis. These effects are reversible with medical theatment. Low iron levels caused by iron deficiency anaemia increase lead absorption and aggregate its toxic effects (17).

Renal toxicity includes nonspecific degenerative changes in the renal tubular lining and cellular necrosis of the proximal convulated tubule. This damage causes Fanconi's Syndrome which was first noted by Chisolm in acute lead poisoning (13).

Lead has toxic effects on capillaries in the brain, resulting in severe cerebral aedema, actual loss of neurons and increase of glial cells. Some affected children experience seizures, mental retardation and behaviour disorders (13), because loss of neu-

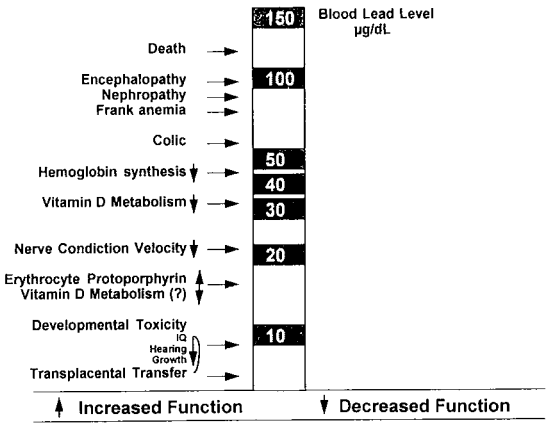

Fig - 2: Lowest observed effect levels of inorganic lead in children (8).

rons and gliasis is irreversible.

Historically, only the acute effects of lead poisoning were known. In recent studies, researchers have tried to answer the question: "How much harm does blood lead level lower than 25 µg/dL cause to the developing nervous system of a child?".

At blood lead levels as low as 10 µg/dL many harmful effects begin, such as decreased intelligence, impaired neurobehavioural development (10, 30), decreased stature and growth (27, 29), decreased hearing acuity (28) and decreased ability to maintain a steady posture (3).

In a study of children with low socioeconomic status, Hawk and Schroder (15) have shown that IQ decreased linearly as blood lead level increased. This result strengthens the consensus of current research concerning of the lead effect on a child's IQ (21). However, there are many important social factors which may affect the findings in this study and the relationship needs to be investigated using different study designs. In another study by Needleman et al., the impaired function of children with high lead levels appears to be an early adverse effect of exposure to lead (22).

Lead poisoning in childhood has been seen to cause a seven-fold risk of poor school results, greater absenteeism, lower class ranking, impairment of reading skills and deficits in vocabulary, problems with attention and fine motor skills, reaction time and hand-eye coordination (24). Needleman et al. reported that the harmful effects of lead on academic progress and cognitive functioning of children continued into young adulthood (22) (Fig 3). These long term findings in young adults emphasize the practical importance of early detection and abatement of lead in the environment before it is absorbed by children.

The lead levels of mother and foetus are found to be highly correlated during pregnancy; this means that lead crosses the placental barrier (34). Shukla et al. (29) reported that "the expected stature of a child born to a mother with a prenatal blood lead level more than 7.7 µg/dL is about 2 cm shorter at 15 months of age if, postnatally, the infant incurred a 10 µg/dL blood lead increase during the 3 to 15-month interval compared with an infant who has no increase".

Infants whit higher lead levels in umbilical cord blood had lower Crude Mental Development Index scores during the first two years of life (2, 11) (Fig

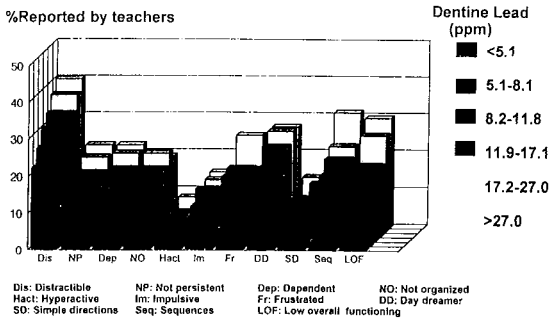

Fig - 3 :Distribution of negative ratings by teachers on 11 class room behaviours in relation to dentine lead concentrations (22).

