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Homeopathic *Plumbum metallicum* for lead poisoning: a randomized clinical trial☆

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Introduction: **Poisoning due to lead and its compounds has short and long-term effects primarily on the nervous, hematopoietic, gastrointestinal, cardiovascular, musculoskeletal, renal and reproductive systems. It can manifest in acute or chronic symptoms. Measuring serum concentration is the primary method for diagnosing and monitoring exposed workers. Presently, elevated lead levels are treated by drugs whose effectiveness is contested on various fronts. Experimental studies suggest that homeopathic preparations may be in controlling blood lead levels in laboratory animals, creating the need for controlled studies to evaluate the effectiveness and safety of these preparations in humans.**
Objective: **To evaluate the effectiveness of the homeopathic preparation *Plumbum metallicum* in reducing the blood lead level of workers exposed to this metal.**
Design: **Double-blind randomized trial.**
Setting: **Workers' clinic in the Ajax battery plant, which employs 900 workers with varying degrees of lead exposure in Bauru, São Paulo State, Brazil.**
Subjects: **131 workers exposed to lead.**
Intervention: ***Plumbum metallicum* 15cH or *placebo*, orally for 35 days.**
Results: **The percentage of workers who demonstrated a reduction in lead counts by a percentage greater than or equal to 25% following treatment was the same for both groups: 20.3% in the homeopathic groups *versus* 21% in the control group [Relative Risk (RR) = 0.95, confidential interval (CI) 95%: 0.47−1.92)]. Analysis by intention-to-treat also did not show any difference between the groups: 18.2% in the treated group *versus* 20% in the *placebo* group (RR = 0.91, CI 95%: 0.45−1.84).**
Conclusion: **The homeopathic preparation *Plumbum metallicum* had no effect, in this study, in terms of reducing serum lead in workers exposed to lead.** *Homeopathy* (2011) **100**, 116−121.

Keywords: Lead poisoning; Controlled clinical trial; Effectiveness; *Plumbum Metallicum*; Homeopathic drug

Introduction

Lead poisoning (saturnism) from workplace or environmental exposure is common in many developing countries, in part because lead is abundant on the planet, but mostly because its physical and chemical products and compounds are useful in manufacturing a variety of products. The expansion of industrial activities in the 19th and 20th centuries, primarily the rise and growth of the automobile industry, unleashed an increase in the use of lead and the distribution of waste in the environment (approximately 300 million tons in the last 500 years) and, consequently, the exposure of individuals to various degrees.[1]

In developed countries, numerous public health campaigns have reduced the number of acute cases.

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Nevertheless, chronic lead toxicity still raises important questions concerning social and workplace healthcare. Lead-exposed workers are found in a number of industries: automobile batteries; leaded paint; ceramic glazes; munitions, PVC manufacturing; electric cables; electric- and electronic-component welding; radiator repair; non-ferrous metal smelting for metals such as bronze, tin and other alloys, in addition to secondary smelting of lead and antimony; shooting instruction; renovations and painting at construction sites, among others.

Non-occupational exposure can occur in persons exposed to environments were batteries are reconditioned, when this activity occurs at home, or with persons who reside in areas near establishments that work with lead and its derivatives (air and soil contamination).[2] There is also the possibility of contamination from a distance due to environmental contamination from compromised underground water tables and the consequent contamination of foods.[2,3] Various cases with clinical signs and symptoms of lead poisoning from an endogenous source (firearm bullets lodged in joints or contact with cerebrospinal fluid) have been reported.[2,3]

Recently, with the development in assay methods, it has been found that lead at concentrations previously considered safe may have adverse effects.[4] The family of a worker exposed to lead runs an increased risk of lead poisoning when the lead is carried home in/on the worker's body, clothing, shoes and motor vehicle. Available evidence suggests that there is no level of serum lead concentration (Pb-S) that is free from health risks for workers and their families.[5]

The exposure routes for inorganic lead are inhalation and ingestion. Adults absorb approximately 10% of in ingested lead through the gastrointestinal tract, while children absorb 50% in the same manner. Once absorbed, lead is found in all tissues, but 90% of the load is deposited in bone, where it accumulates over the carrier's lifetime and can act as an endogenous source of the metal.[6,7] Lead's half-life in the blood and in soft tissues is approximately 28−36 days, and the greatest part of the ingested lead is absorbed and excreted by the kidneys.[8,9] The transfer of lead to the fetus occurs easily during the gestational period.[10]

