Case Report

A Report on Lead Toxicity

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ABSTRACT

Patients with vague complaints with multisystem involvement often present to the Emergency department (ED). Once routine differential diagnoses are ruled out, the rarer differentials although suspected, cannot be confirmed in the ED. Following up patients after admission could improve the diagnostic skills and knowledge of Emergency physicians (EPs), as well as the internist. A middle aged man presented with "twisting" sensation in the abdomen, vomiting , numbness of both hands and difficulty in articulation. Later he developed ventricular bigeminy. All routine investigations and imaging studies were normal. He was taking Indian Herbal medication for alcohol de-addiction. As a definitive diagnosis could not be arrived at, he was admitted for monitoring. Retrospectively, a differential diagnosis of heavy metal poisoning was considered by the EP. The laboratory result was positive for lead. Obstacles faced by the EP were diagnostic challenges in the chaotic ED, to logistic challenges in following the patient up and sending samples in cold storage, to a laboratory several hundred kilometres away.

Keywords: acute lead poisoning; emergency medicine; folk medicine.

INTRODUCTION

Lead has been used for a variety of technological purposes; but it has now become a health hazard.[1,2] Recent human skeletal remains show very high lead levels.[3] Various policies for regulation of lead have failed in the developing world. Further, folk medicines containing heavy metals are widely used in these countries.[4] In contrast with acute intoxication, a chronic, low-level intoxication is a greater diagnostic challenge, as symptoms are usually vague and variable.[2] We describe one such case that presented to the ED with multiple vague symptoms, later found to have acute on chronic lead poisoning.

CASE-REPORT

A 39 year old Indian Hindu policeman presented to the ED with "twisting sensation in the abdomen", numbness along with spasms in his right hand and difficulty in articulation, which appeared simultaneously an hour ago. On examination he was irritable with normal vital signs. Power in all limbs was 5/5. Cerebellar signs (slurring of speech and dysdiadochokinesis) were positive. Considering a possible acute cerebellar stroke, in the window period, a CT and MRI brain were done which were normal. Subsequently, he developed eight episodes of vomiting not responding to multiple doses of ondansetron and metoclopramide, and three episodes of non blood-stained loose stools and continued to complain of abdominal discomfort.Abdominal examination, X rays and ultrasound scan were normal. Further questioning revealed that he had been consuming around 180 ml of hard liquor almost daily for 10 years. Over the last two months he was taking Ayurvedic tablets for de-addiction. His last drink was 48 hours ago. He had no known drug allergies. A 12 lead ECG showed ventricular bigeminy initially and later multifocal ventricular ectopics including a run of three. ABG revealed partially compensated respiratory alkalosis with a potassium of 3.4 meq/L. Serum magnesium levels were ordered and potassium chloride IV 20 meq over 2 hours along with magnesium sulphate 2g IV in 100 ml NS over half an hour were administered. He was transferred to the High Dependency Unit (HDU) with the provisional diagnosis of alcohol dependence syndrome, with VPCs due to dyselectrolytemia. The cause of the abdominal discomfort and neurological complaints remained obscure. Haemoglobin levels, platelet count, LFTs, serum sodium & chloride and routine urine study were all normal. ABG showed respiratory alkalosis. In

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the HDU IV piperacillin and multivitamin were started. Due to persisting cerebellar signs, "viral cerebellitis" was thought of but the CSF study was normal. On the EP's request, heavy metal poisoning was considered. An EDTA sample for heavy metals was sent to a city 12 hours away by cold storage. The patient improved and was discharged after 2 days. A week later his reports arrived showing a blood lead levels (quantitative) of 28.9 mcg/dl and arsenic, mercury, antimony below detectable levels. The high lead levels confirmed that the abdominal and neurological features were due to lead poisoning. However the patient was lost to follow up.

Table 1: Blood Investigation Report

Lab Parameter	Values
Total WBC count	14,300 /microlitre
Differential WBC count	Neutrophils – 96 % ; lymphocytes – 4%
Serum magnesium	1.7mg/dl
Serum potassium	4.1mEq/l
Creatine phosphokinase-kinase MB (CPK-MB)	39 U /L

Table 2: Blood levels of Lead requiring Chelation therapy.

Normal	< 10 mcg/dl
Borderline	11-25 mcg/dl
Abnormal	26-40 mcg/dl
Chelation required	>40-50 mcg/dl

DISCUSSION

Lead poisoning may present acutely or chronically. Although our patient's presentation was acute, there appears to be chronic lead intake , historically. Acute toxicity may affect the renal, hepatic, GI, hematopoietic and the CNS.[2,4] Despite presenting with vomiting, loose stools and neurological dysfunction, strikingly, there was no abdominal pain. The same findings were reported by Rolston et al.[4] mentioning 2 cases with blood lead 50 and 95 µg/dl which did not have abdominal pain. The etiology of the classic 'lead colic', is not well understood. Perhaps, lead competes for transport with calcium and other divalent cations such as magnesium and zinc, which in turn, interferes with mitochondrial oxidative phosphorylation and intracellular signalling processes affecting intestinal motility. Our patient's lead levels were 28.9 mcg/dl, in the "abnormal" category. In 1991 the CDC established a blood lead level of 10 µg/dL as the lower level of medical concern. This level has been adopted by WHO.[2] Further, a study shows that blood lead levels even below 10 µg/dL, although mostly in children, may cause cognitive dysfunction, neurobehavioral and neurological disorders.[2] Alteration in sensory systems and vision, hearing, and balance deficiencies in lead poisoned individuals are also well documented.[5] The common domestic sources of lead are unglazed pottery, cosmetics and herbal remedies, especially those from Asia and India.[6] Case reports by Keen, R.W. et al.[7] mention Indian herbal remedies for diabetes as a cause of lead poisoning. Dunbabin, D.W. et al.[6] also reports lead poisoning from Indian herbal medicine (Ayurveda). In our case since the patient clearly mentioned mixing an Ayurvedic preparation in alcohol before consuming it; we attributed the source of lead poisoning to be the same. Apart from the blood levels, gene expression profiling can be utilized to confirm lead poisoning.[5] The plausibility of the gene profiling needs to be explored further as it could be a useful tool.

Limitations: Hypomagnesemia may have been a confounding factor. Serum magnesium levels were 1.7 mg/dl (normal lab levels- 1.8- 2.2 mg/dl) , the sample being drawn in the HDU after 2 g I.V correction was empirically given in the ED for the ventricular ectopics. Hypomagnesemia is known to occur in alcohol use disorder and can cause similar clinical features. The blood lead test is accurate in brief acute exposures and cannot be used for past or chronic exposures. In contrast, blood ZPP(zinc proto porphyrin) levels remain elevated for several months after the exposure.[5] but this was not available. The source of lead was most likely to be the herbal medicine, however this could not be confirmed by analysis of the compound.

CONCLUSION

Ethnic herbal preparations can cause unexplained symptoms and could sometimes be lethal. Hence their use should be monitored.[7] Heavy metalpoisoning should be considered in patients with multisystem complaints and history suggesting possible exposure. Screening for heavy metal poisoning is critical for the development of rational, cost-effective and science-based public health

policies.Such screening tests should be made more readily available.[8] Despite being in a busy ED, the EP must not give up the quest for differentials. He must follow patients up and find his way through the obstacles that come with investigating relatively rarer diagnoses in select patients, when routine diagnoses are ruled out.

CONFLICTS OF INTEREST

Declared none.

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