4). Although a few studies (12, 23) have reported no association between lead exposure and reduced gestational age, in most studies maternal and cord blood lead levels of 10-15 µg/dL appeared to be associated with reduced gestational age and reduced weight at birth (20).

Lead has no known physiological value. Researchers have not yet completely defined the effects of blood lead levels lower than 10 µg/dL on central nervous system function. However, even these levels may be associated with harmful effects that will be clearer with further research.

"Like war and poverty, lead poisoning is a man-made disease and theoretically should be accessib-

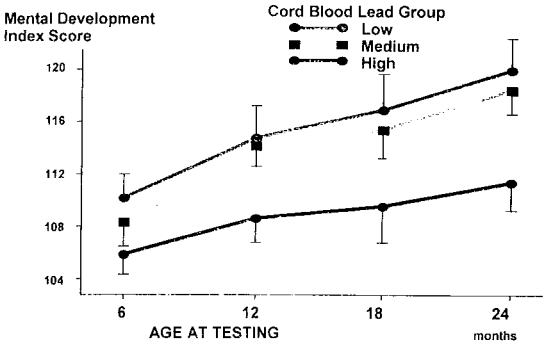

Fig - 4 :Mean mental development index scores at four ages in infants according to the lead level in umblical-cord blood (2).

le to human intervention" (31). In the USA, efforts are increasingly focused on preventing lead poisoning. There are many lead clinics working in collaboration with hospitals and primary care outpatient clinics. The Lead Poisoning Control Service in St. Louis is a good example, for these clinics with mobile units, portable x-ray analysers and well educated staff work for prevention at all levels. Primary prevention consists of public education, strengthening the resistance of children and environmental clean up. Early recognition and prompt treatment are secondary prevention. Tertiary prevention includes treatment of chronic conditions, prevention of sequalae and rehabilitation.

Where to we stand in Turkey? To become aware of the long term harmful effects of lead should be the first step to working against them as quickly and effectively as possible, to the same international purpose; prevention of lead poisoning in young children.

