

Lead Poisoning Mimicking Acute Porphyruria!

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ABSTRACT

We are presenting a case of a 13-year-old autistic boy whose urine porphyrin test came positive on three separate occasions. The child was brought to emergency department of Kasturba Medical College Hospital, Attavar, Mangalore, India, with fever and acute abdominal pain, with no previous history of any serious illness. Investigations revealed thalassemia trait, microcytic hypochromic anaemia while the other biochemical and haematological parameters were normal. False positive urine porphyrin test may be seen in porphyria induced by liver cancer, hepatitis and heavy metal poisoning such as lead, arsenic and mercury. Blood lead (PbB) level was 59.5µg/dl. Further evaluation revealed a daily consumption of native medicine in the form of syrup.

Keywords: Lead poisoning, Porphyruria

CASE REPORT

A known autistic 13-year-old boy was brought with high grade fever, vomiting, severe colicky abdominal pain and constipation of three days' duration to the emergency department of Kasturba Medical College Hospital, Attavar, Mangalore, India. The child had normal developmental history with no major illness in the past, except for autism and was vaccinated to date. Sudden onset of a single episode of seizure during the hospital stay led to extensive evaluation of this patient. History of consumption of native medicine in the form of syrup for a period of two years. No history of pica.

On general physical examination the child was conscious, oriented to time, place and person. He was febrile and had pallor. He had no icterus, enlarged lymph nodes and koilonychia. Per abdominal examination revealed tender abdomen, with no hepatosplenomegaly. Systemic examinations had no detectable abnormality.

INVESTIGATIONS: Hemoglobin was 7gm%, total leucocyte count was 3500 cu mm and platelet count was 1,33,000cu mm. Peripheral smear showed microcytic hypochromic anemia. CT scan of brain and the CSF analysis were normal. Serum electrolytes were normal.

Liver and renal function tests were normal. Hemoglobin variant analysis pattern suggestive of thalassemia trait. Urine porphyrin tested positive on three separate occasions.

Blood lead (PbB) level was 59.5µg/dl, estimated using ESA model 3010B lead analyser (ESA, Inc, Chelmsford, MA) based on the principle of differential pulse anodic stripping voltammetry (DPASV).

Management: Patient was treated symptomatically. Two units of packed cells were given for correction of anaemia. Patient had no further episodes of seizure and did not require anticonvulsant therapy. The elevated lead levels prompted the clinician to elicit a thorough history from the parents which revealed that the child might not have come in contact with other possible sources of lead such as toys, paint or mud. However, they confirmed administering native medicine in the form of syrup for the past two years. Following discharge from the hospital parents were advised to stop the native medicine.

DISCUSSION

The word "porphyria" derived from the Greek means purple. When enough porphyrin is present in the urine, it may have a red-purple, red-brown, or "port wine" appearance. In addition, free porphyrins exhibit a red fluorescence under UV light. Porphyrinuria, is unrelated

to primary disease in the heme synthetic pathway [1]. Lead is a heavy metal, used in industries for manufacturing paints, and an important source of this metal. Ingestion and inhalation are the main routes of absorption. When absorbed, around 99% of lead is retained in the blood for nearly 30-35 days and is subsequently disseminated and accumulated in other tissues – liver, renal cortex, aorta, brain, lungs, spleen and bones [2]. In acute lead poisoning, children present with ataxia or with seizures or reduced level of consciousness [3]. In patients with vague abdominal pain, screening test of the urine with Ehrlich's reagent is routinely done to rule out acute intermittent porphyria [4].

Excess porphyrins in the urine, has been noted in cases of lead poisoning as far back as 1895. Several studies on the effects of lead have revealed inhibition at several sites of heme biosynthesis [5]. This is consistent with our findings. The most common means of exposure are inhalation of air contaminated with lead dust, ingestion of lead -tainted food or water, or direct contact with lead -polluted soil.

Lead exposure can be detrimental to every organ in the human body, but the brain is predominantly susceptible to its deleterious effects, especially in childhood [6]. Normal acceptable blood lead levels are less than 10µg/dl [2].

Major share of native medicines across the world are produced in India and this raised a conspicuous problem when scientific papers did demonstrate the presence of high levels of lead and other toxic heavy metals in the ayurvedic preparations sold via internet in western countries [3].

Lead exposure and the precise mechanisms by which it affects the development and function of the central nervous system are not identified so far. Oxidative stress, deregulation of calcium signalling and abnormal neural transmission/gene expression pathways have been proposed to cause lead neurotoxicity [6].

Lead poisoning can present with the nonspecific signs and symptoms such as abdominal pain, constipation, irritability, and anaemia with individual variation [7].

CONCLUSION

A child with acute porphyria like symptoms with urine porphyrin positive should raise a possibility of heavy metal poisoning. In such cases blood levels of lead, arsenic and mercury should be estimated. The composition of the indigenous preparations should be tested and validated as per the norms before release into the market.

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