JOURNAL OF CLINICAL AND DIAGNOSTIC RESEARCH

How to cite this article :
PAUDEL R, PALAIAN S, RAVI SHANKAR P, PAUDEL B, , BHATTARAI S. ACONITE POISONING: A CLINICAL REVIEW OF THE FIRST FOUR CASES FROM NEPAL Journal of Clinical and Diagnostic Research [serial online] 2008 February [cited: 2008 February 4]; 2:651-655.
Available from
http://www.jcdr.net/back_issues.asp?issn=0973-709x&year=2008&month= February &volume=2&issue=1&page=651-655 &id=137

CASE REPORT

Aconite Poisoning: A Clinical Review Of The First Four Cases From Nepal

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ABSTRACT

Aconite tubers are one of the most toxic plant products. They are taken as medication, and are occasionally confused with some other similar plants which are commonly used as medications. This manuscript describes four cases of aconite poisoning that were managed in the Manipal Teaching Hospital. The patients presented with classical symptoms of aconite poisoning such as perioral paraesthesia, generalized burning sensation, and cardiac manifestations such as hypotension and ventricular tachycardia. Since Aconite is a dangerous life threatening herb, one should be aware of this poison. We report four patients who presented with aconite poisoning after mistakenly ingesting aconite, thinking it to be related to medicinal plants.

Keywords: Aconite poisoning, Cardiotoxicity, Nepal

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Introduction

Aconite tubers are among the most toxic plants known, but have been used in Eastern and Western therapeutics for centuries.[1] There are over 600 reported cases of poisoning in China alone, and in Hong Kong it is estimated that 75% of Chinese herbal medicines related hospital admissions are related to aconite toxicity.[1] In Western countries, aconite poisoning usually occurs after ingesting the wild aconitum plant.[2] Evidence from Hong Kong suggests that the number of hospitalisations due to aconitine poisoning markedly decreased from four to six per year in 1989 to 1991, to one to two per year in 1992 to 1993, following publicity measures to promote awareness among the herbalists and the public. The annual incidence of aconitine poisoning showed a marked decrease from 0.49 to 0.69 to 0.10 to 0.22 per 100 000 population.[3] We could not locate the reported cases of Aconite poisoning from Nepal. In Nepal, it is often confused with another plant [Nirmasi (Delphinium denudatum)], that resembles it. In this article, we provide the details of four cases of Aconite poisoning that were managed in our hospital. We have also put forward an approach towards the management of Aconite poisoning.

Case 1

A 76 year old male was brought to the hospital, four hours after ingestion of the herbal plant 'Ekfale', mistaking it to be the medicinal plant 'Nirmasi'. The patient presented with multiple episodes of vomiting, generalized tingling, a burning sensation and severe restlessness. On presentation in the emergency department, the patient was conscious but restless, cyanosed with oxygen saturation of 80%, with feeble rapid pulse, and blood pressure was not recordable. ECG showed wide complex tachycardia [Table/Fig 1]. The patient was given DC shock 200 J, 360 J and 360 J. Ventricular tachycardia was reverted, but the patient continued to have multiple Ventricular Premature Complexes (VPCs) [Table/Fig 2]. The

patient was started on injection Amiadarone (loading and maintenance dose), after which VPCs were controlled, but the patient had persistent low BP and hypoxia. Arterial Blood Gas analysis done at the time, showed blood pH of 7.225, PaO_2 of 58 mm of Hg, $P_{CO\ 2}$ of 55 mm Hg, $SP_{O\ 2}$ of 84.8%, and HCO_3 22.4 m Eq/L. The patient was intubated and kept on ventilator with inotropic support, with Dopamine/Dobutamine, but BP did not increase despite escalating doses of noradrenaline and adequate central venous pressure being maintained at 10- 12 cm H_2O. The patient was having recurrent episodes of ventricular tachycardia which reverted after DC shock, but finally torsade-de-pointes was seen, which was refractory to DC shock and antiarrythmics including Inj. Magnesium sulphate. The patient succumbed after 24 hours of active management in the ICU.

Case 2

A 40 year old male, after few minutes of intake of 'Ekfale' for abdominal cramps, developed numbness around the mouth and tongue, and experienced tightness of the lower limbs and buttocks, resulting in an inability to walk. There was no history of loss of consciousness, seizure, or palpitation. On examination, the Glasgow Coma Scale was 15/15 with maintained BP and other vital signs. Gastric lavage was done, and the patient was kept in the ICU for observation, with supportive management. ECG did not show major changes during the hospital stay. He was discharged after 48 hours of observation in the ICU.

