Review Article

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LEAD POISONING: AN OVERLOOKED DIAGNOSIS IN CLINICAL PRACTICE

Amol S. Kadu[1]*, Amit R. Nampalliwar[2], Anurag G. Pandey[3], Anita Sharma[4], Vinod kumar Gothecha[5]
[1]M.D. Scholar, P.G. Dept. of Agadtantra, National Institute of Ayurveda, Jaipur, Rajasthan, India
[2]Ph.D. Scholar, P.G. Dept. of Roganidan and Vikruti Vignan, National Institute of Ayurveda, Jaipur, Rajasthan, India
[3]M.D. Scholar, P.G. Dept. of Roganidan and Vikruti Vignan, National Institute of Ayurveda, Jaipur, Rajasthan, India
[4]Assistant Professor, P.G. Dept. of Agadtantra, National Institute of Ayurveda, Jaipur, Rajasthan, India
[5]Associate professor and HOD, P.G. Dept. of Agadtantra, National Institute of Ayurveda, Jaipur, Rajasthan, India

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***Corresponding author**
Dr. Amol Sudhakar Kadu, M.D. Scholar (Final year), P.G. Dept. of Agadtantra, National Institute of Ayurveda, Jaipur. Madhav Vilas Palace, Near Joravar Singh Gate, Jaipur, India Email: dramolkaduayu@gmail.com
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ABSTRCT
Lead is a natural element that is persistent in water and soil. Human exposure occurs primarily through diet, air, drinking water and ingestion of paint chips. Absorption is increased in persons suffering from iron and calcium deficiency. Lead is a multitargated toxicant, causing effects in the gastrointestinal tract, hematopoietic system, cardiovascular system, central and peripheral nervous systems, kidneys, immune system, and reproductive system. Lead poisoning is a common environmental health hazard in developing countries. Incidences of lead poisoning are seen in all age groups, especially in children's and adults working in lead-based industries, where many workers still remain unaware of the adverse effects of exposure to unusually high levels of lead. Unfamiliarity with the symptoms of lead poisoning results in miss or delayed diagnosis, inadequate treatment, and patients' continuous exposure in the work environment. Lead intoxication symptoms such as abdominal pain, constipation, nausea, vomiting etc make lead poisoning an important diagnosis to be differentiated from many gastrointestinal and surgical diseases. For diagnosis of lead poisoning, paying attention to a good occupational history, detail knowledge about lead poisoning symptoms and its sources are essential. For this study, relevant literature was searched, retrieved and synopsized with respect to its sources. Lead poisoning symptoms and few case studies of lead poisoning which initially was misdiagnosed hence our study will be helpful to front-line practitioners and family physicians. They can make great contributions to the discovery of occupational diseases in the future.
Key words: lead, multitargated toxicant, lead poisoning, misdiagnosis, occupational history

INTRODUCTION

Lead has been used as long as recorded history for various purposes ranging from jewellery to weapons and in construction materials, paints and pigments manufacture.[1] Currently, lead is used in more than 900 industries including mining, smelting, refining, battery manufacturing, soldering, and so on.[2] It is also used in production of alloys, cable sheathing, pigments, rust inhibitors ammunition, glazes and plastic stabilizers.[3] By wide use of it, chances of its human exposure is also high. The main sources of lead exposure in United States and many other countries are lead based paint, leaded gasoline; lead-soldered plumbing fixtures, pipes, canned foods, contaminated alcoholic beverages, lead-glazed kitchens/dining utensils, mining, industrial contaminations as well as occupational exposure.[4] Lead may obtain access to the body by inhalation, ingestion or by absorption from the skin or mucous surfaces.[5] The dominant route of lead exposure in adults is inhalation.[4] Ingestion is the most common route of exposure to lead for children.[5]

Lead entering through the respiratory and digestive systems get absorbed and distributed in blood, bone and soft tissues.[6,7] Approximately 99% of blood lead content is bound to red blood cells; only 1% is present in the plasma and is available for exchange with lead contained in the other tissues. The half- life of lead in the blood is ~30 days in individuals with normal renal function and longer in individuals with renal insufficiency.[8, 9] Over 95% of lead content resides in the bone, where half-life of lead is decades long. Consequently, bone serves as the principal repository of this element in the body. Gradual release of lead from the bone serves as a persistent source of toxicity long after cessation of external exposure.[6] Lead exposure is influenced by both community characteristics and individual-level factors.[10] Lead absorption is increased in persons suffering from iron and calcium deficiency and many other factors also.[11] Most of the lead absorbed into the body is excreted either by the kidney or through biliary clearance. Adults may ultimately retain only 1% of absorbed lead, but children tend to retain more than adults.[10] It is impossible to accurately measure total body lead burden. For instance, measurement of blood lead concentration primarily reflects recent/current lead exposure. X-ray fluorescent imaging provides a semi quantitative tool for measurement of lead content in individual bones. In addition, measurement of urinary lead excretion after EDTA administration provides a rough estimation of lead burden.[6]

