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Case Report

Cerebral Stroke in Celphos Poisoning: A Rare Case Report

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I N F O

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A B S T R A C T

Acute aluminium phosphide poisoning is fatal. Till date no specific antidote has been found for it, as a consequence mortality, is extremely high. Celphos is aluminium phosphide (AlP), a solid chemical compound of fumigant nature. This compound is used as an agricultural pesticide and has been seen to be taken orally in suicidal cases. Neurological presentation is very common in celphos poisoning while the occurrence of cerebrovascular ischaemia is rare. Here, we have reported a case of ischaemic stroke as an initial manifestation of celphos poisoning on the third day of ingestion. Angiography conducted on the right middle cerebral artery revealed evidence of in-situ thrombosis. All other aetiologies of young ischaemic stroke were excluded.

Keywords: Celphos, Aluminium Phosphide, Suicidal Attempts, Ischaemic Stroke

Introduction

Aluminium phosphide (AlP), also known as celphos, or rice tablet, was first identified as a cause of poisoning in India in 1980.[1] Since then, it has been widely used among all agricultural pesticides.[1] The lack of a specific antidote has resulted in a high mortality rate. The primary mode of treatment is prompt decontamination and the implementation of resuscitation measures. A major concern regarding AlP is its potential for chemical terrorism due to the rapid release of lethal phosphine gas. Previously, this poison was easily available to people because of loose laws and legislations; but in the last few years, these laws and legislations have become stricter and have reduced its easy availability.[1] Even though suicidal rates are not declining; AlP is commercially available as 3 g tablets and 0.6 g pellets. The tablets are typically dark brown or grey in colour and contain two chemical compounds, aluminium phosphide and aluminium carbonate in a ratio of 56:44. The primary component consists of aluminium phosphide, with aluminium carbonate being incorporated to prevent the self-activation of phosphine (PH_3), which is caused by the reaction of aluminium phosphide with moisture.

$AlP + 3H_2O = Al(OH)_3 + PH_3$ (in air)

$AlP + 3HCl = AlCl_3 + PH_3$ (in air and stomach)

Every 3 g tablet releases 1 g of phosphine gas, while each 0.6 g pellet releases 0.2 g on exposure to moisture. A non-toxic, greyish, aluminium hydroxide residue is left in the pellet. Phosphine is a colourless, odourless, and poisonous gas. However, on exposure to air, a foul odour (garlicky or of decaying fish) is produced due to the formation of phosphines and diphosphines.

Inhalation of PH_3 is dangerous at 300 ppm and deadly at 400–600 ppm over 30 minutes. The lethal dose is usually 0.15 g to 0.5 g, but three or more tablets are always fatal.[2]

Aluminium phosphide poisoning can be confirmed by a Silver Nitrate Test. The presence of a foul odour coming from the oral cavity, as well as a highly variable arrhythmia in a young person who has experienced shock with no prior history of cardiac disease, suggests aluminium phosphide poisoning.

Aluminium phosphide ingestion can lead to a variety of toxic symptoms, typically developing within 30 minutes. Mild intoxication is characterised by irritation of the nasal mucosa, dizziness and headache, chest tightness, breathing difficulties, nausea, and vomiting. Moderate intoxication is characterised by diplopia and ataxia, as well as tremors.[2] In severe cases, acute respiratory distress syndrome (ARDS), cardiac arrhythmia (arrhythmias), convulsions, coma, and eventually death may occur. In some cases, systemic toxicity may occur, including liver and kidney failure.[2] Only a few reports of stroke as a characteristic feature of AlP intoxication are there in the literature. Here we have reported a case of ischaemic stroke in a young male on the third day of celphos ingestion.

Case Report

A 40-year-old man presented to the Emergency Department of King George's Medical University, Lucknow with chief complaints of ingestion of two tablets of celphos in the morning for suicidal attempt. There was no history of any psychiatric illness, chronic disease, or any other drug intake and addiction except tobacco chewing. There was no family history of cerebrovascular or cardiovascular diseases.

