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Case Report

Importance of urine sodium dithionite test in paraquat poisoning: a case study

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ABSTRACT

Suicidal tendencies are one of the major social problems worldwide. It is equally seen in Indian population. Organo-phosphorus compounds are usually preferred because of their easy availability. Paraquat is one of the lesser known herbicide agent, which is not an OP compound. It is highly toxic and causes damage to the lungs, liver and kidneys. Occurrence of paraquat poisoning is rare, but has very high mortality rate. Here we report one such case which was earlier misdiagnosed as organo-phosphorus poisoning, but diagnosed later by qualitative urine sodium dithionite test. It is a simple test for early diagnosis of paraquat poisoning. It is recommended to performing this test in all suspected cases of oral poisoning.

Keywords: Paraquat, Dithionite, Organo-phosphorus poisoning

INTRODUCTION

Paraquat is one of the lesser known herbicide agent, which is not an organ-ophosphorous compound. Chemically, it is N,N-dimethyl-4,4-bipyridinium dichloride, also known as 'methyl viologen'. It is available in Indian market as Gramoxone, Weedol. It is highly toxic and causes damage to the lungs, liver and kidneys. Patient develops multi-organ failure leading to death.

Occurrence of paraquat poisoning is rare in India.[1] It has very high mortality rate. Here we report one such case which was earlier misdiagnosed as organo-phosphorus poisoning, but was confirmed as paraquat poisoning later by qualitative urine sodium dithionite test.[2]

CASE REPORT

17 years old male patient had history of consumption of unknown poison, which was suspected as organo-phosphorus poisoning. Baseline liver function tests and renal function tests were deranged suggesting hepato & nephrotoxic nature of poison. Clinical symptoms matched with OP poisoning & so appropriate treatment was initiated. Despite this treatment, there was no improvement in the patient. On 3rd day, physician noticed scalding over mucosa of left cheek. This led to suspicion of corrosive poisoning such as paraquat.

Urine sample was analysed for the presence of paraquat by sodium dithionite test, which was positive for paraquat. The treatment protocol was then modified accordingly and this led to dramatic clinical improvement of the patient. Blood investigations like LFT, RFT were monitored which returned to normal over next 3-4 days.

Urine sodium dithionite test

Since paraquat is not metabolised and excreted unchanged in the urine,[3] it can be easily detected by qualitative urine analysis.

1% solution of sodium dithionite is prepared freshly in 1N NaOH. To one volume of urine, 0.5 volume of this freshly prepared solution is added. Sodium dithionite reduces paraquat to blue coloured radical form in alkaline medium. The colour is observed at the end of one minute. Blue colour indicates presence of paraquat in excess of 0.5 mg/lit. Both positive and negative controls should be run.

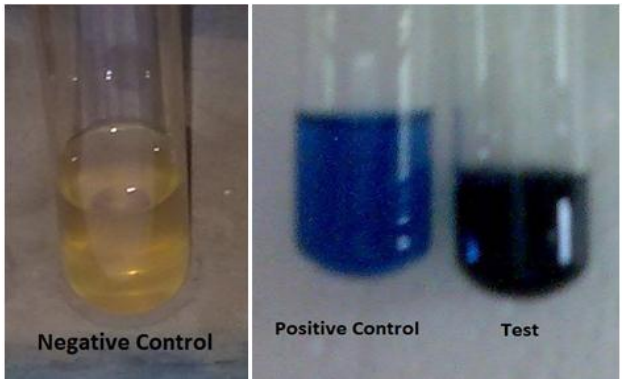

Figure 1: Shows negative and positive controls, and test samples in urine sodium dithionite test respectively.

DISCUSSION

Paraquat poisoning has life threatening effects on GI tract, lung, liver, kidney. The LD_{50} in human beings is approximately 3-5 mg/kg, which translates into as little as 10-15 ml of a 20% solution. The lung is target organ of paraquat and pulmonary effects represent the most lethal and least treatable manifestation. The primary mechanism is through generation of free radicals. Acute pulmonary oedema and early lung damage may occur within few hours of exposure.

Both type I and II pneumatocytes appear to selectively accumulate paraquat. Biotransformation of paraquat in these cells results in free radical production with resulting lipid peroxidation and cell injury, after which there is rapid proliferation of fibroblasts. There is progressive decline in arterial oxygen tension and CO_2 diffusion capacity. Progressive cyanosis and dyspnea reflect deteriorating gas exchange in the damaged lung. In some cases, coughing up of frothy sputum (pulmonary edema) is the early and principal manifestation of paraquat lung injury. Such a severe impairment of gas exchange causes eventual death due to asphyxia and tissue anoxia.

The GI tract is the initial site of toxicity, which is manifested by swelling, edema and painful ulceration of mouth, pharynx, esophagus, stomach and intestine. With higher levels, toxicity includes centrizonal hepatocellular injury which causes elevated bilirubin and enzymes like AST, ALT and LDH.

Impaired renal function may play a critical role in determining the outcome. The hepatic injury from paraquat may be severe enough to cause jaundice, however, hepatotoxicity is rarely a major determinant to clinical outcome.

Oral poisoning patients are treated symptomatically. The routine investigations like LFT, RFT, electrolytes are done only to monitor baseline status of the patient. No special tests are performed on routine basis, which usually miss such life threatening poisoning.

CONCLUSION

Urine sodium dithionite test is a simple test, which is used for detection of paraquat in urine. As paraquat poisoning has very high mortality rate[4] and is often confused with organo-phosphorus poisoning, simple sodium dithionite test on urine sample can confirm paraquat poisoning and save a precious life. It is suggested to carry out this test in all suspected cases.

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