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REFERENCES

1. Annest JL, Pirkle JL, Makuc D : Chronological trends in blood lead levels between 1976 and 1980. N Eng J Med 1983; 308 : 1373-1377.
2. Bellinger D, Leviton A, Watemaux C, Needleman H, Rabinowitz M : Longitidunal analysis of prenatal and postnatal lead exposure and early cognitive development. N Eng J Med 1987; 316 : 1037-1043.
3. Bhattacharya A, Shulka R, Bornschein R, Dietrich K, Kopke JE : Postural disequilibrium quantification in children with chronic lead exposure : a pilot study. Neurotoxicology 1988; 9 : 327-340.
4. Binns HJ, Le Bailly SA, Poncher J, Kinsella TR, Saunders SE : Is there lead in the suburbs? Risk assessment in Chicago suburban pediatric practices. J Pediatries 1994; 93 : 164-171.
5. Bornschein RL : Influence of social factors on lead exposure and child development. Environ Health Prespect 1985; 62 : 343-351.
6. Centers For Disease Control. Increased absorption and lead poisoning in young children. Washington DC: U.S. Dep of Health Education and Welfare, Public Health Service 1975.
7. Centers For Disease Control. Preventing lead poisoning in young children. A statement by the Centers For Disease Control. U.S. Department of health and human services / Public Health Service. Atlanta 1985; 5-31.
8. Centers For Disease Control. Preventing lead poisoning in young children. A statement by the Centers For Disease Control. U.S. Department of health and human services / Public Health Service. Atlanta 1991; 1-30.
9. Chisolm JJ : Jr. Lead Poisoning. Scientific American 1971; 224 : 15-23.
10. Davis JM, Svendsgaard DJ : Lead and child development. Nature 1987; 329 : 297-300.
11. Dietrich KN, Krafft MK, Bornschein RL, Hammond PB, et al : Low level fetal lead exposure effect on neurobehavioral development in early infancy. J Pediatrics 1987; 80 : 721-730.
12. Ernhart CB, Wolf AW, Kennard MJ, Erhad P : Intrauterine exposure to low leves of lead: the status of the neonate. Arch Environ Health 1986; 41 : 287-291.
13. Goyer RA : Lead Toxicity. In Chisolm JJ Jr. and O'Hara DM. Lead absorption in children, management, clinical and environmental aspects Baltimore, Urban-Schwarzenberg 1982; 21-237.
14. Hammond PB : Metabolism of Lead. In Chisolm JJ Jr and O'Hara DM. Lead absorption in children, management, clinical and environmental aspects 1982; 11-20.
15. Hawk BA, Schroder SR, Robinson G, et al : Relation of lead and social factors to IQ of Low-SES children : A Partia (Replication). Am J Ment Deficiency 1986; 91 : 178-183.
16. Lyngbye T, Hansen NO, Trilingsgaard A, Beese I, Grandjean P : Learning disabilities in children : Significance of low level lead exposure and confounding factors. Acta Paed Scand 1990; 79 : 352-360.
17. Mahaffey KR : Nutritional factors in lead poisoning. Nutr Rev 1981; 39 : 353-362.
18. Marrero O, Meigs JW, Stark A, Fitch QR : Seasonal patterns in children's blood lead levels: a second peak in late winter. Conn Med 1983; 47 : 1-5.
19. Millar CR, Schroeder SR, Mushak P, Dolcour JL, Grant LD : Contributions of the care - giving environment to increased lead burder of children. Am J Ment Defic 1980; 84 : 339-344.
20. Moore MR, Goldberg A, Pocock SJ, et al : Some studies of maternal and infant lead exposure in Glasgow. Scottish Med J 1982; 27 : 113-122.
21. Needleman HL, Gatsonis CA : Low level lead exposure and the IQ of children. JAMA 1990; 263 : 673-678.
22. Needleman HL, Gunnoe C, Leviton A, et al : Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N Eng J Med 1979; 300 : 689-695.
23. Needleman HL, Rabinowitz M, Leviton A, Linn S, Schoenbaum S : The relationship between prenatal exposure to lead and congenital anomalies. JAMA 1984; 251 : 2956-2959.
24. Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN : The long term effects of exposure to low doses of lead in childhood. An 11 year follow-up study. N Eng J Med 1990; 322 : 83-88.

25. Nordin JD, Rolnic SJ, Griffing MJ : Prevalence of excess lead absorption and associated risk factors in children enrolled in a midwestern health maintenance organization. J Pediatrics 1994; 93 : 172-177.
26. Robinowitz M, Needleman H, Burley M, Finch H, Rees J : Lead in umblical blood, indoor air, tap water and gasoline in Boston. Arch Environ Health 1984; 39 : 299-301.
27. Schwartz J, Angle C, Pitcher H : Relationship between childhood blood lead levels and stature. Pediatrics 1986; 77 : 281-288.
28. Schwartz J, Otto D : Blood lead, hearing thresholds and neurobehavioral development in children and youth. Arch Environ Health 1987; 42 : 153-160.
29. Shukla R, Bornschein RL, Dietrich KN : Fetal and infant lead exposure : Effects on growth in stature. J Pediatrics 1989; 84 : 604-612.
30. Smith M, Delves T, Landsdown R, Clayton B, Graham P : The effects of lead exposure on urban children Dev Med Child Neurol 1983; 47 : 1-54.
31. Urban GA : Self percieved needs of families with lead poisoning in children. Doctoral thesis, St. Louis USA 1988.
32. Von Schirnding Y, Bradshaw D, Fuggle R, Stokol M : Blood lead levels in South African innercity children. Environ Health Perspect 1991; 94 : 125-130.
33. Winneke G, Hrdina KG, Brockhaus A : Neuropsychologic studies in children with elevated tooth lead concentrations : I. pilot study. Int Arch Occup Environ Health 1982; 51 : 169-183.
34. Wynkoop MS : Childhood Lead Toxicity, A Preventable Epidemic. Doctoral thesis, St. Lous, USA 1987.