Asian Journal of Biomedical and Pharmaceutical Sciences *ISSN 2249-622X*

Brain Imaging and Neurological manifestations in yellow phosphorous poisoning.

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Accepted on 03, January 2022

Abstract

Objectives: We present a case of 18 months old child with yellow phosphorous poisoning. Brain imaging features in yellow phosphorus poisoning have not been described before.

Methods: We have described MRI brain features of yellow phosphorous poisoning and its correlation with follow up neurological assessment.

Results and Discussions: We have found yellow phosphorous has direct toxic effect on brain. Our case also emphasizes the importance of timely diagnosis, excellent supportive care and clinical assessment at regular interval.

Key words:

Yellow phosphorous, Hepatic encephalopathy, MRI, Neurological manifestations

Introduction

We present a case of 18 months old child with yellow phosphorous poisoning. We have described MRI brain features of yellow phosphorous poisoning and its correlation with follow up neurological assessment. MRI brain imaging features of yellow phosphorous poisoning have not been described before in literature. Patient information - Eighteen months old child was brought to emergency room. Child had normal birth, nutrition and developmental milestones. Child did not have any prior illness. Clinical findings and timeline Child was brought with history of accidental ingestion of rat killing poison 4 days prior. Immediately after ingesting poison, gastric lavage was given. On the second day child had one episode of vomiting and became drowsy. In view of hyper acute liver failure child was brought to our centre on fourth day for further management and liver transplant child was afebrile, pulse 130/ min, RR 30/min, BP 110/65 mmHg, spo2 98%, weight 9.6 kg. On general examination child had icterus. Cardiovascular and respiratory examination was normal. Child was drowsy but was obeying commands. Diagnostic assessment Liver function test was grossly abnormal. Renal function tests were mildly deranged. Complete blood count, electrolytes, blood sugar were normal. Ammonia, prothrombin time and fibrinogen were abnormal. 2 d echo was normal. On liver biopsy histopathology, panacinar necrosis with macrovescicular steatosis was seen. MRI brain was done 2 weeks after consumption of poison and later follow up scan at 3 months. Cerebral involvement was diffuse and symmetrical involving occipital, temporal and parietal cortex except in frontal region where it was predominantly on right side. Follow up scan after 3 months showed gross cerebral atrophy. These MRI changes suggest poison has direct irreversible toxic effects on cerebral cortex. Persistent downgaze with bruxism was initially thought as nonconvulsive status epilepticus so prolonged serial EEGs were done. None of the recording showed epileptiform activity. All recordings showed delta to theta background with presence of triphasic waves. Persistent downgaze can be observed in hepatic encephalopathy due to nonstructural midbrain involvement. With improvement in hepatic encephalopathy sensorium improved. Persistent downgaze & bruxism became intermittent in nature and gradually completely subsided. Therapeutic information: Child underwent cadaveric split liver transplantation.

Description

Child received plasma exchange followed by CRRT which gradually improved urine output and renal function. Mannitol and 3% saline were given to reduce cerebral edema. Treatment after liver transplant included steroids and tacrolimus. Follow up and outcome Child sequentially achieved neck holding, unsupported sitting, standing, walking and also regained language & social milestones over 2 months. Child is now able to walk unassisted, plays with peers, communicates with small sentences. Yellow phosphorous is highly poisonous and act at the level of protoplasm. Yellow phosphorous is widely used in rodenticide and firearms. Most common organ to get involved is liver which can lead to toxic hepatitis and acute fulminant hepatic failure. Kidney involvement leads to acute tubular necrosis and acute renal failure. Cardiotoxicity can lead to arrhythmias, hemodynamic instability and cardiogenic shock. Bone marrow toxicity presents as cytopenia. Neurological symptoms are confusion, irritability, drowsiness, lethargy, coma. [1-3]. According to one study, post-mortem organ examination showed diffuse petechial hemorrhages in liver, heart & intestines [4, 5]. As there is no antidote for yellow phosphorous, early gastric lavage is extremely important. Our case received gastric lavage within 24 hours. If patient

develops liver failure timely liver transplant is crucial for survival [6, 7]. Our patient underwent liver transplant on 5th day after poison consumption. Considering abnormal liver function test, hyperammonemia and gradually worsening sensorium, diagnosis of hepatic encephalopathy was made. Presence of hepatic encephalopathy was also confirmed by EEG findings and persistent downgaze with bruxism. All these symptoms improved slowly after liver transplant over a period of one month.

Conclusion

Child had mild pancytopenia which improved after liver transplantation. Acute renal failure was treated with plasma exchange and CRRT. There was no cardiotoxicity. We attributed neurological symptoms largely to hepatic encephalopathy and to some extent effect of toxin on cerebral cortex. Bilateral diffuse cortical diffusion restriction with edematous gyri which gradually resolved on follow Up MRI with setting in of atrophy suggest direct toxic effects of yellow phosphorous. Poison induced micro-hemorrhages were ruled out by normal SWI images. With the help of excellent supportive care and successful liver transplantation, all developmental milestones recovered gradually over a period of 2 months. But looking at gross cerebral atrophy, child may develop learning disabilities, average I.Q, or other cognitive impairment in future. Child needs regular follow up with cognitive and neuropsychological assessment over a period of next few years. According to our case, yellow phosphorous has direct toxic effect on cerebral cortex. Therefore serial MRI brain and regular neurological assessments are important. Our case also conveys the importance of early treatment of multi-organ involvement. Timely liver transplant plays the most vital role in survival and neurological recovery.

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