Lead is multitargated environmental toxicant that is capable of causing numerous acute and chronic illnesses[6]. The toxic effects of lead range from subtle biochemical abnormalities to severe clinical emergencies. Target organs include central nervous system, kidney, cardiovascular system, joints, reproductive system, haem biosynthetic pathway, and gastrointestinal tract and may contribute to morbidity and mortality through its adverse impacts on these systems.[12]

Need of lead poisoning awareness

Many misdiagnoses and malpractices can occur due to unawareness of lead poisoning as an imitator of many organ symptoms.[13] Lead affects all organs and functions of the body to varying degrees. The frequency and severity of symptoms among exposed individuals depends upon the amount of exposure.[7] Because of differences in individual susceptibility, symptoms of lead exposure and onset vary. Frequently, lead exposure appears asymptomatic, but still impairing the health of children and adults. With increasing exposure symptoms of mild toxicity appears at BLLs (Blood lead levels) from 35 to 50 µg/dL in children and 40 to 60 µg/dL in adults, in the form of myalgia, paresthesia, mild fatigue, irritability, lethargy and occasional abdominal discomfort. Symptoms of moderate toxicity are arthralgia, headache, general fatigue, tremor, vomiting, constipation, and weight loss. Severe toxicity is frequently found in association with BLLs of 70 µg/dL or more in children and 100 µg/dL or more in adults. The symptoms of severe toxicity are blue to black lead lines (Burton's lines) on gingival tissue, paresis or paralysis, colic (intermittent, severe abdominal cramps). In adults lead encephalopathy may occur at extremely high BLLs e.g., 460 µg/dL. Renal impairment is a late effect of chronic exposure and may not be detected without specific testing. In lead exposed patients, the peripheral blood smear may be either normochromic and microcytic or hypochromic and microcytic. There may be basophilic stippling in patients who have been significantly poisoned for long period; however these results are not specific to lead exposure (they should be differentiated from other causes, especially iron deficiency anemia).[14] Presence of gastrointestinal manifestations are very important in lead poisoning nowadays. At moderately elevated BLL, many of the symptoms are non-specific and may consist of epigastric discomfort, nausea, anorexia, weight loss, and dyspepsia. At higher concentrations, severe, intermittent abdominal cramping pain may ensue. Physicians often neglect the differential diagnosis of lead poisoning when encountered with patients complaining of abdominal pain, resulting in missed or delayed diagnosis, inadequate treatment, and patient's continuous exposure to lead.[12] Similarly, lead nephropathy still appears to be under recognized by clinicians as a cause and promoter of CKD, especially in people with more apparent or easily identifiable causative factors such as diabetes and hypertension. This assertion is supported by reports of patients in whom the identification and treatment of lead toxicity resulted in improved renal function. These patients were earlier thought to have CKD from more obvious risk factors.[15]

The same wrong diagnosis can be happened with other symptoms of lead poisoning of different systems like nervous system, cardiovascular, and reproductive system also. So it is necessary to study lead intoxication symptoms and probable source of its exposure to make this disease an important diagnosis to be differentiated from many gastrointestinal, neurological and surgical diseases.[13] A history of lead intoxication often requires knowledge of the various sources of lead exposure, laboratory tests, factors enhancing lead absorption and many previously known and some newly described facts which support this diagnosis.[17] In this article, sources of lead poisoning, acute and chronic symptoms of lead intoxication, and some misdiagnosed case studies of lead poisoning will be discussed to highlight an overlooked diagnosis of lead poisoning in medical practice.

