INTRODUCTION

Lead poisoning (LP) has long been recognized as an occupational hazard and indeed Hippocrates described the case of a metal extraction worker with abdominal colic.1 LP (also known as plumbism, colica pictorum, satorism or painter’s colic) can interfere with biochemical processes occurring in the cells throughout the body and as a result adverse effects occur in multiple organ systems. Occupational exposure is the main cause of LP in adults, as in lead miners, smelters, plumbers, glass manufacturers, cosmetics workers, battery workers etc. Another important source of LP is herbal medicines.2,3 Lead can be taken in through direct contact with mouth, nose, eyes and through breaks in the skin. The main body compartments that store lead are the blood, soft tissues and bones. LP can cause variety of symptoms and signs which vary depending on the individual and duration of exposure.4

Blood Lead Levels (BLL) remains the predominant biological marker used in clinical assessment, workplace monitoring, public health surveillance and regulatory authority decisions regarding removal from exposure. Erythrocyte Protoporphyrin IX (EP) which can be measured as Free EP (FEP) or Zinc Protoporphyrin (ZPP) is a measurement of biological effect and is an indirect reflection of lead exposure. Symptomatic patients with blood lead concentration > 2.4 μmol⁻¹ (50 μg/dl) or in any event > 3.8 μmol⁻¹ (80 μg/dl) should receive sodium calcium edetate intravenously followed by succimer by mouth.

CASE REPORT

Case 1: A 40 years old worker in battery manufacturing plant since 18 years presented with severe abdominal pain in medical OPD. He had intermittent episodes of same type of abdominal pain from last 15 years for which he had multiple emergency visits, and had extensive workup for abdominal pain including CT scan of whole abdomen, which was normal. Four years ago, he started developing mood swings and symptoms of psychosis; he remained on anti-psychotic medications for 2 years without any improvement. Two years ago, he consulted a neurologist for tremors of both hands and had been worked up for Wilson’s disease and was put on tablet Penicillamine, which he took for one and half year. In the meantime, during routine investigations, his urea and creatinine was found to be high but extensive workup by nephrologist (including cystoscopy) to rule out the cause was found to be futile. This time when he presented in medical OPD, keeping in mind his occupation, serum lead levels were done which turned out to be 98.83 μg/dl (normal ≤ 10 μg/dl). On detailed questioning, he gave a history of joint pain and headache but never had fits, lost consciousness or difficulty in walking. During breaks he ate, drank and smoked cigarettes at his workplace. He changed clothes before leaving workplace. On physical examination, he was afebrile with respiratory rate of 16/minute, pulse 82/minute and blood pressure 130/80 mmHg. He was pale (anaemic), had slurred speech, gingival lead lines, tremors of both hands. Neurological examination revealed left wrist drop and weakness of grade 4/5 of right hand. His hemoglobin (Hb) was 7.2 gm/dl. MCV was 83 fl, TLC was 4.9 x 10³/L, platelets 176 x 10⁵/L. There was no basophilic stippling. Total bilirubin 0.6 mmol/L, SGPT 44 IU/L (20 - 40), alkaline phosphatase 426 IU/L, urea was 66 mg/dl and creatinine was 1.8 mg/dl. His HBsAg and anti-HCV were normal. Blood glucose and urine analysis was normal. He was...
successfully treated with 2 cycles of intravenous CaNa EDTA. At a dose of 40 mg/kg/day for 5 days other relevant investigations appear in Table I.

**Case 2:** A 38 years old male, working in a battery manufacturing plant as a machine operator for last 23 years presented with complaints of abdominal pain, malaise, vomiting, loss of appetite, vertigo, headache, irritability and burning sensation in feet. He was having these symptoms intermittently from last 3 - 4 years. He visited a general physician several times and received symptomatic treatment. Two years ago, he started developing tremors of both hands, along with mood swings and fatigue for which he consulted neurologist and was treated for parkinsonism for 1 year but symptoms did not improve. After some time, he developed aggressive behavior and irritability and was referred to psychiatrist and was put on antipsychotics. He took these medications for 6 months but despite of these medicines, his symptoms flared up along with intermittent episodes of diarrhea and this time he was labeled as a case of irritable bowel syndrome. He was also seen by hematologist and had bone marrow examination (to look for cause of chronic anemia) which showed erythroid hyperplasia. He had multiple blood transfusions.