On general examination, the patient was found to be drowsy, with a Glasgow Coma Scale (GCS) of E4V5M6. The blood pressure was recorded as 84/50 mmHg, and the pulse rate was 86 per minute. The neurological examination revealed no sign of focal neurological deficit. Bilateral plantar reflexes were flexor. The cardiovascular system was normal with normal cardiac sounds. Bruits were absent on the cervical arteries. All other systems were normal on examination. The laboratory investigations showed that haemoglobin (Hb) was 14.8 g/dl, total leucocyte count (TLC) was 11900 cells/mm^3, platelet count (PLT) was 2.45 per mm^3, blood sugar was 122 mg/dl, sodium (Na) was 142.2 mmol/l, potassium (K) was 3.98 mmol/l, calcium (ionic) was 4.64 mg/dl, serum bilirubin was 4.14 mg/dl, Serum glutamic oxaloacetic transaminase (SGOT) was 54 U/L, Serum glutamate pyruvate transaminase (SGPT) was 140.1 U/L, Serum Alkaline Phosphate (SALP) was 192.7 U/L, prothrombin time was 20.9 seconds, International Normalised Ratio (INR) was 1.73, blood urea was 46.9 mg/dl, serum creatinine was 1.01 mg/dl, and viral markers were negative (Table 1). Arterial Blood Gas (ABG) showed metabolic acidosis with a pH of 6.696, HCO_3 of 4.3 mmol/l, pCO_2 of 35.7 mmHg, and lactate of 11.89 mmol/l. Troponin-T was 1.41 ng/ml. The lipid profile was within the normal range. On the third day of admission, the patient developed a sudden onset of hemiparesis on the left side of the body. Neurological examination revealed 2/5 power in the left upper limb and lower limb. Non-contrast computed tomography (NCCT) was done showing ischaemic lesions in the right middle cerebral artery (MCA) territory (Figure 1).

Brain MR angiography also revealed stenosis of the right MCA stem. Cervical MR angiography and colour Doppler sonography of cervical carotid and vertebral arteries did not reveal any abnormality.

Cardiac investigations including chest radiography, electrocardiogram and transthoracic echocardiography were normal. The laboratory investigations showed normal values of protein C, protein S, antithrombin, fibrinogen, homocysteine, and factor VIII. Infectious or immunological disorders were ruled out by negative viral markers, collagen vascular diseases and absence of hypercoagulable states which are common causes of young age stroke. The psychiatric opinion revealed borderline-type personality disorder.

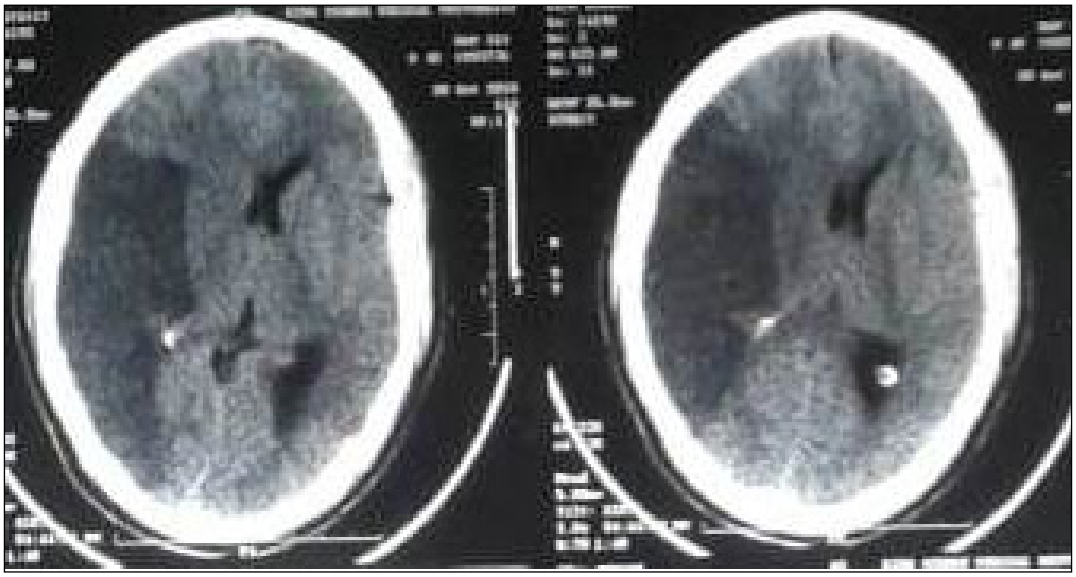

Figure 1.Right Sided MCA territory infarct in NCCT head of Patient

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Table 1.Laboratory Data on Day One

