RESEARCH PAPER | Medical Science | Volume : 5 | Issue : 7 | July 2015 | ISSN - 2249-555X

Brain Death in Organophosphorous Poisoning - Images in Clinical Medicine

KEYWORDS: Brain Death, MRI, Brain Herniation

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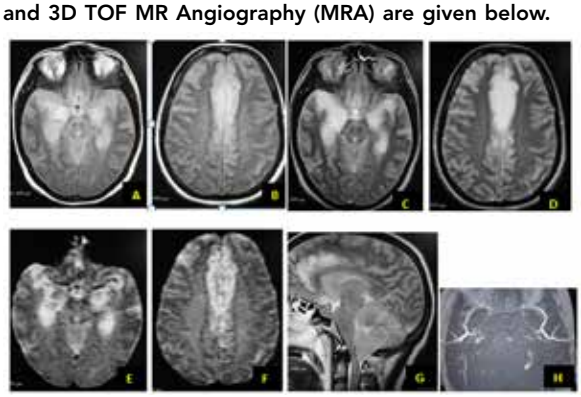
ABSTRACT *Acute organophosphate (OP) poisoning is one of the most common poisonings in emergency medicine. A large number of fatalities are seen in routine clinical practice at all levels of medical care. The common final pathway is probably respiratory failure leading to development of anoxic encephalopathy. Clinical diagnosis of brain death is often challenging and imaging modalities will aid in unambiguous diagnosis in all cases.We present a case of OP poisoning with fatal outcome and correlate it with MRI findings in brain death.*

CASE REPORT

25 yr old lady presented with accidental ingestion of Anti bug agent (NUVAN). She developed blurring of vision, increased salivation and breathlessness after 45 mins of consumption. Initially she was managed at a peripheral district hospital with gastric decontamination and was put on Inj Atropine and Inj Pralidoxime. She had recurrent episodes of tonic clonic seizures for which she was administered Inj Sodium Dilantin. At the time of admission this hospital she was comatose, with tachycardia, tachypnoea with pink frothy sputum. Pupillary examination revealed anisocoria (Right pupil normal in size and left pupil dilated) and both plantars were extensor. Detailed neurological examination could not be carried out. She was put on ventillatory support. She developed sudden fall in Blood pressure for which inotropes were added. Patient was taken up for MRI to rule out any intracranial pathology for recurrent seizures. She was managed with cerebral decongestants, Inj Atropine and Inj Pralidoxime and broad spectrum antibiotics. In spite of all the measures the patient had cardiac arrest and died the next day.

FIGURES

MRI images of T2 Fluid Attenuated Inversion Recovery Axial (FLAIR),T2 weighted Axial (T2WI), Diffusion Weighted Image (DWI), T2 weighted Sagittal (T2WI Sag) and 3D TOF MR Angiography (MRA) are given below.



IMAGING FINDINGS

There is poor differentiation between grey and white matter with extensive areas of increased signal on T2WI, FLAIR and DWI in temporal lobes and parietal lobes bilaterally with preferential involvement of the grey matter suggesting ischemic changes in these areas. There is compression of the lateral ventricles and basal cisterns with effacement of normal sulci suggesting cerebral edema. The normal intracranial flow voids are absent and MRA shows no flow within the intracranial circulation. There is evidence of transtentorial herniation of medial temporal lobe and tonsillar herniation through Foramen Magnum with obvious compression of medulla.

In view of these findings on MRI in the given clinical setting, the patient was considered to be in a state of brain death.

Discussion

Acute organophosphate (OP) poisoning is one of the most common poisonings in emergency medicine. The pathophysiological basis for the clinical manifestations of OP poisoning is inactivation of the enzyme, acetylcholinesterase at the nerve terminals and junctions. Central nervous system is uncommonly involved in acute OP poisoning and occurs with OP compounds that cross the blood-brain barrier. In severe poisoning, patients may have convulsive seizures.[1]

In our patient respiratory failure leading to development of anoxic encephalopathy may have occurred due to respiratory muscle weakness, central respiratory depression, and pulmonary factors (pulmonary edema, bronchoconstriction, bronchorrhea). The sudden hypotensive event may have occurred due to herniation of cerebellar tonsils and brainstem compression.

Clinical diagnosis of brain death relies on the presence of unresponsive coma, no brain stem function, no respiratory reflex and supported by further tests including nuclear medicine studies, cerebral angiography, electroencephalography or Doppler ultrasound. MRI has been shown to be accurate in diagnosing brain death. Orrison et al have laid down criteria of brain death on MRI including transtentorial herniation, foramen magnum herniation, absent intracranial vascular flow voids, absent intracranial enhancement and hot nose sign on contrast MR angiography.[2] Karl-Olof et al have demonstrated the value of DWI in identifying the ischemic changes which are the end result of cerebral hypoperfusion due to compromised intracranial arterial flow.[3]

CONCLUSION

This case demonstrates all the classical findings associated

Volume : 5 | Issue : 7 | July 2015 | ISSN - 2249-555X

with non contrast MRI in brain death which includes the anatomic changes related to increased intracranial pressure and clearly demonstrates brain herniation associated with this condition and also depicts the physiological hypoxic changes seen as ischemic areas with bilateral distribution. It also goes on to demonstrate that the state of brain death is an ominous cascade and not a sudden event.

REFERENCE

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