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Case Report

ARSENIC POISONING IN AN OPIUM ADDICT

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ABSTRACT
Arsenic has been known to be a deadly poison from time immemorial. Here we present a case of chronic arsenic poisoning in an opium addict. Opium is known to have been adulterated with arsenic in certain parts of north India. Patient a 45year old farmer presented in emergency department with altered sensorium, decreased appetite, periorbital puffiness. He had history of opium abuse. On physical examination icterus, chemosis, proptosis was seen. Mees lines and hyperparakeratosis of soles were also seen. Toxicology report confirmed arsenic poisoning. Patient was given Dimercaprol and was dialyzed following which his condition improved and he was discharged. We suggest that in patients with history of opium abuse and above said complaints should be evaluated for arsenic poisoning.

Key Words: *Arsenic Poisoning, Adulteration*

INTRODUCTION
Arsenic, element 33, historically a sinister poison from maurya era(india) and napoleon era to present times ranks about 20th in abundance in the Earth's crust and 12th in human body (Mandal and Suzuki, 2002). Arsenic affects multisystem of the body. Both acute and chronic forms have varid presentation and manifestations.

CASES
We present a case of 45 year old male, a farmer by profession, who was a chronic opium addict for past 20 years and a reformed alcoholic for 20 years, was brought into emergency with chief complaints of altered sensorium, decreased appetite, loose stools, vomiting, decreased urine output, periorbital puffiness since 3 days prior to admission. There was no history suggestive of infective pathology. Past medical history revealed history of intake of some indigenous medications and opium for joint pains. Patient also used to consume opium before going to fields to work.

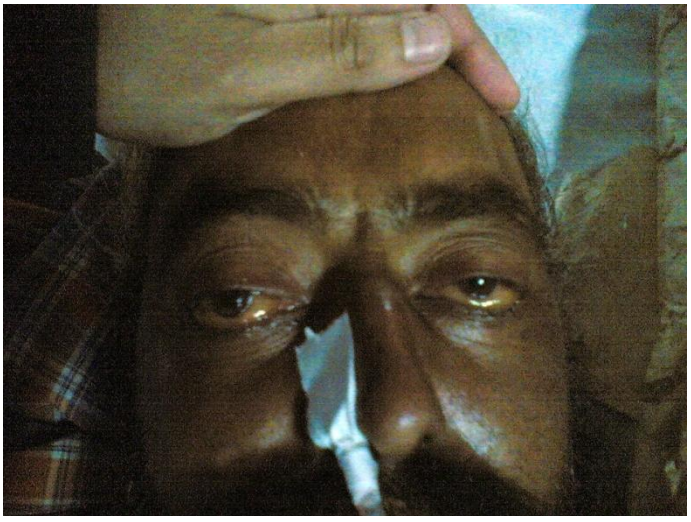

Figure 1: The patient

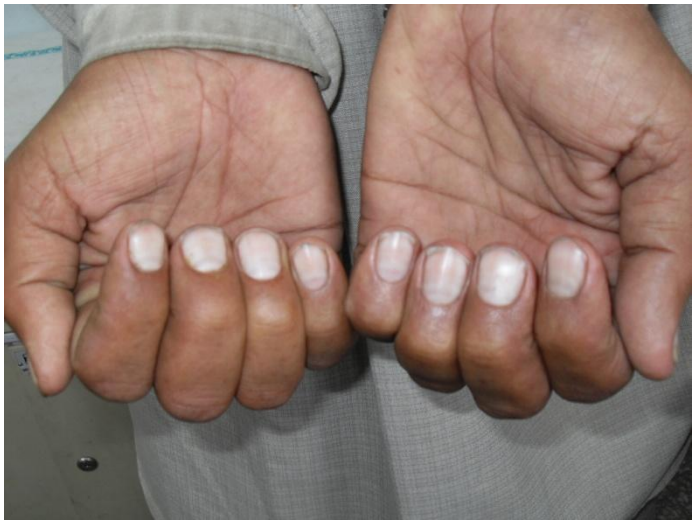

Figure 2: Nails revealing Mees lines

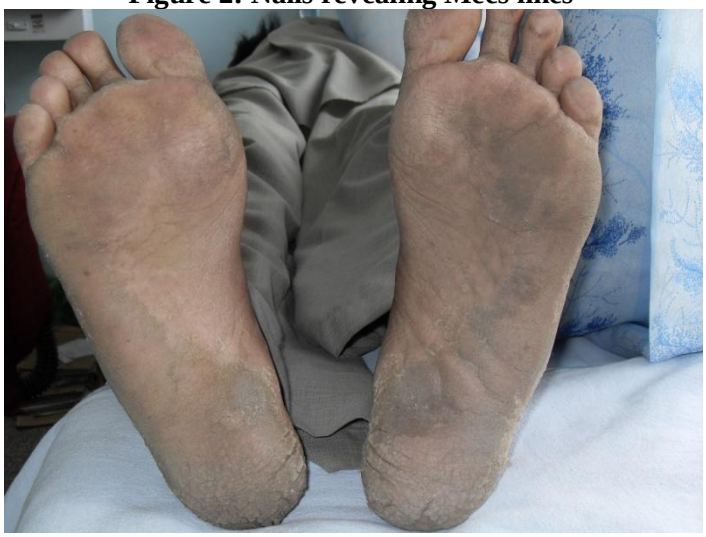

Figure 3: Hyperkeratosis

General physical examination revealed middle aged restless afebrile man , disoriented to time place and person, he was pale dehydrated and had a pulse rate of 180/min, blood pressure of 150/90 mm of Hg, respiratory rate of 24/min, jvp raised 10 cm. Icterus, chemosis and proptosis (Figure 1) was present. Local examination of nails revealed Mees lines (Figure 2), hyperkeratosis (Figure 3) and dark pigmentation of palms and soles.

