Aconite Poisoning: A Case Report

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ABSTRACT

Aconite is the well-known toxic plant as well as valuable drug since decades. Most of the intoxications are accidental whereas suicidal attempt is rather rare after ingestion. This case report is about two patients who landed in our emergency department after consuming Aconite accidentally as an herbal medicine and were managed immidiatley.

Keywords: Accidental; Aconite; Poisoning.

INTRODUCTION

Aconite is a well-known toxic plant belonging to the Family Ranunculaceae with over 250 species which has been used in Eastern and Western therapeutics as well as a poison for centuries.[1,2] It is commonly known as Monk's hood, mitha zeher and contains aconitine and other C19- diterpenoid alkaloids, which are known neurotoxins and cardiotoxins.[2,3] Aconitine alkaloids are particularly present in the roots and tubers of the plants and have antipyretic, analgesic, cardio-tonic and local anesthetic effects. But they are highly toxic and has a narrow margin of safety between a therapeutic and toxic dose. The lethal dose of aconitine for human is estimated to be between 1 mg and 5 mg and mostly the intoxications are accidental whereas suicidal attempt is rather rare after ingestion of plant tubers.4,5 In Nepal, it is often confused with a plant of similar morphology called Nirmasi (Delphinium denudatum).[6]

In this article, we report two cases of aconite poisoning from the same family who took an herbal medication given by a local vendor for joint pain which was later identified as Aconite.

CASE REPORT

Case 1 : 50 years old hypertensive lady under medication presented in the emergency department after 9 hours of ingestion of herbal plant. She took it along with homemade liquor about 100ml. After an hour of ingestion, she began to develop altered sensorium and abnormal body movement characterized by stiffening of body and frothing from mouth which lasted for 15 minutes. She also gave history of tingling sensation of bilateral hands and feet and circumoral area along with twitching of muscles of hand and feet, followed by difficulty in speaking and swallowing and multiple episodes of vomiting. On presentation in the emergency department, the patient was restless and irritable. Her blood pressure and pulse were not recordable with GCS: E1V1M4. The patient was given DC shock 150 J for three cycles and injection Amiodarone (loading dose and maintenance dose). But the patient continued to have multiple Ventricular Premature Complexes (VPCs). Arterial Blood Gas(ABG) analysis done at the time, showed blood pH of 7.42, PaO2 of 115 mm of Hg, PCO2 of 9.8 mm Hg, HCO3 6.2 mmol/L and lactate of 10.7 mmol/L. After 2 hours of treatment in ER, her blood pressure was recorded 110/90mmHg but

ECG revealed wide complex tachycardia with pulse of 200bpm which was later controlled with lignocaine. The patient was subsequently managed in the ICU with intravenous fluid with multivitamins, inj MgSo4, inj calcium gluconate 10%, antihypertensive and antiemetic. She had occasional VPCs which disappeared after 24 hours of observation in ICU and finally reverted to sinus rhythm. In spite of recovering well, the patient left against medical advice (LAMA) due to financial conditions on the second day. She was discharged with antihypertensive, anti-arrhythmic, beta-blocker and multivitamins.

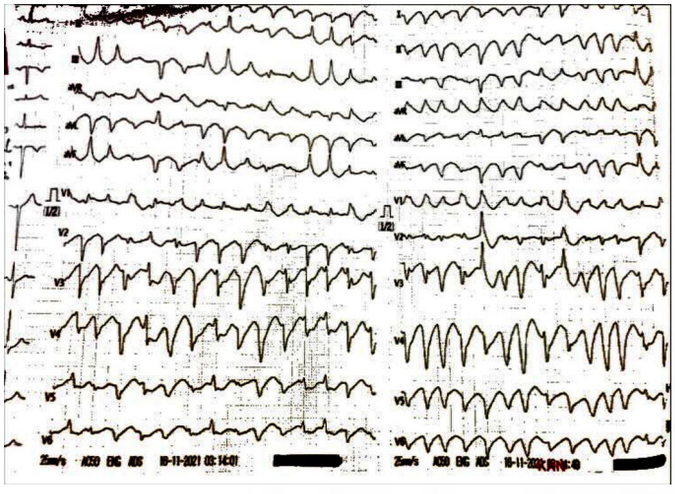

Figure 1: Electrocardiogram of Case 1 showing multiple Ventricular Premature Complexes (VPCs) with wide complex tachycardia

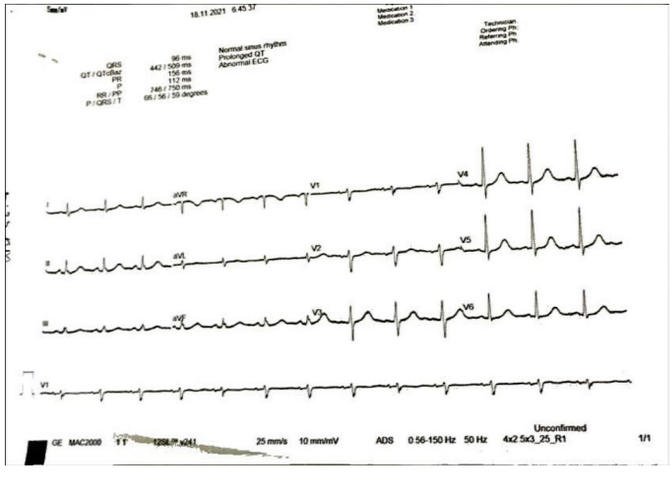

Figure 2: Electrocardiogram of Case 1 reverted to sinus rhythm after management

CASE REPORT 2

39 years old female also consumed the same herb and had history of dizziness followed by vomiting, tingling sensation of foot, hand, face and weakness of limbs. On examination, she was anxious and ill looking, and had increased pulse rate and respiratory rate of 118 bpm and 28 per minute respectively. Blood pressure was within the normal limit. ABG parameters were normal. ECG showed atrial fibrillations and premature ventricular contractions. She was managed with injection atropine 1mg stat and inj amiodarone (loading and maintenance dose) along with the fluid. Later Inj 10% calcium gluconate and beta blocker was also given. She was also recovering well but went on LAMA(Left Against Medical Advice) the next day with anti-arrhythmic drugs and multivitamins.

