Lead exposure

Implications for general practice

Donald Howarth

Background

There is increasing awareness of the importance of environmental pollution as a cause of health problems.

Objective

This article explores the effects that lead can have on children's health and draws on two recent pollution episodes to highlight the need for continued community and medical vigilance.

Discussion

High levels of lead in the environment can have significant adverse effects, particularly on the developing brains of children. The main effects are intellectual loss and behavioural disturbances such as attention deficit and hyperactivity disorder. Exposure can occur from activities such as the removal of lead based paints around the home. More commonly, significant lead exposure in children occurs in towns that mine, process or transport lead. General practitioners in communities hosting lead industries need to be alert to the possibility of a diagnosis of lead poisoning.

Keywords

child, health; environmental medicine; general practice

High levels of lead in the environment can have adverse effects on both children and adults. However, of major medical concern is the toxic effects lead can have on children.

Sources of lead in the environment have changed over the past 100 years. In the 19th and early 20th centuries, lead was found in paint, pottery glazes, cast lead soldiers (tin soldiers), lead-reinforced candle wicks, artist paints and electrical solder. While lead in artist paints and electrical solder is being addressed, solder available on the market in Australia still contains lead. Lead-free solder is now readily available and is the standard in Europe. Importantly, while most household paint is now lead-free, some nonhousehold paints (especially bright yellow industrial paints) still contain lead. Also, renovation of old houses may involve scraping and burning of lead based paints off walls and window frames. A typical house built in 1900 may contain hundreds of kilograms of lead in the paint layers.[1]

During the 20th century, the development of tetraethyl and tetramethyl lead as antiknock agents for petrol, and the massive increase in vehicle consumption of leaded petrol, has deposited a layer of fine lead particles over much of our environment, especially in inner city areas. This was the major population source of lead in children's blood until relatively recently. The elimination of leaded petrol in many countries has resulted in a major reduction in the levels of lead in children's blood.[2–4] Until about 1999 in Australia, lead was still used in solder to join copper pipes in houses, and the brass in Australian taps still contains 4% lead.[5,6] Recently, copper plumbing has largely given way to polyvinyl chloride (PVC) plumbing and some of this PVC contains lead carbonate ($PbCO_3$) to mop-up free chlorine atoms and prevent them from degrading the plastic. Lead may leach out of such pipes.[7]

Causes of lead exposure

There are some individuals whose activities increase their exposure to lead, such as cigarette smokers and shooters who fire lead bullets or who eat animals killed with lead bullets or shotgun pellets.[8,9] People who repair radiators were historically at risk, but this is safer now that plastic tanks are used instead of soldered brass tanks.

Children can be exposed to lead by parental activities such as melting down car batteries to manufacture sinkers and the removal of lead based paints in the home. More commonly, significant lead exposure in children is restricted to towns where community-wide exposure results from mining, processing or transporting lead. Towns such as Broken Hill (New South Wales), one of the largest lead ore mine sites in the world, and Port Pirie (South Australia), where there is a major lead smelter are areas of significant risk (*Case example 1*). Both have ongoing monitoring and intervention programs that have been successful in reducing

the lead exposure of children. Unfortunately, the same cannot be said of Esperance (Western Australia) where birds literally dropped dead in mid-flight, effectively becoming the monitors of lead pollution when the official system of monitoring failed (*Case example 2*).[10,11]

Lead metabolism and measurement

Environmental lead is ingested via the gut using the same divalent cation pump that absorbs iron, or occasionally via the lungs if particles are small enough. The divalent cation pump in the gut is more active in iron-deficient children, therefore deficient children absorb more of the lead they ingest. In addition, pica (soil eating) in these children may mean they are ingesting lead via contaminated soil. It is therefore important to measure iron status and correct any deficiency in any child found to have lead toxicity.

