Ischemic Stroke as a Rare Manifestation of Aluminum Phosphide Poisoning: a Case Report

Mahmoud Abedini[1], Farzad Fatehi[2], and Nasim Tabrizi[1]

[1] *Department of Neurology, School of Medicine, Mazandaran University of Medical Sciences, Sari, Iran*

[2] *Department of Neurology, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran*

Received: 14 May 2013; Accepted: 21 Mar. 2014

Abstract- Aluminum phosphide (AlP) is a solid fumigant which is widely used for a suicide attempt in Iran. Although neurologic symptoms are commonly reported, cerebrovascular stenosis is rare in AlP poisoning. We described ischemic stroke as a delayed complication of AlP intoxication. A 30-year-old man was admitted because of sudden onset left side hemiplegia, 11 days after intentional ingestion of three rice tablets. Investigations revealed in situ thrombosis in right middle cerebral artery (MCA) while other causes of stroke in young adults were excluded. Ischemic stroke should be considered as a delayed complication of AlP intoxication even after the acute phase of intoxication.

Acta Medica Iranica, 2014;52(12):947-949.

Keywords: Aluminium phosphide; Poisoning; Intoxication; Stroke

Introduction

Aluminum phosphide (AlP) also called rice tablet is an extensively used solid fumigant. Easy availability, low-cost and famous potential fatality has made it a suitable agent for a suicide attempt in a few countries like Iran (1).

Symptoms related to nervous system injury are commonly reported in acute AlP intoxication including headache, dizziness, diplopia, paraesthesia, agitation, ataxia, altered mental state, intention tremor, convulsion and hypoxic encephalopathy (2).

A few reports of stroke as a delayed feature of AlP intoxication present in the literature. We reported a case of ischemic stroke in the recovery phase of AlP intoxication.

Case Report

A 30-year-old right-handed man was admitted to the emergency department (ED) of Buali Sina hospital four hours after a sudden onset left side hemiplegia. He had a suicide attempt just 11 days before by ingestion of 3 rice tablets and was discharged after hospitalization for 10 days, without any neurologic sign. At the day 11 after a short nap, he suddenly experienced left side hemiplegia and dysarthria. He had no specific medical history and also he was not alcohol or drug abuser, but he smoked 2.5 pack/year. Family history was negative for cerebrovascular or cardiovascular diseases.

Physical examination

On initial presentation, he was lethargic and febrile (oral temperature: 38.4°C). Neurologic examination revealed left hemifacial paresis and left hemiplegia. Left plantar reflex was also unobtainable. The national institute of health stroke scale (NIHSS) was assessed to be 16.

On systemic examination, no other abnormality was detected. Cardiac sounds were normal, and no bruit was heard on cervical arteries.

More investigations

The results of initial medical check-up in ED are reported in table 1.

At the first brain computerized tomography (CT) scan no abnormality was detected. The patient underwent brain magnetic resonance (MR) imaging that showed ischemic lesions in right middle cerebral artery (MCA) territory (Figure 1). Brain MR angiography also disclosed stenosis of right MCA stem (Figure 2).

Investigation of cervical carotid and vertebral arteries including cervical MR angiography and color Doppler sonography did not reveal any abnormality.

Corresponding Author: N. Tabrizi
Department of Neurology, School of Medicine, Mazandaran University of Medical Sciences, Sari, Iran
Tel: +98 911 1263538-, Fax: +98 11 33343017, E-mail address: n.tabrizi@ymail.com

Cardiac investigations showed normal chest radiography and a junctional rhythm in electrocardiogram. The results of transthoracic echocardiography demonstrated normal left ventricular ejection fraction and mild mitral and tricuspid regurgitation and transesophagial echocardiography showed no mass, vegetation or any other abnormalities.

Laboratory work-up for hematologic disorders (protein C, S and antithrombin deficiency, activated protein C resistance, dysfibrinogenemia, hyperhomocysteinemia and elevated factor VIII) showed no significant abnormality. Other causes of stroke in young adults, such as infectious or immunological disorders, were also excluded on the basis of tests for viral markers, collagen vascular diseases and hypercoagulable state inducing factors.

The psychology consult was requested for the patient because of recent suicide attempt that suggested borderline type personality disorder and recommended more interview sessions and psychotherapy after discharge.

Table 1. Laboratory data at ED

Parameter	On admission
WBC Count (per mm^3)	5000
Hemoglobin (g/dl)	13.8
Platelet Count (per mm^3)	103000
Serum Calcium (mg/dl)	9.8
Serum Magnesium (mg/dl)	2.4
Blood glucose level (mg/ml)	92
Triglyceride (mg/dl)	83
Cholesterol (mg/dl)	125
HDL	22
LDL	81
Erythrocyte sedimentation rate (mm/h)	15
Partial thromboplastin time (s)	30
Prothrombin time (s)	13
Alanine aminotransferase (ALT) (U/L)	220
Aspartate aminotransferase (AST) (U/L)	58
Urea (mg/dl)	37
Serum creatinin (mg/dl)	1
Creatine kinase (CK) (mU/ml)	1056