DISCUSSION

Aconite, a fast acting toxin, are the herbaceous perennial plants growing in the temperate to alpine zones.[2] It is well known to the ancients as a powerful poison as well as traditional medicine. It is commonly known as bikh or Ekfale in Nepal and Monk's hood or aconite in English. It is widely distributed from the east to west of Nepal. Taxonomical studies reported 38 Aconitum species here in Nepal.[2] The level of aconitines in roots is about 10 times that in stems and about 300 times that in leaves.[6] Fujita Y et.al have reported the lower alkaloid content of leaves as compared to roots in their study.[5] While aconites are rarely used in modern medicine due to its toxicity, it is still being used as a traditional as well as a culinary herb in homeopathy to decrease fever, painful joints, as cardiac depressant, to treat neuralgia and also applied as in liniments as rubifacient for external application. In Nepal, people often use it as a health supplement to enhance appetite.[2,8]

Due to relatively fewer studies, precise toxic and lethal doses of aconitine are not well defined. It may vary individually depending on factors such as species and part ingested, inadequate decoction preparation, additional consumption of other drugs or alcohol or metabolic disorders (liver and kidney failure)[4,9] as per our case too. The toxic alkaloids in aconite roots dissolve in alcohol efficiently. The half-life of Aconitine has been reported to be about 3 hours but intoxication symptoms may persist for 30 hours.[10] The onset of symptoms occurs rapidly, within 10 to 20 minutes. Skin paresthesia, followed by numbness and unexplained ventricular tachycardia, with the history of ingestion of herbal medications alerts clinicians to the possibility of aconite poisoning which are relevant with our case. S. Tak et.al in his study mentioned that the patient decided to self-medicate with ''Bachnaag'' (Aconite) root for residual hemiparesis after reading a book of traditional/Ayurveda medicine. Patient became symptomatic within 1 hour of taking about five gram of dried root of aconite plant.[7]

Toxic symptoms include a tingling or burning sensation in the fingers and toes at first, followed by sweats and chills, generalized paresthesia, feeling of dryness in the mouth and circumoral numbness. Later on, violent vomiting, colicky diarrhea, skeletal muscle paralysis, cardiac rhythm

disturbances and intense pain develop.[3] All our three cases developed symptoms within an hour.

The main causes of death in aconite poisoning are cardiovascular collapse and ventricular arrhythmias.[8] If higher concentrations are present, respiratory paralysis, supra ventricular tachycardia, ventricular tachycardia, torsade's de pointes and other conduction disturbances may be seen.[1,6] Dwivedi S et.al in his series of studies concluded that the main ECG findings in the person who died due to Aconite poisoning were ventricular premature beats and ventricular tachycardia.[11] Management of aconite poisoning needs immediate attention to the vital functions and close monitoring of blood pressure and cardiac rhythm.[6]

In Nepal as an ayurvedic treatment, the aconite toxicity is managed by giving ghee with mixture of turmeric juice, or cow milk, a strong coffee or tea or tannic acid to precipitate the alkaloid.[2] In some cases, species of Aconite, A. oreochryseum is also used as the antidote.[12]Neermashi or its mixture with root of Asparagus are recommended as a preventive measure of aconite poisoning in Annapurna region of Nepal.[13] Activated charcoal is advisable if the patient presents within one hour.[3] However, Chan CP et.al mentioned that three cases of aconite poisoning in Nepalese family were managed with activated charcoal in Hong Kong after three-and-a-half hours of ingestion despite the delay whereas our case also arrived very late after nine hours thus charcoal was not used.[6] Patient must be managed in the ICU or cardiac care units.[2]Inotropic therapy is required if hypotension persists and atropine should be used to treat bradycardia. Aconite-induced ventricular arrhythmias are often refractory to direct current cardioversion and antiarrhythmic drugs.[6] With no specific antidote, we rely on case studies which suggests that amiodarone and flecainide are reasonable first-line treatment.

Magnesium is sometimes effective for polymorphic ventricular tachycardia.[14] Ventricular tachyarrhythmia following this intoxication are rarely responsive to lidocaine.[15], unlike in our case. Consistent with our study, similar finding was also noted by Paudel R et.al in which four cases of wide complex tachycardia in aconite poisoning was controlled with lignocaine in Manipal teaching hospital.[1] In a recent review, Coulson et al describe the management of 65 cases of probable aconitine poisoning resulting in ventricular dysrhythmias, reporting that flecainide or amiodarone seems to be more associated with a return to sinus rhythm than lidocaine and/or cardioversion. Moreover, mexiletine or procainamide may be used to manage tachyarrhythmias caused by aconite poisoning.[10,15]

CONCLUSION

Even though aconite has long been used as one of the effective herbs in traditional medicines, misidentification has resulted in most of the poisoning. Upon occurrence of the poisoning, the patient should be provided intensive care treatment with continuous monitoring of blood pressure and cardiac rhythm through ECG. Health policy-making authorities and the local community should be made aware and alerted about the adverse effects of such herbal medications consumption without prior consultation.

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