Sources of lead and its use

The principle salts of lead which produce toxic effects are- Lead Acetate (sugar of lead or salt of Saturn) was used to sweeten wine in olden days. Lead Carbonate ($PbCO_3$) - is extensively used as a pigment in oil painting. It is also used as an ointment. Children who suck and bite painted toys with white lead suffer from poisoning. Lead Chromate (chrome yellow) -Fatal cases of poisoning have occurred from the use of sweetmeats coloured with this salt. There are 3 cases of chronic poisoning are reported from the use of tobacco snuff adulterated with lead chromate as a colouring agent. Lead Chloride ($PbCl_2$) used as pigment. Lead Sulphide (Galena) (PbS) - is naturally found in the form of cubic crystals, but is sold in the bazaar in powder form as surma in place of sulphide of antimony which is used as a collyrium for the eyes. Lead Monoxide (Litharge*, Mudrasang*) (PbO) - Quacks use monoxide as a remedy for syphilis. It is also commonly used by painters and glaziers, and is a constituent of certain hair dyes.[17] Tetra-Ethyl lead , Tetra-methyl lead- are added to petrol to prevent 'knocking' and the mixture, known as ethyl petrol or ethyl gasoline, is used as fuel for motor cars.[21, 22]

Occupational exposure to lead

Workers are also exposed to lead in many occupations such as motor vehicle assembly, panel beating, battery manufacture and recovery, soldering, lead mining and smelting, lead alloy production, and in the glass, plastics and printing industries. Other occupations include ceramic and paint workers, automobile radiator repairers, petrol attendants and petroleum refining workers as well as welding, pottery and ceramic ware production and the production of jewellery.[1, 2, 23]

Various Routs of Lead Exposure

Through Ingestion - Drinking water is an important source of lead exposure. Previous almost universal use of lead compounds in plumbing fittings and as solder in water-distribution systems resulted in significant lead exposure from drinking water.[1, 2, 23]

Food - which can be contaminated by lead in water, air or food containers[11] (Tinned food contaminated lead from the soldered cans or leaded ceramics)[24] Use of ghee stored in brass or copper vessels lined inside with tin in brass or copper vessels lined inside with the tin in which oleate of lead is formed and also by taking food cooked in tinned vessels.[25]

Traditional medicines - Lead in products such as herbal and traditional medicine increases the chances of lead poisoning.

Through Inhalation - The dominant route of exposure through inhalation in adults is associated with smelting and burning processes, as well as inhalation of lead-containing dust from scraping, burning, or sanding lead paint from surfaces, as well as exhaust from vehicles and planes powered by lead-containing gasoline.[6] Inhalation of lead dust and fumes by makers of white lead and makers and users of lead paints, smelters, plumbers,

glass-polishers, printers, enamel workers, glass blowers, etc.[5] Interior house dust can become contaminated with lead as a result of the deterioration or disturbance of lead paint on the walls, doors, and windows of a home. Used car batteries and open burning of waste can also contribute to lead poisoning.

Through skin - Lead absorption is possible through raw or intact skin. Absorption of vermillion applied to scalp.[24] In addition, tetraethyl lead, a previously common gasoline additive is absorbed via the cutaneous route.[6] From the use of lead containing cosmetics like surma.

The skeleton – It is an important endogenous source of labile lead as the bones and teeth contain more than 95% or the total lead in the body.[12]The rate of release of lead from the skeleton is increased in conditions associated with heightened bone resorption/turnover, such as a pregnancy, lactation, menopause, osteoporosis, immobilization, and hyperthyroidism.[6]

Factors enhance the lead absorption

Nutritional status is being increasingly shown to influence the extent of lead absorption. High intake of fat, inadequate intake of calories has also been associated with enhanced lead absorption. Lead absorption is increased when the stomach is empty; small frequent meals reduce absorption.[18] The experimental evidence obtained with laboratory animals shows that the toxicity of lead can be increased by deficiency of certain essential nutrients such as calcium, iron, zinc and selenium. It was suggested multiple marginal nutritional deficiencies may be of importance in determining the response of humans to the toxic effects of various heavy metal pollutants.[19] Irregular food intake, high dietary fat intake, low dietary calcium and iron deficiency can increase the risk of lead toxicity in a contaminated community.[20] Ascorbic acid intake is one of several nutritional factors that may influence lead toxicity through an influence on absorption, elimination, transport, tissue binding, or secondary mechanism of toxicity.[21] The association of both high blood lead levels and low dietary ascorbic acid intake (in adults) with poverty raises the possibility of occurrence of lead poisoning manifestation.[20,22]

Lead toxicities and symptoms

Lead poisoning may be acute or chronic.