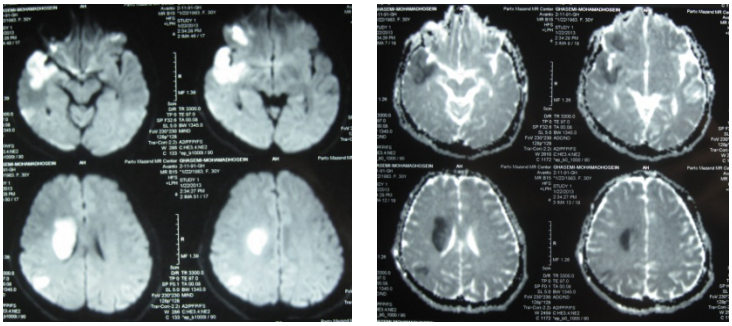

Figure 1. Brain MRI 24 hours after event shows ischemic lesion in right MCA territory

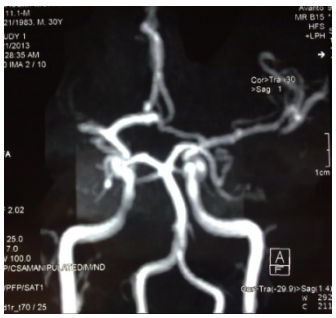

Figure 2. Brain MRA shows a stenosis in right MCA stem

Treatment

Antibiotic therapy was initiated at the first day because of fever and pyuria in urine analysis. Antiplatelet therapy (aspirin 80 mg daily) was also initiated when no evidence of hemorrhage was detected in the brain CT scan. He was also treated by atorvastatin and enoxaparin in prophylactic dose.

Course and prognosis

At the second day after antibiotic therapy, fever was discontinued, and patient's general condition improved. He was alert with no dysartheria and just complained of mild generalized headache. His motor deficit was persistent. He experienced no complication during hospitalization and abnormal blood tests including liver function tests; creatine kinase and cell blood count improved significantly. He was discharged at day 11 with NIHSS 13 and modified Rankin scale score 3.

Re-evaluation after a month showed improvement in left side hemiparesis to grade three of medical research council scale for grading muscle strength with no other significant changes in patient's state.

Discussion

AlP is available in Iran as 3 g tablets. Each tablet

contains 56% AlP and 44% of ammonium carbonate. Acute AlP poisoning leads to multi-organ involvement and variable clinical features. Phosphine produces free radicals in several tissues therefore; organs that need more oxygen like heart, brain, lungs, kidneys and liver have more susceptibility to AlP damages (3). Thus, nervous system involvement is a known complication of AlP intoxication. The most striking manifestations are almost always because of central nervous system (CNS) injury while peripheral and autonomic nervous systems are hypothetically or clinically involved (4).

Other than commonly reported nervous system manifestations, there are just a few reports of long-term neurological disabilities. Brautbar and Howard (5) described a patient with weakness and loss of sensation in the left-side extremities following AlP intoxication. In another report, Kurzbauer and Kiesler (6) described a similar patient with neurological abnormalities, such as left Rossolimo reflex and bilateral Babinski. There is also another report of delayed hemorrhagic stroke following accidental AlP ingestion by Dave and colleagues (7).

In the presence of AlP, cellular superoxide and peroxide radicals are generated, with subsequent cellular damage by lipid peroxidation (8). Biochemical and histopathological findings in postmortem cases reveal petechial haemorrhages on the surface of the brain (9) and changes such as congestion and coagulative necrosis in brain tissue (10). It seems that a similar mechanism causes injury in cerebral vessels and activates thrombosis the formation cascade which leads to vascular stenosis and ischemic stroke as in situ thrombosis development in MCA stem in current patient.

It seems that the amount of systemic complications could not predict delayed cerebrovascular injury as in current patient the event happened in recovery period when he was asymptomatic and the laboratory tests were improving however, in other studies, patients with more dramatic symptoms and complications did not experience cerebral infarct.

Ischemic stroke should be considered as a delayed complication of aluminium phosphide intoxication even after the acute phase of intoxication.

References

1. Wahab A, Zaheer M, Wahab S, et al. Acute aluminium phosphide poisoning: an update. Hong Kong J Emerg Med 2008;15(3):152-5.
2. Moghadamnia AA. An update on toxicology of aluminum phosphide. Daru 2012;20(1):25.
3. Moazezi Z, Abedi SH. A successful management of aluminium phosphide intoxication. Caspian J Intern Med 2011;2(3):286-8.
4. Nath, NS, Bhattacharya I, Tuck AG, et al. Mechanisms of phosphine toxicity. J Toxicol 2011;2010:494168.
5. Brautbar N, Howard J. Phosphine toxicity: report of two cases and review of the literature. Toxicol Ind Health 2002;18(2):71-5.
6. Kurzbauer H, Kiesler A. Zawodowe zatrucie fosforowodorem [Occupational phosphine poisoning, in Polish]. Neurol Neurochir Pol 1987;21:415-7.
7. Dave HH, Dave TH, Rakholia VG, et al. Delayed haemorrhagic stroke following accidental aluminium phosphide ingestion. J Assoc Physicians India 1994;42(1):78-9.
8. Almasieh M, Lieven CJ, Levin LA, et al. A cell-permeable phosphineborane complex delays retinal ganglion cell death after axonal injury through activation of the pro-survival extracellular signal-regulated kinases 1/2 pathway. J Neurochem 2011;118(6):1075-86.
9. Anger F, Paysant F, Brousse F, et al. Fatal aluminum phosphide poisoning. J Anal Toxicol 2000;24(2):90-2.
10. Sinnha US, Kapoor AK, Singh AK, et al. Histopathological changes in cases of aluminium phosphide poinsoning. Indian J Pathol Microbiol 2005;48(2):177-80.