HK J Paediatr (new series) 2016;**21**:273-279

Review Article

Hong Kong Lead Burden: Poisoning in the Former Years

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Abstract Lead poisoning is seen as a past disease. The difficulty of recognising these patients and identifying the source is described. Surveys were done, but the standard of toxicity level has been rapidly changing. Dramatic clinical presentations were not common in Hong Kong, being often asymptomatic or missed. Legislation on lead sources was expectedly slow when only relying on collections of full evidence. The picture described in this paper on what has been in Hong Kong may give us more understanding of the present interest in drinking water contaminated with lead from smelting material used to connecting water pipes.

Key words Fisherman; Herbs; Lead poisoning

Despite ancient and earlier descriptions, the protean issues of lead (Pb) surfaced very gradually over centuries. Childhood Pb poisoning was described in 1892 in Australia, in full in 1933 by Russell, and in infants in 1914 by Blacken in USA. Yet action against Pb was slow, problems seen as remote. Long follow-up effects described of 20 children with symptomatic Pb poisoning in 1943 by Byers increased awareness but only as a concern in children with severe symptoms. The following is a description of the changing standards in the past decades.

The earliest Hong Kong report on Pb poisoning was from Queen Elizabeth Hospital, as Tsang and Wong reviewed 15 cases of chronic Pb poisoning admitted in 1964 to 1967. The things to look for in Pb poisoning are now well documented, including Pb colic, anaemia with basophilic stippling of red cells, searching for gingival lead lines, evaluation of neurophysiologic and renal function. Technology now allows testing of hair, teeth, or fingernails for Pb, radiographic imaging or X-ray fluorescence of long bones, and other tests. Describing what it has been in Hong Kong may give us more understanding of the present.

The Lead Burden in Fisherman

In 1981, Queen Mary Hospital (QMH) admitted a well built 21-month-old girl from a fishermen's family with 10 days fever with bronchopneumonia, vomiting, lethargy and dullness, followed 5 days later by deteriorating consciousness for encephalopathy with EEG changes and increased CSF protein. Bilateral diaphragmatic paralysis in two weeks required ventilator care. She had haemoglobin (Hb) 6.8 g/dL, basophilic stippling of the red cells, radiologically dense epiphyseal line, and transient peripheral neuropathy demonstrated by loss of tendon jerks, electromyogram and nerve conduction. Blood lead level (BLL) was >120 μg/dL (5.9 μmol/L). Nephropathy was asymptomatic with low sodium at 127 mmol/L, tubular loss of concentrating ability, and rising creatinine 1.7 fold in 2 weeks. She needed ventilation care for 2½ months even with chelation therapy but eventually recovered with no neurological deficit. There were no obvious common sources of Pb exposure. Her mother and two elder siblings

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Received October 26, 2015

also showed clinical, haematological and radiologic evidence of Pb poisoning.

These dramatic clinical presentations were not common in Hong Kong. Retrieval of prior hospital paediatric records for Pb poisoning noted a few admissions in 1966, 1975, and 1977 of three children related to battery factory, and two related to fisherman or proprietary infant herbs. To note, the normal BLL in those days was allowed up to 50 μg/dL (2.5 μmol/L). Pb poisoning cases might be more common, being often asymptomatic or missed. Noting Pb weights in fisherman, the Department of Paediatrics jointly with the Department of Chemistry of the University of Hong Kong studied the magnitude of the problem by a pilot study of Pb poisoning in fishermen's children, and, an overall study of Pb levels in children of Hong Kong.