Acute poisoning

It occurs mostly from lead acetate.[11]

Symptoms - Such as, sweet and metallic astringent taste, a sensation of burning and dryness in the throat, salivation and intense thirst immediately after swallowing the poison occurs. Vomit occurs within half-an-hour, the vomited matter being white or tinged with blood. Colicky pain comes in paroxysm, but is relieved by pressure. The abdominal walls are tender and contracted. Constipation is a constant feature, though purging has occurred in some exceptional cases, when stools are offensive and dark or black from the formation of lead sulphide. The urine is scanty. The tongue is coated and the breath is very foul and offensive. Great prostration occurs with cold and clammy skin and a quick, feeble pulse. The nervous symptoms which include drowsiness, insomnia, headache, vertigo, muscular cramps, convulsions, numbness and occasionally paralysis and oliguria. Wasting follows and death occurs generally from exhaustation.[17]

In acute poisoning by tetra-ethyl lead, gastric symptoms are absent or slight. The central nervous system is affected. cerebral symptoms, the so called lead encephalopathy, predominate.[5] It is characterized by - Irritability, nervousness, insomnia, frightening dreams, headache, vertigo, mental excitement, tremors, muscular weakness, bradycardia, low blood pressure, hypothermia, delirium, and convulsions. Earlier there may be nausea, vomiting, loss of appetite and weight.[17]

Sub acute form

The administration of repeated small doses of a soluble salt such as lead acetate results in the sub acute form of poisoning. A blue line is observed on the gums and gastro-intestinal symptoms are usually present. The face is livid and sunken and the look is anxious. The secretions are mostly arrested. The urine is scanty and deep-red. The nervous symptoms are more prominent, such as numbness, vertigo, dragging pain in the loins, cramps and flaccid paralysis of lower limbs. Death though rare, may occur from convulsions and comma within three days. After apparent recovery the symptoms sometimes return probably in an aggravated form and the illness lasts for a long time.[17]

Chronic Lead Poisoning

Lead poisoning is nearly always of the chronic type.[11] Lead poisoning is also called 'plumbism'. This is because of the ill-effect of lead and lead-containing materials on various organs. Lead especially affects the nervous system and kidneys. In children, lead decreases IQ and in adults, causes kidney- muscle- nerve related disorders.

Hematological Toxic Effect

Facial pallor - particularly about the mouth is one of the earliest and most consistent sign which is due to vasospasm.[5]

Anaemia - Anaemia is the classic clinical manifestation of lead toxicity in erythrocytes. The severity and prevalence of lead induced anaemia correlate directly with the blood lead concentration. Younger and iron deficient children are at high risk of lead induced clinical anaemia.[26] The red blood cells show marked **punctuate basophilia** (presence of many dark-blue coloured pinhead likes spot in the cytoplasm of red blood cells) with a moderate degree of hypochromic anaemia associated with polychromasia, reticulocytosis, poikilocytosis, and an increase in mononuclear cells and decrease in polymorphonuclear cells.[5,17] The anaemia is probably due to the decreased survival time of red blood cells and inhibition of haem synthesis by its interference with incorporation of iron into protoporphyrin. The condition is due to toxic action of lead on porphyrin metabolism. Lead suppresses enzymes involved in the process of the synthesis of the haem.[17]

Gastrointestinal Toxic Effects

Colic and Constipation - Colic generally affects intestines, ureters, uterus and blood vessels.[5] Severe colicky pain is felt in the abdomen. It is relieved by pressure, and is associated with obstinate constipation known as dry belly ache, anorexia and metallic taste. It is a late manifestation but constitutes an acute symptom of chronic plumbism. There may be profuse sweating and vomiting before an attack of colic, which is often sudden and at night.[17]

Lead line on gingival tissue - This is stippled bluish-black line at the junctions of the gums and teeth especially on the upper jaw, is clearly noticeable when the teeth are dirty and infected due to subepithelial deposition of lead sulphide granules liberated by microorganism from decomposing protein food around carious teeth in the presence of circulating lead.[5] The blue line signifies lead absorption, but is likely to disappear, if the mouth is kept properly cleaned and the bad teeth removed. The patient has a sweetish metallic taste in the mouth, foetid breath, dyspepsia and sallow earthy complexion.[17]

