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Original Research Paper

General Medicine

OCULAR DIPPING IN TOXIC ENCEPHALOPATHY DUE TO ORGANOPHOSPHORUS POISONING

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ABSTRACT Ocular Dipping is a slower, arrthymic downward movement followed by a faster upward movement in patients with normal reflex horizontal gaze. It is most commonly reported in case of severe anoxic brain injury with associated diffuse cortical and lenticular pathology, but it has also been described in prolonged status epilepticus and prion disease.[1,2,3] A 26 year old Female presented with acute onset of intermediate syndrome and simultaneous early toxic encephalopathy after ingestion of combination of Chlorpyrifos with Cypermithrin who had ocular dipping.

KEYWORDS : Ocular Dipping, Toxic Encephalopathy, OP poisoning.

INTRODUCTION

Ocular Dipping is a slower, arrthymic downward movement followed by a faster upward movement in patients with normal reflex horizontal gaze. It occurs spontaneously but may at times be elicited by moving the limbs or neck as it is proposed to involve an activation of arousal pathways. Interestingly, either a return of consciousness or pentobarbital may abolish ocular dipping. A mechanism for ocular dipping may involve dysfunctional cortical/lenticular connectivity with rostral brainstem vertical gaze centers and arousal pathways.3,4 The prognosis for patients displaying ocular dipping is variable.

OP poisoning is the common cause of mortality and morbidity with acute cholinergic crisis is the commonest form of presentation of such cases, the intermediate syndrome and delayed encephalopathy can be also presented in op compound. Thus we report a 26 years female with toxic encephalopathy who had ocular dipping.

Case Report

A 26-year-old female presented with alleged history of OP Poisoning (Chlorpyrifos and cypermethrin) on admission, she was conscious, oriented with history of loose stools associated with abdominal pain and salivations. The pupils were mid-dilated reacting to light. The pulse was 64 bpm and blood pressure 100/60 mmHg. Lungs were clear. No obvious lacrimation or fasciculation was observed. Primarily she was treated with antidotes atropine and pralidoxime. Laboratory examinations revealed a white blood cell count of 11,700/mL, Hb-11.2 g/dL, platelets -160000/cumm, sodium 136 mEq/L, potassium 4.0 mEq/L, Urea-28 and creatinine 0.69 mg/dL. SGPT 13 u/L, SGOT 26 u/L and S.bilirubin 0.65 mg/dL. Serum Cholinesterase-886 IU/L. An electrocardiogram showed normal sinus rhythm. On day 3 of admission, we noticed that her condition was deteriorating and there is an increase in respiratory rate, sweating, restlessness with neck weakness. In ICU, she was put on assisted respiratory support with mechanical ventilation and patient had focal right upper limb seizures of about 2 episodes each lasting for about less than 1 minute on day 6 of admission who was sequentially treated with leviteracetam 500mg twice a day since then.

During a consultation for encephalopathy, the patient was unable to respond well, follow commands, and move her limbs purposefully, with only minimal limb withdrawal to noxious stimulation. When her eyes were opened and she showed spontaneous frequent nonrhythmic, synchronous slow downward conjugate eye movements followed by a faster upward jerk, consistent with ocular dipping. The dipping movements occurred more frequently during passive limb movements and noxious stimulation. She had superimposed conjugate roving horizontal eye movements and her brainstem reflexes were intact. Eventually tracheostomy procedure was done to the patient and consciousness regained on day 18 of admission. With clinical improvement, the patient was discharged after closure of tracheostomy and full regain of muscle power without any neurological deficit after 30 days of hospital stay.

DISCUSSION

Ocular dipping is the term used to describe an arrhythmic slow conjugate downward movement followed in several seconds by a more rapid upward movement; it occurs spontaneously but may at times be elicited by moving the lims or neck. Anoxic encephalopathy has been the most common cause, but a few cases have followed drug overdose.

There are several ocular movement abnormalities that may be mistaken for ocular dipping. Reverse dipping is a variant of ocular dipping that has been described in the context of metabolic encephalopathy or encephalitis; in this variant, the eyes deviate slowly upward with a fast, downward phase to a primary position.

In contrast to ocular dipping, ocular bobbing involves fast downward eye movements and a slow return to a primary position; in ocular bobbing, which most often localizes to pontine lesions, the preservation of spontaneous, conjugate horizontal eye movements, and oculocephalic reflexes is variable and the is etiology is dependent.[2] Ocular dipping and bobbing are differentiated from vertical nystagmus by their irregularity, brief pause between changes in phase, and facilitation by noxious stimuli.[2]

CONCLUSION

Ocular dipping is commonly seen in anoxic brain injury, rarely in toxic encephalopathy with reversal once the patient gain consciousness in some cases.

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