CASE REPORT

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Acute organophosphate poisoning presenting with Cerebral Infarction: Association or Chance?

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ABSTRACT

Common neurological complications of acute organophosphorous poisoning are coma, seizure, and muscle weakness with respiratory failure which occurs due to cholinergic crisis. Report of cerebral infarction associated with acute organophosphorous poisoning has rarely been published. Here we report a case of 46 year old male patient admitted for alleged history of suicidal

organophosphorous poisoning presented with acute hemiplegia and his MRI brain revealed acute infarctions in left cerebral hemisphere.

Keywords: organophosphorous, poisoning, cerebral infarction, cholinergic crisis.

1. INTRODUCTION

Acute organophosphorous poisoning (OPP) is a common public health problem in India, especially in rural region because of financial debt. This poisoning is common because of easily availability of the compounds over the counters (Singh et al., 2000; Akbarizadeh et al. 2019). Organophosphorus compounds inhibits acetylcholineesterase and butyrylcholine esterase which leads to the accumulation of acetylcholine at synapses causing serious neurologic complications like coma, seizure, and muscle weakness with respiratory failure (Singh et al., 2000; Teke et al., 2004). Acute stroke, as cerebral infarction associated with acute OPP is extremely rare, and on literature search, only few cases has been reported (Jin-Hyuck et al., 2009).

2. CASE

A 46-year old middle aged farmer from rural Wardha district of Maharashtra was admitted to our hospital with the history of organophosphorous compound poisoning in suicidal attempt due to financial debt. Before brought at this hospital he was treated outside primary health centre with gastric lavage, atropine and pralidoxime for one day. There was no history of seizure. On asking history from his relative he was not having any history of hypertension, diabetes mellitus and ischemic heart disease. He was chronic alcoholic and had consumed alcohol on the day of poisoning. On admission to this hospital the patient was hemodynamically stable with pulse rate of 106 per minute, regular and blood pressure of 110/76 mm of Hg in right arm supine position. On evaluation the patient was febrile, drowsy with pinpoint pupil and bilateral crackles in the chest. Fasciculation were noticed over the thighs and arms. His cardiac examination was within normal limits. There were no respiratory crackles or wheeze on chest examination. He was not moving his right side upper and lower limbs.

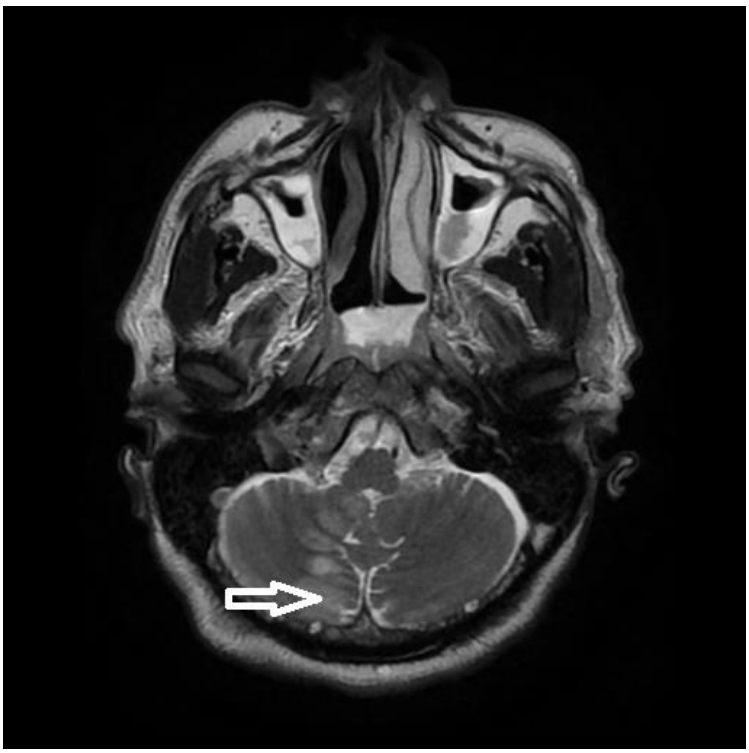

Figure 1 Brain MRI showing hypodensity lesion in left cerebral hemisphere (arrow).