Neurological toxic effects

This is usually a late manifestation, seen in less than 10% of cases. Onset may be gradual or sudden.[17] It is commoner in adults than in children, and men are particularly affected. There may be tremors, numbness, hyperesthesia and cramps before the actual muscle weakness.[4] Wrist / Foot drop - Typical paralysis affecting the extensor muscles of the fingers and the wrist except the supinator longus causes 'wrist drop' and 'claw shaped hand' paralysis may spread to the extensors of the foot, resulting in dropped foot.[11]

Peripheral neuropathy - The wasting is usually seen in those muscles, which according to the type of work done are most liable to strain and fatigue and is result of the purely motor type of peripheral neuropathy due to axonal degeneration and segmental demyalination. The exraoccular muscles may be affected. Pain in the large joints and tremors which are increased by movement are observed before the paralysis sets in. There is no sensory involvement.[5, 17]

Central Nervous system - Various researches indicate that the developing central nervous system (CNS) is the most sensitive target for lead, with children being much more sensitive to neurological and behavioral effects than adults. The most serious CNS effects at low Pb levels in children include impaired concentration, hyperactivity, poor classroom behaviors, and decreased IQ Scores. An IQ reduction of between 1-3 points has been measured in studies as Pbs increases from 10 µg/dL to 20 µg/dL.[26]

Encephalopathy - lead encephalopathy, in some form is said to be present in almost every case of plumbism. Sometimes seen in adults associated with chronic poisoning but usually it is common in children showing stupor, convulsions, and coma in acute poisoning. This involves cerebral psychical affections such as intense headache, loss of concentration, and memory, insomnia, optic neuritis, amaurosis, epileptiform convulsions, hallucinations, delirium, insanity, and comma.[5]

Renal Toxicity

Chronic lead nephropathy which results from long-term lead exposure is a slowly progressive interstitial nephritis which is frequently associated with hypertension and hyperuricemia. Chronic lead nephropathy is usually identified after blood lead levels have exceeded 40-60 µg/dL; however, it has been shown that at blood lead levels less than 25 µg/dL the inhibition of the metabolic activation of vitamin D takes place in the kidneys, as a result of lead accumulation. With continuous lead exposure and blood lead levels of 40-80 µg/dL, acid fast intranuclear inclusion bodies consisting of lead-protein complexes are formed and deposited in the tubules. These changes result in defective tubular function manifesting as hyperuricemia which is a common feature of lead nephropathy. As a result, the combination of hyperuricemia and renal failure in the absence of renal intratubular uric acid deposition or stone formation should increase the suspicion of lead nephropathy.[15] Blood concentrations greater than 40 µg/dL are associated with an increased risk of nephropathy and related renal failure. Lower levels of exposure to lead can act as a cofactor that increases the risk of renal dysfunction and rate functional decline. People with diabetes and hypertension are at increased risk of clinical renal dysfunction.[26]

Cardiovascular toxicity

Among occupationally exposed workers, long term, high dose exposure to lead has an increased risk of hypertension and stroke as lead causes vascular constriction.[5]

Reproductive system

Menstrual derangements, such as amenorrhoea, dysmenorrhoea, menorrhagia, sterility of both sexes and miscarriages are frequent. Abortion occurs in pregnant women between 3-6 months.[17]

Developmental defects

Fetal lead exposure can increase the risk of reduced birth weight and premature birth, prenatal exposure to deficits and delays in intelligence, motor skills and behavior (neurobehavioral).[25]

Carcinogenic Effects

Several studies involving rats and mice associated tumour formation, most often in the kidney, with ingestion of soluble lead salts.the results have been criticized because of the very high doses given the animals. however, bases upon sufficient evidence in animal studies, the environmental protection agency has classified lead as a Group B_2, probable human carcinogen.[25]

Diagnosis

However the most sensitive and specific test in the evaluation of lead toxicity is the whole blood lead level.[4] Measurement of urinary lead might be useful as a proxy for plasma lead levels in studies of lead toxicity.[11] Because of differences in individual susceptibility, symptoms of lead exposure and the onset vary.

Overlooked diagnosis of lead poisoning

Abdominal pain is nearly always a diagnostic challenge in clinical practice. Among its many causes, lead poisoning has been known for a long time and is still taking place now-a-days.[26]

Here some cases of lead poisoning are illustrated in detail for understanding that how can patients not get diagnosed accurately.