Once absorbed, lead has a very high affinity for porphobilinogen synthase, a haemoglobin synthesis enzyme found in red blood cells. As a result, when testing for lead toxicity it is important to measure the whole blood lead level and not the serum lead. A blood lead level gives a measure of absorption in the past 3 months. Absorbed lead can be excreted through the kidneys or accumulate in bone where it can be substituted for calcium in the mineral component. A small but important part of the lead in our blood finds its way to the central nervous system.

Bone lead is generally stable but it can mobilise during periods of bone resorption, such as during pregnancy or lactation.[12] Measuring the lead content of bone by X-ray fluorescence is possible, but not available in Australia outside research settings. Importantly, hair and urine lead levels do not give consistent estimates of recent exposure. In some research settings, the lead content of decidual teeth is used as a measure of past exposure.

The first published Australian account of lead poisoning in children was in 1892 in Queensland when a series of 10 patients with colicky abdominal pain and paralysis were described.[13] Later the connection to children chewing weathered lead based paint on verandah rails was made.[14] This resulted in the 1922 banning of house paints containing lead in Queensland, with other states following some time later. Then, the blood lead level at which it was thought brain damage occurred was about 60 micrograms per decilitre. Subsequent studies showed that such levels cause considerable permanent damage. Indeed, over the ensuing decades the level of concern was reduced to 40 µg/dL then 25 µg/dL and finally 10 µg/dL as studies showed lower levels caused just measurable intellectual loss.

For 2 decades now the accepted blood lead level of concern has been 10 µg/dL. However, recently there have been suggestions that levels below 10 µg/dL may cause damage.[15] The evidence in favour of a further lowering was recently reviewed by the American Centres for Disease Control and the Australian National Health and Medical Research Council (NHMRC) and rejected in favour of keeping 10 µg/dL as a pragmatic level. In a practical sense, it appears that an average child exposed to that blood level throughout its infancy and childhood will have a measurable reduction in intelligence.[16]

It is now also possible to identify the source of lead in a patient's blood by analysis of the isotope composition. Lead has four stable isotopes. By measuring the isotope ratios in a patient's blood

Case example 1. Broken Hill and Port Pirie

Broken Hill is a town in an area of desert in far western New South Wales near the South Australian border. It is the site of a huge deposit of lead, zinc and silver. The town has developed around ore deposits, with open cut workings, tailings heaps and old smelter dumps in the centre of the town. In the early days lead poisoning was common. However, for much of the past century it was felt the problem had been resolved after smelting of ore in the centre of town ceased and the dust storms were controlled by a concerted revegetation program. In 1981, a small survey of school children's blood lead levels was taken to prove that there was no problem. In retrospect, that survey was not reassuring as the upper limit of accepted blood lead levels was then 40 µg/dL, which is four times the accepted upper limit today.

It has been suggested that the awareness of the recent lead problem in Broken Hill came from noticing that dogs were being poisoned.[22] However, in the 1980s, a geochemist working in Broken Hill measured the lead content of ceiling cavities and found up to almost 4% lead in the often 10 cm of ceiling dust in houses.[23] Meanwhile, experienced miners raised concerns about the re-opening, by open-cut mining, of the old workings in the middle of town and the author, along with a paediatrician, reported to the Department of Environment that 32 paediatric patients had been sampled and returned worryingly elevated levels of lead.

Action was taken to survey children's blood lead levels after the issue was raised on ABC television in a report by journalist Justin Murphy. Subsequently, there has been ongoing monitoring of paediatric blood lead levels in Broken Hill since 1991, which has confirmed a major problem of childhood lead exposure. Meanwhile, suggestions that the lead was coming from petrol and old house paint, rather than the mines, was laid permanently to rest by blood lead isotope analysis which showed the majority of the lead in the blood of Broken Hill children came from the local mines.[24] In addition, soil contamination surveys showed that lead pollution fell rapidly as the distance from the mines increased.