On physical examination, he was afebrile, BP: 120/80 mmHg, R/R: 18/minute, was anaemic and nails were discoloured (Figure 1). He had slurred speech and a tremor of both hands; rest of the examination was normal. Later in relation to his workplace and symptoms, his lead levels were done which came out to be 120.20 µg/dl. His Hb was 7.1 gm/dl, MCV 72 fl, TLC 9.8 x 10³µl, platelets 140 x 10³µL. There was no basophilic stippling. Urea 105 mg/dl and creatinine 3.0 mg/dl, bilirubin 0.4 mmol/L, SGPT 49 IU/L, viral markers were negative. He was successfully treated with intravenous EDTA at a dose of 50 mg/kg/day for 5 days. Post-treatment levels were 41.75 µg/dl.

At review 14 days later, his Hb was 9.0 g/dl as other relevant findings appear in Table I.

### DISCUSSION

Lead intoxication is highly prevalent among persons chronically overexposed to lead. Occupational exposure is the main cause of lead poisoning in adults, as in lead miners, battery workers, smelters, plumbers, glass manufacturers, cosmetic workers etc. In our country, the main source of occupational lead toxicity is battery manufacturing plant. Constant exposure results in accumulating lead in blood (where 95% of lead is associated with the erythrocytes) and soft tissues until a steady state is reached. About 60 - 90% of body's lead burden is deposited in the skeleton. The half-life of lead in these tissues is measured in weeks for blood, months for soft tissues and years for bone (may be stored for upto 30 years). Lead is removed from the body very slowly, mainly through urine. Smaller amounts of lead are also eliminated through the faeces, hair, nails and sweat.

The amount of lead in the blood and tissues as well as the time course of exposure determines the toxicity. Lead Poisoning (LP) may be acute (from intense exposure of short duration) or chronic.

Gordon et al. reported 3 cases of acute lead toxicity, related to removal of leaded paint, and another case by Hart et al. Lead may cause variety of symptoms and signs which vary depending on the individual and duration of lead exposure. Symptoms are non-specific and may be subtle and someone with elevated lead levels may have no symptoms. Early symptoms of LP in adults are commonly non-specific and include depression, loss of appetite, intermittent abdominal pain, nausea, diarrhea, constipation and muscle pain. Unusual tastes in mouth and personality changes are also early signs. With prolonged exposure, adults might develop headaches, abdominal pain, memory loss, kidney failure, male reproductive problem and weakness, pain or tingling in the extremities. Sometimes patients develop severe abdominal pain (lead colic) that mimic acute abdomen.

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Normal range</th>
<th>Before chelation</th>
<th>After 1st chelation</th>
<th>After 2nd chelation</th>
<th>Six months after discontinuation of chelation</th>
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<tr>
<td>Blood Lead</td>
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<tr>
<td>Case I</td>
<td>Acceptable up to 10 µg/dl</td>
<td>98.83</td>
<td>62.2</td>
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<td>120.20</td>
<td>71.05</td>
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<td>Hb Level</td>
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<tr>
<td>Case I</td>
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<td>8</td>
<td>9.4</td>
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<tr>
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<td>9</td>
<td>9.7</td>
<td>10.1</td>
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Table I: A dose of 40 mg/kg/day for 5 days.
Mohammad et al. reported a case of occupational lead poisoning in an adult battery worker with severe abdominal colic who initially underwent appendectomy. Later he was diagnosed with lead poisoning and was treated. Other overt symptoms include hypertension, peripheral neuropathy, ataxia, tremors, gout, nephropathy and anaemia. In general symptoms increases with increasing BLLs.

Both these cases worked in battery manufacturing plant for several years. They presented several times with non-specific symptoms of abdominal pain, received symptomatic treatment and remained undiagnosed for almost 15 years. Misdiagnosis may occur due to unawareness of lead poisoning symptoms. Both patients had involvement of almost every system, nervous system, kidney, gastrointestinal and liver as indicated by mild elevation of ALT as a result of chronic lead exposure.

Both patients were inappropriately treated for Parkinsonism and Wilson’s disease. Paying attention to a good occupational history will prevent many unnecessary or avoidable medical interventions. The setting of exposure, working in the battery factory represents established source of dangers.

As adult lead poisoning becomes less common, it is important to be aware of the problem in persons who present with non-specific symptoms and to take full occupational history. The dangers of lead exposure in workers, who are involved in manufacturing of battery, should be appreciated for there are relatively few reports of this potentially important source of intoxication.

Occupational lead poisoning is preventable disorder and a serious challenge for health and labor authorities in the developing countries.

The single most important aspect of treating lead poisoning is removal from exposure, yet there may be important socio-economic constraints for a given individual that limits this approach. Same happened in here as both patients returned back to the same work after 6 months of treatment. Despite of removal from the source of exposure, both showed sustained elevation of blood lead long after therapy, this is compatible with continued release of lead from skeleton.

REFERENCES