Case I

A 41 year old married male who is heavy smoker (about 30 pack-years) has been working as an operator of a machine and used to cut and finish lead plates for 14 years in battery manufacturing plant. He had severe abdominal colic since 4 months. He underwent an appendectomy operation as diagnosed c/o appendicitis (pathology revealed normal tissue of appendix) without any improvement in symptoms. He has developed other symptoms including headache, lethargy, fatigue, irritability, insomnia, muscle pain (especially in legs), constipation, decreased libido, nausea, vomiting, tremor,

loss of appetite, and weight loss. After discharge from hospital without any improvement, he was referred to occupational medicine clinic with suspicion of lead intoxication by an occupational medicine specialist. At this time he had the aforementioned abdominal pain. On Physical examination Tem- 37.2 C, R/Rate-14/min, BP-145/90 mmHg. His conjunctivae were pale, had mild tenderness in deep abdominal palpation. Blood test revealed high blood lead level. Not having symptoms related to neuropathy. After chelation therapy of lead poisoning, he recovered completely.

Here, an adult battery worker with abdominal colic who initially underwent appendectomy with removal of normal appendix, later on he was diagnosed with lead poisoning and was treated appropriately with lead chelator ($CaNa_2EDTA$).[12]

Case II

Another one case of an adult battery worker, c/o pain in joints and epigastrium. Who initially received symptomatic treatment because of clinical misdiagnosis. Later, he was treated with appropriate chelators, which helped to decrease blood lead levels drastically. However, being unable to change his occupation, he continues to be exposed to potentially lethal doses of lead.[13]

Case III

35 years old serving officer presented with c/o upper abdominal colicky pain of 4 month duration which had aggravated 3 days prior to admission having no other complaints. He was K/C of cholelithiasis for which he was under surgical follow up and taking Ayurvedic medication for psoriasis since past 6 months with excellent response. On examination, the vital parameters were normal. There were multiple healed psoriatic plaques over abdomen and lower limbs. (USG) abdomen revealed chronic calculus cholecystits. Hence he was planned for elective cholecystectomy. On routine preoperative evaluation revealed HB-9.7g/dl, TLC-6000/cu.mm, Platelets -1.8 lakh/cu.mm., bilirubin-2.3g/dl (predominantly indirect), AST-146 IU/L, ALT- 180/IU/L, alkaline phosphatase-195 IU/L and normal routine urine examination. Peripheral blood smear (PbS) revealed normocytic, normochromic anaemia with increased polychromasia. The reticulocyte count was 3%. In view of the anaemia, increased polychromasia, raised bilirubin and transminases, the patient was further evaluated. Detailed liver function test, upper gastro intestinal (UGI) endoscopy, hepatitis viral markers, isotope study for occult gastrointestinal (GI) hemorrhage and colonoscopy did not reveal any abnormality. The cause of anaemia and bilirubinemia remained obscure. Hb electrophoresis, Coomb's test, osmotic fragility, iron studies, serum B_{12} and folate levels did not reveal any abnormality and PbS again showed normocytic normochromic picture with increased polychromasia. Repeat haemogram showed a gradual fall in Hb levels to 8.2 and 7.6 g/dl with rising reticulocyte count of 6% and 8% respectively. Repeat PBS done for the third time showed normocytic normochromic anaemia with basophilic stippling of the red blood corpuscles (RBCs). In this case, a decline in HB% with increasing reticulocytosis and indirect hyperbilirubinemia suggested haemolysis. Basophilic stippling in RBCs raised doubt of lead poisoning, more so the patient was on Ayurvedic medication for psoriasis. So that serum lead level was sent for estimation which reveals a value of 115 µg/dL (normal < 14 µg/dL). Neurological and renal function evaluation did not reveal any abnormality qualitative chemical analysis of Ayurvedic powder was done which reveal very high lead levels.[24]

In this case, patient presented with pain in abdomen and was planned to be taken up for cholecystectomy. Although cholelithiasis cannot totally absolved as the cause of pain abdomen in this case, the very fact that the pain subsided on chelation therapy and has not recurred despite patient not having undergone any surgical intervention, led us to believe that it is most likely related to lead toxicity which is initially overlooked as the cause of pain in abdomen.