For fisherman, between 1984-85, the team took effort to get on board of boats in Aberdeen.[1] Their environment for Pb sources was studied. Boats were mostly painted with varnish, which should not be Pb related. Scattered Pb weights not been used in fishing nets were often found over the place. Some children were seen biting on Pb weights as a pretence play of line fishing. Pica was common. Occasionally, Pb weights were seen in or around openings of water tanks. The children were studied with BLL and with a questionnaire on developmental, health, and nutritional status. Thereon, 52 families and 125 children 1-14 years old on 34 boats were studied with capillary sampling for BLL, precautions well taken against sample contaminations.[2] 19.2% had BLL >30 μg/dL, while this occurred in 34.6% families and 32.4% boats, suggesting that it is family and boat related. More than half of them have BLL over 40 μg/dL.[2] Eight of these children with BLL >30 μg/dL confirmed with venous sampling were subsequently admitted to the hospital to have a more detailed assessment of body state and living environment, and included a psychometric assessment by psychiatrist.[3] 5 of them also demonstrated positive EDTA provocative test indicating unacceptable Pb overload requiring chelation therapy. To note, the revised normal BLL at that time was <30 μg/dL.

For Pb levels in normal children, a survey in 1984 used both BLL (which determination was expensive for screening and needed larger volume blood samples not acceptable to children), and red cell zinc protoporphorin (ZPP) levels to screen. 5,608 school students 6-17 years from 37 schools territory-wide were first studied for ZPP. BLL were determined if ZPP high (above 50 μg/dL). 0.18% had BLL >30 μg/dL. The mean BLL 23.6±17.0 μg/dL in the school with mainly fishermen's children was significantly higher than those in other schools.[2,4] To clarify, randomly selected 700 primary school students were checked for ZPP and BLL together, and revealed ZPP not very satisfactory for screening. Later studies also confirmed this as the considerable individual ZPP variability, poor sensitivity at lower Pb exposure, poor specificity and delayed changes in unstable exposure conditions indicate against this test for screening programs.[5] Here, probably missing 2/3 high BLL student, the incidence of BLL >30 μg/dL could reach 0.7% range (rather than 0.18%). The mean BLL was 14.0±5.1 μg/dL (17 to 45.0 μg/dL), similar to or lower than those in large cities then. Looking for association with children BLL, the occupation of parents and mouthing habits are more important than the environmental factors such as floor level of residence and the time spent on the street. Among the students who have confirmed high BLL, half of them have their father's occupation associated with increased risk. In high BLL children with no other family members affected, fathers worked as a clerk, an interior decorator, two building workers, and a fishing equipment sales, while in those having family members also affected, fathers worked as a fisherman, paint colour controller a news printer, and a car mechanic. Family members were more affected with high-risk occupations.

In that mean time (1985-87), three other fishermen's children from the Aberdeen and Cheung Chau fishing community were admitted to QMH because of convulsions and Pb poisoning with BLL of 193, 186 and 66 μg/dL respectively. Two presented like febrile convulsions. Only one (with BLL 186 μg/dL) could be related to having Pb-containing herbal medicine.

The Aberdeen fishermen were further studied. A total of 37 schools and over 100 adult fishermen were studied. Results showed significant increase in urinalysis of N-acetylglucosaminidase (NAG), which may signify insult by Pb poisoning. In 1988, another survey checked 123 fishermen 14-70 years old from 31 families for capillary BLL, ZPP, serum urea, creatinine and urate, urine NAG and urinalysis,[6] and a similar questionnaire on possible sources of Pb contacts and health status. These were analysed statistically against non-fisherman control subjects. The mean BLL and ZPP level were significantly higher in fishermen than controls, but no significant difference in Hb. Two adults have BLL >25 μg/dL. Taking values as elevated if above the upper limit of the control group (BLL >l5.9 μg/dL, ZPP >63.7 μg/dL), the prevalence of relatively increased BLL and ZPP would be 13.3% and 7.1% respectively. These elevated BLL subject were again

more family clustered. Serum urea was significantly higher (25% above normal) in the fisherman than in controls and correlated with age and ZPP. Anyhow, renal insufficiency could not be correlated with BLL, though correlation with ZPP might demonstrate chronic Pb exposure since ZPP could be more reliable as a marker of chronic exposure than BLL because of its longer persistence in blood cells than Pb in blood. ZPP elevations in both chronic and acute exposure settings lag behind elevations in whole-blood Pb by approximately 8-12 weeks. Correlations for serum creatinine, urate, and urine NAG were not found. Recent studies did demonstrate Pb-related deterioration in renal function. In the proximal tubular cells of the renal tubules, Pb binds to specific Pb-binding proteins. BLL increase of 10 μg/dL was associated with a 9% reduction in creatinine clearance. A longitudinal analysis found that a BLL 10-fold increase predicted a 7 μmol/L serum creatinine increase.[7] A recent Taiwan study found faster progression in diabetics with high-normal body Pb burden.[8]