Cardiovascular and respiratory system revealed so significant findings. Neurological examination revealed restless, disoriented man, moving all four limbs to painful stimuli with no obvious cranial nerve palsy or autonomic disturbance. Blood culture revealed normocytic, normochromic anemia with hemoglobin level of 11.8 gm. His total bilirubin was 2.13, no electrolyte abnormality was noted, and however he had blood urea level of 191 mg/dl and creatinine of 4.3mg/dl. Rhuematoid factor, ANA and Anti DSDNA, c ANCA and p ANCA were negative. Stool examination was positive for occult blood and mucous but no ova of cyst were seen.MRI study of brain was essentially normal. Ultrasound abdomen revealed hepatomegaly, dilated portal veins, thickened and edematous gall bladder, and spleen enlargement with cranio caudal span of 14 cm.

Case Report

In view of history of ingestion of opium and indigenous medications (which are sometimes adultrated with arsenic) and clinical findings patient was evaluated for heavy metal poisoning including arsenic poisoning. His hair and nails were sent to toxicology lab for evaluation of heavy metal poisoning and it showed very high levels of arsenic (Datta, 1978).

Patient was given Dimercaprol in dose of 5 mg/kg every 4 hours for one day, followed by every 6 hours on second and then every 8 hours for 7 additional days. He was also dialyzed following which his sensorium improved gradually. He was discharged after 2 weeks in a satisfactory condition.

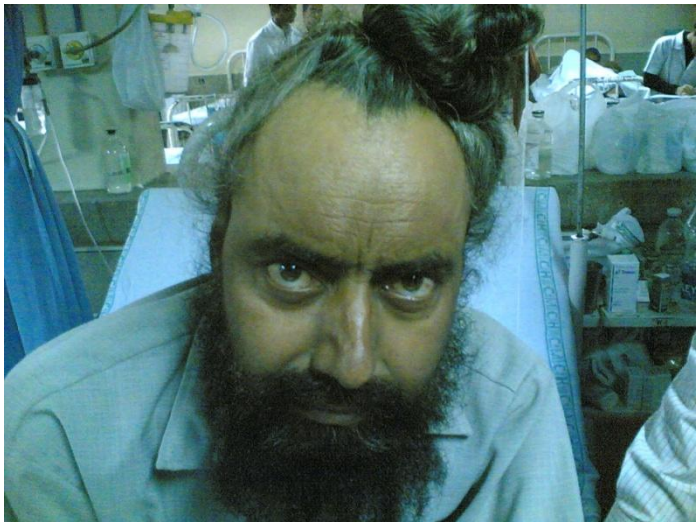

Figure 4: Recovery

DISCUSSION

Arsenic occurs in 4 valence states with arsenic gas as most toxic arsenical. Arsenic poisoning source ranges from industrial exposure, to contaminated wine (moonshine whiskey), electronic industry, and pressure treated woods and primarily in the treatment of acute promyelocytic leukemia and other cancers (Emadi and Gore, 2010).

Arsenic inhibits pyruate dehydrogenase thereby inhibiting acetyl coA production, citric acid cycle, gluconeogenisis and fatty acid oxidation. It has been found to alter monocyte superoxide anion production and inhibit nitric oxide production (Luna *et al.,* 2010). It increases repolarisation phase of action potential, accelerating atherosclerosis, increasing platelet aggregation and reducing fibrinolysis (Balakumar and Kaur, 2009). It has been associated with skin, lung and bladder cancers.

Acute Arsenic affects multisystems of the body. In gastrointestinal system it can cause GI lesions, extensive inflammation and necrosis of gastric mucosa and sub mucosa, hemorrhagic gastroenteritis and gut wall perforation. In kidneys acute tubular necrosis with acute renal failure, cortical necrosis, protienuria can occur.

Acute arsenic poisoning causes diffuse capillary leakage and cardiomyopathy leading to hypotension, shock and vasodilatation. These changes manifest in ECG as broadening of QRS complex, prolonged QT interval, ST depression, flattening of T waves. Gangrene of extremities known as Blackfoot disease pathologically due to arteriosclerotic changes has also been reported .Arsenic poisoning can cause peripheral neuropathy with sensory neurons involvement more than motor neurons in a glove and stocking distribution (Murphy *et al.,* 1981).

Chronic toxicity manifests as hyperkeratosis with classical dew drops on a dusty road appearance, peripheral neuropathy and MEES lines on fingernails. Chronic hepatic and renal damage may also be seen. Arsenic gas typically presents with acute hemolytic anemia and shaking chills. Bone marrow depression may result from acute or chronic arsenic intoxication presenting as pancytopenia.

Diagnosis mainly depends upon exposure history, clinical presentation, and laboratory tests. Early (24-48hrs) total urinary arsenic levels greater than 100micrograms per liter is considered abnormal. Blood picture showing arsenic induced anemia, leucopenia, thrompocytopenia and basophilic stippling may aid in diagnosis. Elevated transaminases may also be seen.

Conclusion

We suggest that in patients presenting with altered sensorium, malena and renal failure and mees lines, history of opium ingestion should be taken and possibility of heavy metal arsenic poisoning should be kept in mind since there are reports of high level adulteration of opium with arsenic in certain parts of north India (Datta, 1978).

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