INDUSTRIAL LEAD POISONING A CASE REPORT

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SYNOPSIS

A factory using lead-based stabilizers to compound polyvinyl chloride granules was found to have excessive lead in its environment. Of the thirteen workers, nine were found to have excessive lead absorption. One of them had persistent anaemia despite being removed from the working environment and was treated with chelation agents. This paper is a reminder that plumbism can occur in industries not directly involved in the manufacture or processing of lead products.

INTRODUCTION

Lead poisoning is still a frequent industrial hazard. It usually results from inhalation of fumes as from solder, spray painting or processes that require the re-smelting of metallic lead. In Singapore lead is widely used in various industrial processes example printing, painting of ships, soldering of electronic products, coating of wire, smelting, manufacturing of dry battery and also plastic granules. However to date only five cases of industrial lead poisoning have been confirmed. We described a case of industrial lead poisoning that occurred in a worker in a plastics manufacturing factory to point out that lead poisoning can occur in industries not directly involved in the manufacture or processing of lead products.

CASE REPORT

SLS, a 34 year old Chinese male has been working in a plastic manufacturing factory for the past ten years. Together with twelve other workers, his work involved contact with a lead-based stabilizer that was used for compounding polyvinyl chloride granules. During their work, masks and gloves were not worn. Using static and personal sampling of the mixers, the lead-in-air concentration was found to be in the range of 0.2 mg/m^3 to 1.83 mg/m^3 far exceeding the threshold limit of 0.15 mg/m^3 and even the limit for short-term exposure of 0.45 mg/m^3.[1].

The thirteen workers were examined and were asymptomatic except our patient who had bouts of abdominal colic and was pale. All thirteen workers had their blood lead levels estimated (Table 1). Our patient's blood level level was 100 ug/100 ml — the highest amongst the workers.

The department intervened and measures were introduced to reduce the lead content in the air. The patient was then transferred to another section that was not involved in lead handling but despite this his anaemia did not improve and he was referred for further management.

He was found to be pale, no clubbing noted, BP was 140/70 mm Hg, pulse rate 76/min. Burtonian lines were not seen. There was no visceromegaly in the abdomen. Neurological examination was unremarkable. Electromyographic studies failed to show any neuropathy.

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TABLE I

Mean Blood Lead Leve of Workers With Various Duration of Exposure

Duration of Exposure	Number of Workers	Mean blood lead level (µg/100 ml)
1 year	7	65.6
1 to 5 years	4	87
5 to 10 years	5	—
10 years	2	90

The haemoglobin was 9.2 gm% and red cells were nomocytic and hypochromic but no punctate basophilia noted. Reticulocyte, leucocyte and platelet counts were normal. Bone marrow showed impaired haemoglobinisation. Haemoglobin electrophoresis was normal. Serum iron and total iron binding capacity were normal. Serum lead was 71 ug/100 ml. The urinary lead per 24 hours was 440 ug/litre. Urine corproporphyrin was present. Calcium disodium versenate 1000 mg (15 mg/kg) was given intravenously over 24 hours and the 24-hour urinary lead excretion rose to 2200 ug/litre, while the serum lead level decreased to 51 ug/100 ml. Qualitative test for corproporphyrin was negative after intravenous versenate. He opted for oral therapy thereafter and was on oral penicillamine 250 mg/tds for two weeks. At the end of the treatment his blood lead level was 44 ug/100 ml and his haemoglobin rose to 12.9 gm%.

DISCUSSION

Lead is a highly malleable metal with a low melting point and is a versatile material in industry. It is used as sheet lead for flashings in the building industry, as battery plates in the lead accumulator industry, as a lining for vessels in the chemical industry, as type metal in the printing industry, and with an admixture of tin as solder. Its oxides are used in the paint industry whilst the organic compounds are used as anti-knock agents in motor spirit. Industrial lead poisoning usually arises from inhalation of dust or fume or from contact with the organo-lead compounds. As early as 1972, lead poisoning as an occupational hazard was reported in Singapore when Lai described a Chinese opera actor with acute encephelopathy from prolonged exposure to lead containing facial make-up powder[2]. A year later Chan[3] reported one case of lead encephelopathy due to poisoning with organo-lead compounds. It is of relevance to note, that lead poisoning can occur in industries that are not traditionally associated with high risk of lead poisoning as in the case of our patient who works in a plastic manufacturing plant.

The diagnosis of lead poisoning in this patient was based on a history of abdominal colic, the presence of anaemia, a high lead concentration in the factory environment and repeatedly high blood lead levels. Coproporphyrin was detected in his urine and his 24-hour urinary lead excretion (440 ug/litre) was well above the dangerous level of 250 ug/litre set by Lane et al[4]. There was also a clear history of exposure for 10 years. However the diagnosis of lead poisoning is not always straightforward. Beritic[5] described cases with severe lead poisoning and yet the blood levels were low, including some cases where the blood levels were less than 40 ug/100 ml. On the other hand, some people with high blood levels may not show symptomatic manifestations of plumbism at all.

In dubious cases measurement of 24-hour urinary lead excretion after 24 hours of intravenous administration of calcium disodium verenate may be helpful. Urinary excretion of more than 500 ug is diagnostic of lead poisoning [6].

The immediate treatment of lead poisoning is prevention from further exposure. Chelation therapy should be considered in severe cases particularly those with encephelopathy. Either intravenous calcium disodium versentate or oral penicillamine can be used. Chelating agents should not however take the place of simple preventive measures which keep the lead-in-air concentration in the working environment to a safe level. Our patient when first seen had a blood level of 100 ug/100 ml and on transfer to another department not actively involved in handling of stabilizers, his blood lead level fell to 71 ug/100 ml. This blood lead level was not truly reflective of the lead burden in his body as 90% of the total body lead is stored in bones[7]. During illnesses, demineralisation can cause mobilisation of toxic quantities of lead into the soft tissues and excerbate plumbism.[6] This fact together with the anaemia led us to treat the patient wth chelating agents.

This case illustrates the importance of monitoring of workers exposed to lead so that corrective preventive measures can be implemented before further or serious poisoning occurs. At the time of writing, the factory had complied with the recommendations and the lead-in-air concentration has decreased to between 0.06 mg/m^3 — 0.11 mg/m^3.

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