Pb poisoning is a unique problem among the fishing community. Apparently, Pb weights are an obvious source but somehow these are not readily leachable, though water acidity from possible rainfall spilt into water tanks was not tested then. In the 1984 study, water samples from their 'fresh water tanks' showed presence of Pb. Putty, which may contain white Pb, was applied between wood pieces to expand and prevent rotting. Else, boat batteries were not easily accessible. There could be many other possible sources. The 1988 study circumstantially showed that one of major source was Pb weights in fishnets. Those fishermen who repaired their fishnet daily and who did not store them up properly were found to have higher BLL and ZPP level than in those otherwise. One of the ways by which they may have ingested excessive Pb amounts may be their habit of using their mouth when repairing the fish-nets as those who had this habit had a significantly higher ZPP level than those who did not. Significantly higher BLL was also observed in fishermen who lived on older boats and used iron instead of plastic spare water containers. Interestingly, fishermen who obtain their water from the main water pumping station had lower BLL than those getting it from other sources. No relationship between BLL and ZPP level and the use of paint either on the boat or in the water container was observed, noting painted water containers were infrequent.

Nutritional deficiencies were common in fishermen's children and could contribute to Pb absorption.[9,10] Their suboptimal nutritional status, demonstrated by "immediate 24 hour recall" in the 1984 study that showed low calcium 89.8%, vitamin C 54.8% and D intake 69.2% below WHO standards, could enhance their prevalence of Pb toxicity. Children absorb Pb well orally (~50%), adults poorly (~10%). Children also have more hand-to-mouth activity. In general, BLL peaks at 18-24 months of age. The team has during those boat visits made checkups, therapy to scabies and other illnesses and instituted health education programs to help, apart from those through informed nearby clinics. Fishermen and their children anywhere in Asia need improvement in their living conditions. In the mean time, there started much effort to move Aberdeen fishermen's abode on shore. Now visiting Aberdeen, these large sea-faring boats are less seen. Other areas have yet to be explored.

Lead Contamination at Processing for Herbs and Others

In 1971, Queen Elizabeth Hospital admitted three young patients with Pb poisoning within 3 weeks.[11] Two presented with abdominal pain suspected as appendicitis and BLL were 63 μg/dL and 83 μg/dL. All had herbal pills given by one herbalist, and a brother attending the same herbalist checked has BLL 43 μg/dL. Case description was short, but the symptomatic two had anaemia. The prescribed herbal pills showed high Pb content: 1% by weight (10000 ppm) (300 μg per pill), and as for 140 pills each packet, Pb given each dose was about 42000 μg/d. In 1977, another case of acute Pb poisoning was reported in a 4-month-old infant given Po ying tan to aid sleep.[12] In QMH, apart from the 3-year old boy admitted with Pb poisoning related to proprietary infant herbs in 1977, another hospital admission came in 1984: a 2 month old Cheung Chau fisherman's boy of normal birth started with occasional regurgitations since day 4 of life, and was treated by a herbal practitioner by a buccal application of a powder mixture.[13] He was reared in flats on shore but moved on board of parent's boat 2 weeks before admission. Admitted for constipated followed by encephalopathy at 2 months, he had generalised convulsions and was semiconscious. Hb was 6.8 g/dL and reticulocyte count 4.6%, but red cells showed no basophilic stippling. BLL was 190 μg/dL (9.15 μmol/L), while δ-ALA was normal. Blood zinc was normal. BLL reduced with 4 hours of infusion of dimercaprol followed by EDTA chelation but resurged after therapy and he needed oral penicillamine longer to maintain lower BLL. In this fisherman family, parents had BLL at 16.3 and 19.5 μg/dL, and the elder brother at 31.2 μg/dL. Somehow, the Pb