On motor system examination his right sided deep tendon reflexes were brisk and same side planter reflex were extensor. Other brainstem reflexes were relatively well preserved. There was no seizure and other neurological complication during the hospital stay. His serum level of acetylcholine esterase was 948 IU/L (normal levels 4000-12000 IU/L), white blood cell count 13,600 /mm3, blood glucose level 228 mg/dL, glycosylated hemogloin level 5.6 mg%, and serum creatine kinase 72 IU/L (22-269 IU/L). His other parameter like liver function, renal functions, Electrocardiogram and lipid profile were within normal limits. His antidote therapy with atropine and pralidoxim were continued with that his cholinergic symptoms gradually improved. In view of neurological finding he was planned for brain magnetic resonant imaging (MRI) which revealed hypodensity lesion suggesting acute infarctions in left cerebral hemispheres (Fig.1).

His 2 D Echocardiocardiography was also normal, performed to look for any evidence of cardiac dysfunction as a source of thromboembolic phenomenon. His bilateral carotid Doppler was also normal. Patient was treated with intravenous mannitol, aspirin and statin in view of acute stroke. His acetylcholine esterase level came to 3498 IU/L on 4 th day and cholinergic symptoms disappeared. There was also sign of improvement in hemiplegia.

3. DISCUSSION

Central nervous system is uncommonly involved in acute OP poisoning and occurs with OP compounds that cross the blood-brain barrier. After acute intoxication with organophosphorous compounds, anticholinesterases lead to neurological syndromes in the form of acute cholinergic crisis, intermediate syndrome in which cranial nerve palsies, proximal muscle weakness and respiratory muscle weakness are common and delayed organophosphate induced polyneuropathy (Jin-Hyuck et al., 2009). Other neuro behavioural changes known as 'chronic organophosphate induced neuropsychiatric disorders', Parkinsonism, and cerebellar syndrome have been reported (Jin-Hyuck et al., 2009; Pilato et al., 2009).

Various authors has reported neuroimaging findings of acute stroke in the territory of vertebrobasilar system, hyperintense lesions on cerebellum and brainstem in T2-weighed MR image after OPP (Pilato et al., 2009; Karki et al., 2004). Ischemic stroke might be accompanied by co-morbidity of OP intoxication. Although cases of stroke have not been well studied in the critically ill patients, serious systemic illnesses may be risk factors for stroke like Coagulation disorder, infections, proinflammatory state and hyperglycemia (Pilato et al., 2009). This case had systemic illness due to OP poisoning such as fever, hyperglycemia and infection as his WBC count was raised.

Other mechanism may include a brief period of increased sympathetic tone and a prolonged period of parasympathetic activity which leads to vessel spasm and ischemia which is an important factor in pathogenesis of cardiac infarction (Karki et al., 2004; Kumar et al., 2014). Also, pesticides release increased the amount of catecholamines and other vasoactive amines (histamines and neutral proteases) that penetrate the collagen matrix of plaque causing erosions and rupture which can lead to ischemia. These inflammatory mediators can cause thrombosis, leading to cerebral stroke (Karki et al., 2004; Kumar et al., 2014). In our case this may be one of the pathogenesis. But the exact pathophysiology behind organophosphorus compound leading to these manifestations is not clearly understood. More studies are required to prove this hypothesis.

4. CONCLUSION

Patients with OP intoxication may present neurologically such as acute hemiplegia due to acute infarct, brain imaging should be considered in all these patients. Though over activity of cholinergic or nicotinic receptors, the increase in sympathetic and/or parasympathetic activity and acidosis have been hypothesized to play a role in the thrombosis, more research and case reporting are required.

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