Lead poisoning, both acute and chronic, frequently requires prolonged medical care that involves specific treatment and removal from the source.[11] There is a large variation in individual susceptibility to lead poisoning, but symptoms generally appear in adults with a Pb-S concentration greater than approximately 40 μg/dL.[1] The number and severity of symptoms worsens with the increase of Pb-S concentrations.[1]

Initial symptoms are frequently subclinical and non-specific, such as general malaise, a decreased appetite, weariness, fatigue, irritability, sleep disturbance, headaches, concentration difficulties, memory disturbances, decreased libido, abdominal cramps, anorexia, nausea, constipation, diarrhea, epigastralgia, arthralgia, generalized myalgia and/or localized myalgia (in the calves).[4,5] Small increases in blood pressure are observed with Pb-S concentrations between 14 and 30 μg/dL.[12,13] Peripheral neuropathy can occur with chronic exposure and is characterized by weakness of extensor muscles, particularly in the dominant arm or leg, with a discreet loss of sensory perception and a decrease in nervous conduction observed with Pb-S concentrations in the range of 30 μg/dL.[14] A decrease in hemoglobin was found in exposed workers with Pb-S concentrations greater than 50 μg/dL.[4,15] Abnormal sperm morphology and decreased sperm counts were observed in cases with Pb-S concentrations of approximately 40 μg/dL.[16,17] Lead easily crosses the placental barrier and is present in breast milk, with impaired fetal cognitive development reported in women exposed to lead.[10,18]

To evaluate lead exposure, markers related to metal absorption and/or its absorption in tissues are used.[19] Blood is the medium in which lead is mostly frequently measured as an exposure marker. Lead poisoning shows relatively recent exposure since lead's half-life is short (28−36 days).[4,8] In Brazil, the Pb-S considered safe for workers is up to 40 μg/dL.[20] In the United States of America, in 1978, the Center for Disease Control (CDC) adopted the limit of 25 μg/dL and, in 1991, 10 μg/dL.[8] The criteria for diagnosis of inorganic lead poisoning are: (a) clinical symptoms (b) laboratory results confirming excessive exposure, like increased lead levels and/or the presence of biochemical alterations (an increase in porphyrin levels verified by determination of delta-aminolevulinic acid in the blood and urine, urinary porphobilinogen, coproporphyrin in urine, and erythrocyte protoporphyrin).[6,21]

Treatment

The first step in managing lead poisoning is to remove from exposure but, unfortunately, there are no clinical studies that point to the best evidence to guide which therapy route should be pursued.[22] Current treatment includes the use of chelating agents which link to metallic elements, forming stable complex molecules that can be excreted in urine.[11] The chelating agents used to combat lead poisoning are calcium disodium ethylenediaminetetraacetic acid ($CaNa_2EDTA$) or dimercaptopropanol (BAL or dimercaprol), dimercaptosuccinic acid (DMSA) and D-penicillamine.

The two main difficulties in treating lead poisoning are: (a) available evidence concerning the efficacy of chelating agents is based on case reports or case series[11]; (b) the diagnosis of lead poisoning is not precise in cases with moderately high Pb-S levels and when symptoms and signs are non-specific.

Treatment with $CaNa_2EDTA$ or DMSA is implemented in cycles of 5 consecutive days, with intervals of 10−15 days between cycles, that continue until the levels of urinary lead excretion fall within the reference values immediately after treatment.[6,11] $CaNa_2EDTA$ forms a stable complex molecule with lead and is excreted by the kidneys, unmetabolized; nevertheless, it is nephrotoxic and depletes the body's supply of calcium and zinc.[23] When used in isolation, it can lead to a worsening of acute encephalopathy and, in such cases $CaNa_2EDTA$ should be administered in conjunction with dimercaprol or DMSA alone should be administered.[8,9] Dimercaprol (BAL) forms a stable complex molecule with lead that is eliminated in the feces and urine, and it has various reported negative side effects,