content analysed in the three given herbal powders showed one for buccal application extraordinarily high in Pb: 23.2% by weight (232,000 ppm), 15.6% as leachable, and >20,000 µg Pb/dose. To note, Joint FAO/WHO Committee on Food Additives at that time allowed <50 µg Pb/kg bw/wk (3000 µg/wk in adults), and permissible in food 6 ppm Pb in solid and 1 ppm in liquid.

The source of Pb in herbs is best illustrated by another story. One hundred and twenty-one Gurkha soldiers in 1968 at the British Military Hospital had Pb poisoning in one admission, 75 being symptomatic with nausea, vomiting, colic and constipation.[14] Those early years faced initial diagnostic difficulties requiring assistance by measuring the success of penicillamine chelation therapy and subsequent coproporphyrinuria and urinary Pb excretion measurements. Tests for blood Pb were unavailable. Extensive food sampling for Pb showed it the cause: food poisoning with lead chromate contaminating chili powder (Cayenne Pepper), which the Gurkhas used in liberal quantities to flavor their curried meals. The chili powder contained 10,000 ppm Pb, indicating Pb ingested >11,000 ug/g food. Circumstantial evidence showed that one batch of chili power had become contaminated during pulverisation in grinding machines in a small Hong Kong factory.

The most common and notable source of Pb in herbs had always been the grinders and pans during pulverisation. There was no legislation on Pb in herbs then. In the three cases in 1971 related to one herbalist, the main source was the grinder for pill processing by a manufacturer in a domestic flat. Pb content in the various pill components through processing were elaborated, and found worst in the rice used as starch coat: Pb content before grinding was 15 ppm and after grinding 460 ppm. The herb mixture for the pills was less contaminated, being 82 ppm Pb after grinding. Similarly, Tuen Mun Hospital reported in 2002 admission of three patients with Pb poisoning following use of the Chinese herbal pill Bao ning dan prepared by one attending traditional Chinese medicine practitioner.[15] A 23-year-old woman with non-specific musculoskeletal pain investigated for Hb 72 g/L, basophilic stippling a nd raised reticulocyte count, found BLL 60 µg/dL (3.03 µmol/L). After others learned of this news, a 35-year-old woman with 8 years' anxiety disorder and a 48-year-old woman with common cold attending the same practitioner were checked for BLL: 5.6 µmol/L and 6.7 µmol/L respectively. In these three cases, BLL did not correlate with clinical severity. To note, revised normal blood Pb level, was <25 µg/dL (1.21 µmol/L) (Table 1, reference BLL over the years).

Manufacturers' problem was seen worldwide. In 1988, a pill "chuifong tokuwan" from the Nan Ling Pharmaceutical Company of Hong Kong was investigated for illegal banned sales in the Texas Department of Health and found to contain Pb, cadmium and western drugs.[16] As recent as 2014, FDA warned and continues to warn the world of Bo Ying Compound for its associated Pb intoxication of an 18-month-old toddler in New York City.[17] Another review over journals reporting toxicity by heavy metal in herbs in Korea retrieved 10 publications over 47 years from PubMed and International Pharmaceutical Abstracts.[18] It re-emphasized strong evidence for processed herbs, including proprietary Chinese medicines (PCM) products at risk for heavy metal contamination.

The degree of Pb contamination in raw and unprocessed Chinese herbs is more controversial. Somehow, a 2011 USA federally funded research analysed heavy metals and pesticides in commonly prescribed raw herbs from a wide range of geographical areas in China.[19] 334 samples of raw herbs (representing 126 individual species) were collected and examined in duplicate for arsenic, cadmium, chromium, Pb, and mercury and 294 samples for pesticides. 100% of these samples were found to contain at least one metal and 34% of the samples contained all the metals tested. Pb, detectable if >0.04 ppm, was found in 95.8% of total raw herbs, at a median of 0.437 ppm (0.04-8.15 ppm).