DISCUSSION

All these cases revealed that, most of the time, patients come in the OPDs of civil hospitals with complaints of recurrent abdominal pain, bloating, nausea, vomiting, anorexia, constipation and body weight loss. The symptoms were non-specific and medical history did not help narrate the diagnostic possibilities. Occupational history constitutes the first key element in the discovery of occupational diseases, but due to short of time, lack of knowledge, or not given the importance to occupational history, it is frequently omitted. The family physician should observe carefully and asked relevant questions without omission, thus obtaining the first clue to a possible relationship.[12] Many times, the manifestation resembled gastrointestinal obstruction involving both upper and lower segments and appendicitis so that unnecessary surgical operation could be performed. Therefore, paying attention to a good occupational history will prevent many unnecessary and/or avoidable medical interventions.[13]

Sometimes, history of other medication especially Ayurvedic and Siddha medicine taken by the patients reveals the history of lead poisoning, as happened in case no. III. There are various factors which enhances the lead absorption as mentioned earlier, so that it is not only necessary to know the source of lead exposure but also to know the various factors which increases lead absorption for proper diagnosis as well as for treatment purpose. Sometime, lead missiles remaining embedded in the tissues owing to gunshot injuries may produce poisonous symptoms within weeks or even after years so detailed history of patient is mandatory for proper diagnosis of lead poisoning.

Cases of chronic lead poisoning may be referred to a medical practioner under the workmen's compensation Act 1923, for the workmen who contract the disease in the course of and by reason of their employment are entitled to compensation from their employer during such time as they are incapacitated from earning their living, or if death occurs from the disease, the dependants of the deceased are entitled to compensation.[17] It is important that, beyond diagnosis of the case of lead poisoning, it is necessary to prevent exposure to lead. For that reason detailed knowledge about sources of lead poisoning should be known to physicians. The significant point is

that lead intoxication is preventable and its treatment is straightforward. Therefore, paying attention to good occupational history will prevent unnecessary and or avoidable interventions in clinical practice.[9]

CONCLUSION

In conclusion, these cases illustrates that patients having clinical manifestation of GI tract symptoms like chronic recurrent abdominal pain, constipation, nausea or neurological symptoms found in children, symptoms related to renal- cardiovascular or reproductive system. Through proper history of such patients (working in a lead-related industry or taking medication of other systems or in case of children who unknowingly exposed to lead), it is not difficult to diagnose lead poisoning in time. Detailed knowledge of symptoms of lead poisoning, like pseudo-obstructive phenomenon, mild anemia plus basophilic stippling, high BLL, and a laboratory results feedback mechanism will helpful for proper diagnosis. Hence key element of all the facts is proper history (occupational, medication), without which every step will be impossible. Front-line practitioners, family physicians can prove themselves invaluable contributing to the discovery of occupational diseases. The most important factor in the management of such cases is to prevent exposure to lead. For these reasons, physician should know detailed knowledge about lead and its sources, various modes of its human exposure, lead poisoning symptoms, diagnostic tests for proper and early diagnosis of lead poisoning to avoid further complications and other medical intervention. Education, communication, documentation, media and out-reach programmes must be extensively used in this effort.