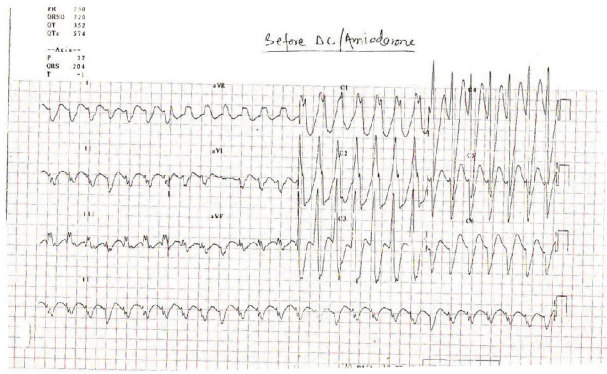
Case 3

A 60 year old male took 'Ekfale', mistaking it to be a herbal medicine for fever and throat pain. Soon after ingestion, the patient developed perioral numbness, generalized tingling sensation, and excessive vomiting. On presentation in the emergency department, the BP was 80/50, which increased after fluid resuscitation, but ECG revealed wide complex tachycardia [Table/Fig 3], which was controlled with lignocaine. The patient was subsequently managed in the ICU with supportive measures and antiarrythmics, and did not develop fatal arrhythmias. He had occasional VPCs which disappeared after 24 hours of observation in ICU, and finally reverted to sinus rhythm [Table/Fig 4]. He had persistent perioral numbness and an excessive nauseated feeling, which persisted till the fourth day of admission, and gradually subsided. The patient was discharged after 6 days of hospital stay.

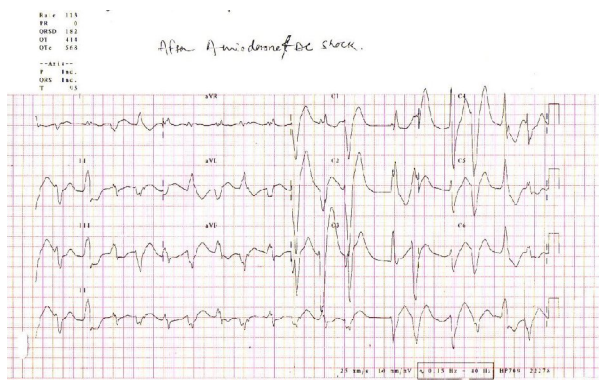
Case 4:

A 45 year male who was brought along with the third patient, who actually tasted the herbal plant to identify whether it was really the medicinal herbal "Nirmasi" or the poisonous "Ekfale" as his friend, developed untoward symptoms. Soon after tasting the scrapings of the plant root, he also developed perioral numbness and an excessively nauseating feeling .When brought to hospital after 2 hours of ingestion, he was cold and clammy, with a palpatory systolic blood pressure of 60 mmHg. He was resuscitated with fluids, after which, the blood pressure increased to 120/84 mmHg. ECG showed junctional rhythm, which reverted to normal after 12 hours of observation in the ICU.

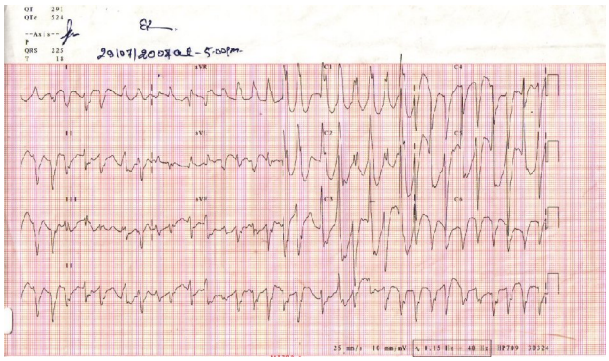
Table/Fig 1: Electrocardiogram of Case 1 showing wide complex tachycardia



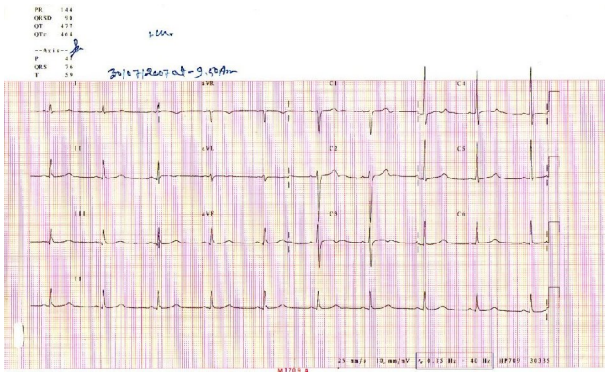
Table/Fig 2: Electrocardiogram of Case 1 after DC shock and Amiodarone infusion showing reverted wide complex tachycardia but persisting VPCs



Table/Fig 3: Electrocardiogram of Case 3 showing wide complex tachycardia



Table/Fig 4: Electrocardiogram of Case 3 after lignocaine infusion, reverted to sinus rhythm



Discussion

Background:

Aconite is well known, because it is extremely toxic. The tuberous root has been used in traditional medicine, although all parts of the plant are considered to be toxic. While the

extracts of the plant are rarely used in modern medicine today, they continue to find use in liniments as rubifacients, for external application. Extracts of the plant are used in homeopathic and traditional medicine like hypotensives, to decrease fever, as cardiac depressants, and to treat neuralgia.[4] Herbal decoctions of aconite are generally prepared by soaking the roots in water or saturated lime water, and then boiling. This causes hydrolysis of aconite alkaloids to less toxic benzylaconine and aconine derivatives. Many variables can affect the concentration of the processed aconite root (e.g., species, place of harvest, adequacy of processing), therefore, poisoning may occur following the consumption of processed roots. [5]