Lead from Other Sources

Car battery with Pb was considered a significant source for Pb poisoning in the early years. This could not be demonstrated in this review of cases of Pb poisoning, only noting three children related to battery factory in 1966. In petrol, tetraethyl lead was found useful as anti-knock for car engines since 1921 and contributed to a major source of Pb in the 1960-70s. Environmental Protection Agency of USA starting 1972 phased out Pb in gasoline and achieved it in 1986. In Hong Kong, oil companies took voluntary

Table 1 Blood lead levels considered toxic over the years

Prior to 1971	≧60 µg/dL (2.88 µmol/L)
1972-1975	≧40 µg/dL (1.93 µmol/L)
1975-1985	≧30 µg/dL (1.45 µmol/L)
1985-1991	≧25 µg/dL (1.20 µmol/L)
1991-2010	≧10 µg/dL (0.48 µmol/L)
2010-present	≧5 µg/dL (0.24 µmol/L)

action in reducing Pb content of petrol by 20% after 1981. Pb emissions from motor vehicles fell significantly in the 1980s. Related general decrease in the Pb level in roadside dust was noted.[20,21] Pb concentration depicted over the years[22] showed that the ambient Pb concentrations was at very low levels in 1980s and got further reduced after the introduction of unleaded petrol in April 1992.[23] Study of indoor floor-dust Pb levels in nursery schools and kindergartens in various Hong Kong districts in 1997 showed it worst in North Point and Kwun Tong and in schools close to freeways, to gas stations and to factories.[24] Supply of Pb petrol was banned in April 1999. Pb level in air and soil was largely controlled.

In a 2000-2001 study by a community-based (Kwai Tsing) convenient sample of 101 children who participated in the Telehealth project, a remarkably high prevalence was suggested. BBL and occipital hair Pb (PbH) were estimated. BLL was 6.52±5.0 μg/dL (0.51-25.9 μg/dL) and 18.2% >10 μg/dl.[25] 6.2% had PbH above the harmful level of 11 μg/g. None had related symptoms. Lowered economic status and age below six were noted as risk factors. Peeling paint chips and second hand cigarette smoke from household member were significantly correlated to PbH level. Well under surveillance, there are yet no legislation to regulate Pb in paints in Hong Kong.

The Chinese University did a school survey in 2012. 2209 secondary school children and 893 preschool children were screened for Cadmium (Cd) and Pb in blood and urine (BLL not done in children <6 years old). BLL was <4.219 μg/dL (0.2038 μmol/L),[26] dependent on gender and residential district. Students from schools of lower academic grades had higher blood Cd and Pb than those from higher academic grades. Over Hong Kong districts, BLL was higher in the New Territories than in Kowloon, than in Hong Kong Island. A larger study is needed to make definite conclusions and clarify sources.

High-risk Occupations and Sources

Numerous clinical reports on Pb poisoning in children and adults were made in the early half of this century.[27] Pb sources mostly in inorganic form were painted furniture or toys, nipple shields, drinking water from Pb pipes or tanks, and fumes from burning battery casings. Childhood plumbism has long been done away: Pb pipes have been banned in Hong Kong since the 1930s. In Japan, Pb-containing face powder of nursing mothers was said to be a common source. Tetramethyl lead and poisoning described in Lancet in 1972[28] started more advocates for firmer control. Apart from these, Pb has been noted in stationeries, houses, drinking water, solder, household dust settled from air pollution, ceiling dust, in the food chain (via contaminated soil), vegetables, herbal medicines, traditional toys, ceramic glazes, pesticides (lead arsenate), mines, smelters, electronic wastes, TV's, computer monitors, batteries, bullets, sinkers, aviation, X-ray shields, crystal-ware (high levels in decanters), explosives, non-stick linings of pots, plastic colouring (wire, blinds), and pewter. The search for related occupational and pollution hazards expanded. One began recognising high-risk occupations including the manufacturing of Pb glass and crystals, ceramics, steel products, rubber products, bullets, plastics, weights, industries with soldering, gasoline, vehicle batteries and radiators, paints and newsprints, sound-proof material, cable covering, shipbuilding; the construction industry; waste incineration; the recycling industry incineration of Pb containing products, etc.