REFERENCES

1. Jayanti mani, Nalin chaudhary, Makarand Kanjalkar, Pravina U shah. Letters to the editors: Cerebellar ataxia due to lead encephalopathy in adults. J. Neurol. Neurosurg. Psychiatry 1998;65:797-806
2. S Mohammadi, AH Mehrparvar and M Aghilinejad. Appendectomy due to lead poisoning: A case report. Journal of occupational Medicine and Toxicology 2008;3:23 http://dx.doi.org/10.1186/1745-6673-3-23 PMid:18928540 PMCid:2575197
3. Lysaght MJ. Maintenance dialysis population dynamics: Current trends and long term implications. J Am Soc Nephrol 2002;13: 37 - 40
4. Muntner P, Menke A, DeSalvo KB, Rabito FA, Batuman V. Continued decline in blood lead levels among adults in the United States: The National Health and Nutrition Examination Survey. Arch Intern Med 2005;165:2155-2161, http://dx.doi.org/10.1001/archinte.165.18.2155 PMid:16217007
5. Dr CK Parikh. Parikh's textbook of medical jurisprudence, forensic medicine and toxicology. 6th edition Reprint 2006; sec-ix,9. Pp 19-9.22
6. Nosratola D Vaziri. Mechanism of lead- induced hypertension and cardiovascular disease. Am J Physiol. Heart Circ Physiol. 2008;295:H454-H465 http://dx.doi.org/10.1152/ajpheart.00158.2008 PMid:18567711 PMCid:2519216
7. Lead Exposure in Adults - A Guide for Health Care Providers www.health.ny.gov/publications/2584.pdf] [cited 2012/4/30]
8. Hryhorezuk DO, Rabinowitz MB, Hassl SM, Hoffman D, Hogan MM, Malin K, Finch H, Orris P, Berman E. Elimination kinetics of blood lead in workers with chronic lead intoxication. Am J Ind. Med 8:33-42 http://dx.doi.org/10.1002/ajim.4700080105 PMid:3927718
9. Rabinowitz MB. Toxicokinetics of bone lead. Environ Health Perspect 1991; 91:33. http://dx.doi.org/10.1289/ehp.919133 PMid:2040248 PMCid:1519353
10. TD Matte. Reducing blood lead levels: Benefits and strategies. JAMA, 1991;281(24): 2340-2342 http://dx.doi.org/10.1001/jama.281.24.2340
11. Ragaa M. Abdel-Maaboud, Madiha M.El-Attar, Nadia A. Mohamad, Sohair M. Ahmed, Ahmed Medhat. Lead toxicity in some rural communities in assiut governorate. Ass. Univ. Bull. Environ. Res. October 2005;8(2):325-332
12. Nai-Fan Lin, Pai-Tsang Huang. Lead Poisoning: An Often Overlooked Cause of Abdominal Pain; Taiwan J Fam. Med 2005; 15: 46-53
13. S Mohammadi, AH Mehrparvar and M Aghilinejad. Appendectomy due to lead poisoning:A case report. Journal of occupational Medicine and Toxicology 2008;3:23 http://dx.doi.org/10.1186/1745-6673-3-23 PMid:18928540 PMCid:2575197
14. ATSDR, Agency for toxic substances and diseases Registry: "case studies in environmental medicine:lead toxicity.http://www.atsdr.dc.gov/HEC/CSEM/lead/casestudy_pretest.html. 19/01/03, 2003
15. Datonye Dennis Alasia. Lead nephropathy: revisiting an overlooked cause of kidney disease. Nephrology Reviews.2012; 4(1):38-41
16. Brewster UC, perazell MA. A review of chronic lead intoxication: an unrecognized cause of chronic kidney disease. Am jmed sci. 2004; 327 (6): 341-7 http://dx.doi.org/10.1097/00000441-200406000-00008 PMid:15201648
17. K Mathiharan, Amrit K Patnaik. Modi's Medical Jurisprudence and Toxocology. 23rd edition Published by Lexis Nexis Butterworths Wadhwa. Nagpur; 2005: pp 158-172
18. W.Michael, M Shannon. "Lead" cited in Clinical management of poisoning and drug overdose, 3rd edition, chapter 57, pp 770, 1998.
19. OA Levander. Metabolic interaction between metals and metalloids. Environ Health Prospect.1978Aug; 25:77-80 http://dx.doi.org/10.1289/ehp.782577 PMid:720305 PMCid:1637173
20. KR Mahaffey. Nutrition and lead: Strategies for public health. Environ Health Prospect. 1995;103 (suppl. 6): 191-196 http://dx.doi.org/10.2307/3432373 PMid:8549473 PMCid:1518938
21. MA Peraza, F Ayala-Fierro, DS Barber, E Casarez, LT Rael. Effects of micronutrients on metal toxicity. Environ Health Prospect. 1978;103 (suppl. 1): 203-216
22. G Block and A Sorenson. Vitamin C intake and dietary sources by demographic characteristics. Nur Cancer, 1978;10: 53-65 http://dx.doi.org/10.1080/01635588709513940 PMid:3615217
23. Meyer P, Pivert T, Dignam T, Homa D, Schoonover J, Brody D. Surveillance for elevated blood levels among children. United States 1997 -2001. MMWR 2003; 52: ss -10
24. Wg Cdr AVS Anil Kumar, Maj Sunitha BS, Gp Capt RC Panda, vsm Gp Capt AK Pujhari, Col PG Kumar, Wg Cdr S Sampath. Case report: An unusual case of pain Abdomen. MJAFI 2010; 66: 172-174
25. ARD-EHP-10. Lead: Health information summary. des.nh.gov/organization/commissioner/pip/ard-ehp-10pdf. [cited 2012/5/1]
26. World health organization. Childhood lead poisoning. 2010; www.who.int/ceh/publications/leadguidance.pdf][cited 2012/4/2]

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