Parameters	Values
WBC count (per mm^3)	11900
Haemoglobin (g/dl)	14.8
Platelets (per mm^3)	2.45
Serum calcium ionic (mg/dl)	4.64
Serum magnesium (mg/dl)	2.44
Blood glucose level (mg/dl)	122
Triglyceride (mg/dl)	98
Cholesterol (mg/dl)	122
HDL (mg/dl)	45
LDL (mg/dl)	55
Erythrocyte sedimentation rate (mm/h)	10
Partial thromboplastin time (s)	15
Prothrombin time (s)	20.9
Alanine aminotransferase (ALT) (U/L)	140.1
Aspartate aminotransferase (AST) (U/L)	54
Urea (mg/dl)	46.9
Creatinine (mg/dl)	1.01
Creatine kinase (CK) (U/L)	102
ECG	Normal
CXR	Normal

WBC: White blood Cells, HDL: High-density Lipoprotein, LDL: Low-density Lipoprotein, ECG: Electrocardiograph, CXR: Chest X-Ray

Treatment

Gastric lavage with potassium permanganate ($KMNO_4$) 1:1000 solution was done meticulously to remove/oxidise unabsorbed AlP poisoning. Fifty grams of activated charcoal were given to absorb PH_3 gas. Oral antacid and an intravenous H2 blocker were given to relieve gastrointestinal manifestations of PH_3. Magnesium sulphate was given to increase its excretion through the gut. Two to three litres of normal saline was administered within the first 8–12 h and after that injection dopamine (4–6 µg/kg/min) was given to raise the systolic blood pressure to greater than 90 mm Hg. After the development and confirmation of ischaemic infarct by the NCCT Head, antiplatelet therapy (aspirin 80 mg daily) was also started along with atorvastatin and enoxaparin. The patient was conscious and oriented with a mild headache and no sign of slurring of speech or dysarthria. Hemiparesis of the left side was persistent. No further complications were there and all his abnormal blood parameters got normalised. The patient was discharged on the tenth day with a National Institutes of Health Stroke Scale (NIHSS) score of 13 and a modified Rankin scale score of 3.

Discussion

In India, AlP is available in tablet and powder forms. Multiple organ dysfunction syndrome is a fatal complication of acute AlP poisoning. Free radicals produced by phosphine gas cause damage to the heart, brain, lungs, kidneys and liver because these organs use higher quantities of oxygen.[3] As a result, nervous system injury is a well-known complication.[4] Long-term neurological disability is very rare in such cases. Brautbar and Howard also reported a case of weakness and loss of sensation in the left side of the body after ingestion of AlP.[5] Kurzbauer and Kiesler also reported a case with neurological dysfunction like left Rossolimo reflex and bilateral plantar extensor.[6] A similar report was also described by Dave et al. with delayed haemorrhagic stroke after accidental AlP ingestion.[7] Superoxide and peroxide radicals are produced in the presence of AlP, with subsequent injury of cells by peroxidation of lipids.[8] Petechial haemorrhages were found at the brain surface on biochemical and histopathological results of postmortem reports[9] along with congestion and coagulative necrosis of neurons in the brain[10]. Now it can be assumed that injury to vessels of the brain resulted in active thrombus formation

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and led to stenosis and ischaemic stroke development in MCA of our patient. The later occurrence of hemiparesis was not a part of the early complication. In other studies, patients with many complications did not have any episode of cerebral infarct. Thus we might assume that the development of ischaemic stroke is a delayed complication of aluminium phosphide intoxication in our patient.

Conclusion

Acute aluminium phosphide (APS) poisoning is highly fatal due to the lack of an effective antidote. Its management is based on prompt decontamination and the implementation of resuscitation measures. The mortality rate has decreased over the past few decades due to the implementation of intensive care, stringent pesticide regulation, restriction of the availability of the poison, recognition of its toxicity, and the provision of improved medical care and intensive care after consultation with a regional or national poison centre. This case report aims to identify ischaemic stroke as one of the rare presentations of aluminium phosphide poisoning affecting its management by increasing morbidity and mortality of the patient.

Conflict of Interest: None

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