As recent as 2014, a 41-year-old woman with progressive weakness of four limbs was found in United Christian Hospital to have elevated BLL 1.83 μmol/L (38 μg/dL) during routine screening for treatable causes of neuropathy. BLL rechecked 2 weeks later was 54 μg/dL. She had no other clinical features of Pb poisoning. Analysis of the Chinese talismans revealed a high Pb concentration of 17,342 ppm, an odd cause worth noting.[29]

Lead Effects and Awareness

Reduction in BLL does not necessarily lead to improvement in cognition, as the effects could be irreversible. Pb nephropathy is a potential complication of prolonged high Pb exposure. Moderate symptoms occur in children with BLL >25 μg/dL (1.2 μmol/L) or in adults with BLL >60 μg/dL (2.92 μmol/L), including muscle pains, paraesthesia, mild fatigue, aggrensiveness, lethargy, and abdominal discomfort. Somehow most of the children with BLL <45 μg/dL and adults <60 μg/dL are asymptomatic. Higher BLL are more severe. BLL in Children >70 μg/dL and in adults >80 μg/dL would be treated as emergencies.

Chelation therapy is advised when BLL exceeds 45-69 μg/dL (2.17-3.37 μmol/L) but not without potential risk. On the other hand, garlic was tested experimentally useful for Pb toxicity,[30] and studied recently as effective as d-penicillamine in significantly reducing BLL, has less side effects and more clinical improvement.[31] Though larger

studies are required to make a general conclusion, garlic is worth using for less toxic cases.

Those early years were the times when Pb was often used in tools and accepted, there being not so many alternatives as material science allowed nowadays. It is cheap, useful, and easy to mine; therefore Pb is ubiquitous in air, food, water, soil, homes etc. From cases of Pb poisoning, sources of Pb were being established, and their importance gradually understood. Then prevalence of high BLL was studied particularly in regard to high-risk sources.

In that time, no legislation or regulation restricted Pb content in herbal pills and patent medicine. The Department of Health did have surveillance on common proprietary herbs for Pb content. In the 1984 study, the Hong Kong Medical and Health Department clarifying our quest, listing 44 proprietary herbs tested for Pb content within the 12 months: 2 from the 44 PCM were above standard, while the rest far safe. Still, to identify and raise concerns, it depends on the work of interested parties. Legislation was expectedly slow when only relying on collections of full evidence.

The Chinese Medicine Ordinance was enacted since 1999, and Chinese medicines are classified into two categories, Chinese herbal medicines (CHM) and PCM. CHM and PCM even as food products are regulated under the Ordinance. Food products containing PCM must be registered before they can be imported to and sold in Hong Kong and must meet certain safety, quality and efficacy standards, including surveillance sampling. Legal requirements for good manufacturing practice (GMP) for PCM involve inspection of many processing points.

From the changing standards, recent years are stricter for allowed BLL or Pb intake. Finally, a large twelve-year 2006 study of 13,946 adults published in Circulation suggested that no Pb level other than zero is safe i.e. zero Pb tolerance. A BLL of just 2 μg/dL had been sufficient to significantly increase adult risk of developing heart attacks (155%), having a stroke, dying from heart disease, and dying prematurely.[32] But currently, nearly 40% of Americans have BLL above 2 μg/dL.[33] Well intended, most government will see zero Pb levels not achievable and aim to control and regulate with only strict standards.

A tribute to Professor CY Yeung for his good efforts in Pb studies.

Declaration of Interest

There are no financial support for the work and other potential conflicts of interests.

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