Since then, there has been a concerted campaign to reduce lead levels in children in Broken Hill that has included public health education, home remediation and reducing the flow of new lead into the environment while stabilising or removing the most polluted soils. The program has been very successful. Over a 16 year period the mean childhood blood lead level in Broken Hill has fallen by 65%.[22–26]

Importantly, a major study of lead pollution and its impact on the children in the town of Port Pirie had resulted in the Port Pirie Lead Implementation Program being established there in 1984.[27] Port Pirie receives its lead from the Broken Hill mines by rail and is the site of a very large lead smelter. The problem in Broken Hill might have been recognised earlier had officials made the connection that there was likely to be similar lead exposure in the two towns.

Note: The author worked as a GP in Broken Hill in the 1980s and 1990s

Case example 2. Esperance

Esperance is a port town on the southern coast of Western Australia that exports large quantities of grain and in more recent times, iron ore and nickel.

In 2005, the port started exporting finely ground lead carbonate concentrate in a process that involved sending the concentrate along a semi-enclosed conveyor belt and dropping it into the open holds of ships. Importantly, Esperance is a very windy place, the material was finely ground and lead carbonate is a particularly bioavailable form of lead.

In 2007, lead exports came to an abrupt halt after several thousand birds died in the town. Local pharmacist Michelle Crisp campaigned to keep the plight of the birds firmly in the local spotlight and demanded an explanation. After a year of testing the cause was found to be lead poisoning. Concern soon shifted to the possibility that humans may also have been poisoned. Testing of water tanks revealed that many of the tanks near the port had been contaminated.[28] This was significant because many Esperance citizens prefer to drink tank water due to the strong taste of the local reticulated bore water. Blood testing then revealed a number of elevated levels, especially, as would be expected, among children. Most of the cases with elevated levels were clustered around the port. Isotope testing of lead in the dead birds, water tanks and the children's blood proved the major source of lead was the port.[10]

Note: The author currently works as a GP in Esperance

and comparing it with known sources of lead in their environment, it is possible to determine the relative contributions of these environmental sources in the blood.

Lead exposure in children

Children are much more vulnerable to lead poisoning than adults. This is partly because they absorb four times more lead than adults and are more likely to be exposed to lead from crawling around floors and hand-to-mouth activity. Typically, children's blood lead levels peak at about 1.5–2 years of age. The growing child's brain also has a much greater sensitivity to lead than the adult brain and exposure to lead can have significant permanent effects on the developing brain. The main effects are a reduction in intelligence and behavioural disturbances such as attention deficit and hyperactivity disorder (ADHD). Elevated lead levels have also been shown to be associated with a higher rate of criminality, including violent crime.[2,17,18]

Lead exposure in adults

In adults, high levels of lead can result in decreased haemoglobin synthesis and subsequent anaemia, as well as renal damage and hypertension. At levels above 80 µg/dL, encephalopathy occurs. Previous occupational exposure to lead has been shown to be associated with subtle cognitive deterioration and brain matter loss.[19] Lead freely crosses the placenta, therefore pregnant women need to avoid exposure (including by avoiding home renovation projects such as sanding back and burning off old paint).[20]

The importance of community and GP awareness

In communities where lead is mined or handled it is possible to limit community exposure to lead. For instance in Derby (Western Australia), careful monitoring from the beginning of potential pollution was accompanied by minimal exposure of children to lead, despite the ore being transported down the main street of the town in trucks.[21] However, there is a danger in assuming that authorities and potential polluters will be effective in monitoring risk.

Communities hosting lead industries need to be alert to the risks, and should feel they have the right to ask questions regarding how these risks are being managed. General practitioners in these communities need to be alert to the possibility of a diagnosis of lead poisoning and advocate on their patients' behalf when the systems that should protect our health fail. General practitioners should also be aware of the risk of lead exposure in families who are involved in activities such as lead sinker production or home renovation.

Conclusion

While serious lead poisoning is now rare, there is always the risk of population exposure in areas where lead is mined, transported or processed. Population exposure to low levels of lead may make only very small differences to the intelligence of any one individual. However, if an entire population has its intelligence distribution curve moved a few units, major social and economic consequences may occur. General practitioners and communities should remain vigilant in their attempts to persuade corporations and government bodies to minimise lead in the environment.

Author

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