Chemical composition of aconite:

Aconite is a fast-acting toxin. The active principles are aconitine and related alkaloids. Alkaloids account for up to 1.5% of the dry weight of the plant. These consist primarily of the related alkaloids aconitine, picraconitine, aconine, and napelline.[6] Aconitine is hydrolyzed to picraconitine, which hydrolyzes to aconine. Several minor alkaloids have been isolated from the various species of aconite. These minor alkaloids include sinomontanitines, lappaconitine, ranaconitine etc. [7]

Symptoms:

The onset of symptoms occurs rapidly, within 10 to 20 minutes. A tingling or burning sensation in the fingers and toes is usually seen first, followed by sweats and chills, a generalized paresthesia, a feeling of roughness and dryness in the mouth, numbness, and a feeling of intense cold. Later there is violent vomiting, colicky diarrhoea, skeletal muscle paralysis, cardiac rhythm disturbances, and intense pain.[7] Skin paresthesia, followed by numbness in the setting of ingestion of herbal medications, can raise the possibility of aconite poisoning. All our patients had such a presentation. This presentation was one of the main reasons for us to diagnose the poison. The combinations of symptoms (like parasthesia) and signs (arrhythmias) in the setting of ingestion of herbal medications suggest aconite poisoning. The main causes of death in aconite poisoning, are cardiovascular collapse and ventricular arrhythmias. [1]

Complications

In our patients, we mainly found cardiac complications. In general, vagal slowing is seen in 10 to 20% of fatal intoxications. If higher concentrations are present, supraventricular tachycardia, ventricular tachycardia, torsades de pointes, and other conduction disturbances may be seen. Ventricular fibrillation may be seen, and is often the cause of death. Patients may have a severe oppressive feeling in the chest. Hypotension may be present. In one of our patients (case 1) torsade de pointes was seen, and in all of them hypotension was a common finding.

Approach towards management:

In general, the onset of symptoms is rapid, and prognosis after ingestion is generally poor. There is no specific antidote. Treatment is symptomatic and supportive after decontamination. There were

also reports in which refractory arrhythmias were being successfully managed with percutaneous cardiopulmonary support system and cardiopulmonary bypass. [8] We put forward an approach for managing the patients with aconite poisoning.[1, 9]

- Symptoms begin within one hour after ingestion. Ipecac-induced emesis is not recommended because of the potential for cardiovascular instability and seizures

- If patients are seen with in 1 to 2 hours, gastric lavage may be attempted. However, the hazards of gastric lavage need to be kept in mind, especially in countries with limited resources for performing the procedure safely. [10]

- Serum levels of aconites are of no value.

- Aggressive supportive care is the mainstay of therapy.

- In general, activated charcoal could be considered in patients who present early. In patients who are unable to protect their airway, charcoal should not be administered, given the risk of charcoal aspiration and pneumonia, unless the airway is protected. Even in intubated patients, the risk of aspiration is still there at 4%. [11] If used, administer charcoal as slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years). One should consider risk versus benefit, prior to charcoal administration.

- Respiratory support should be available, since paralysis of respiratory muscles may occur.

- Extensive vomiting and diarrhoea may require that fluid and electrolytes be monitored and replaced as necessary.

- Dysrhythmias are relatively refractory to drug management. A number of different agents have been tried, with varying levels of success. Some of the agents tried are atropine, quinidine, dichloroisoproterenol, digitalis, parasympathomimetics, and inorganic ions for different types of arrythmias. Amiadarone and magnesium sulphate are the preferred drugs in management of ventricular tachycardia and torse de pointes, respectively.

- Hypotension: Measure the central venous pressure by keeping central venous line, and if it is less than 10 cm of H_2O, administer 10 to 20 mL/kg isotonic fluid bolus. If hypotension persists despite normal central venous pressure, administer inotropes like dopamine (5 to 20 mcg/kg/min) or norepinephrine (Adult: begin infusion at 0.5 to 1 mcg/min; Child: begin infusion at 0.1 mcg/kg/min); titrate to desired response.

Relevance to Nepal:

Aconitum ferox is the species found in Nepal, India, and the Himalaya Mountains. An extract of this plant has been used as an antipyretic in Ayurvedic medicine, after "detoxification".[12] We could not find literature regarding the therapeutic use of Aconite in Nepal. In our cases, the patients took this medicine, confusing it with another plant (Nirmasi) that resembles this plant.

Conclusion

These case reports may be just the tip of the iceberg. There can be cases which go unnoticed, and many persons might have died due to undetected aconite poisoning, after either taking it raw, or as herbal medications. Since Aconite is a dangerous life threatening herb that causes poisoning, one should be aware of this poison. We report four patients who presented with aconite poisoning after ingesting it mistaking it to be a related medicinal plant. The quick onset of action and the difficulty in accessing health care because of the difficult terrain, can make it an important cause of mortality in Nepal. Upon occurrence of the poisoning, the patient should be provided intensive care treatment.

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