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CASE REPORTS

Acute cerebral infarction following aconitine ingestion

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Abstract

Aconitine is a main component of *Aconitum carmichaeli*, a Chinese herb known to be effective for arthritis and neuralgia. Many senior citizens consume the herb as a folk remedy. Aconitine-associated cardiogenic toxicity has been observed but aconitine-induced cerebrovascular event has not been reported so far. We report two elderly patients who became unconscious and showed neurological dysfunction soon after ingesting aconitine for pain control. We speculate that the aconitine induced cardiac arrhythmia. This resulted in cardiogenic emboli formation with subsequent large territory cerebral infarction.

Keywords: aconitine; cardiotoxicity, cerebral infarction, Chinese herb, adverse effect, neurological toxicity

INTRODUCTION

Aconitum carmichaelii (*Chuanwu*), a frequently used Chinese herb, is a species of flowering plant in the genus *Aconitum*. Aconitine is the major active and toxic component of *A. carmichaelii.*[1] The known pharmacological activities of aconitum include depolarization of voltage-gated sodium channels, and prolongation of action potentials.[2]

A patient with aconitine poisoning typically presents with a combination of neurological, cardiovascular, and gastrointestinal signs and symptoms. Within minutes after ingestion, a patient experiences paresthesia, insensitivity to pain, and weakness, followed by gastrointestinal symptoms.[3] Ventricular arrhythmia is the main cardiac effect of aconitine poisoning. Ventricular arrhythmia usually leads to ventricular fibrillation and subsequent death. The cardiac effect is often also accompanied by respiratory dysfunction due to central respiratory failure. Therefore, aconitine intoxication may result in cardiac arrest and/or respiratory failure.[2]

While many aconitine poisoning cases have been reported, most of these cases involved cardiac adverse effects caused by heart rate changes.[4-6] Herein, we report two unusual cases of acute aconitine intoxication manifesting as hemiparesis and mental deterioration, from acute cerebral infarction.

CASE REPORTS

Patient 1

A 68-year-old man, who has a history of chronic obstructive pulmonary disease (COPD), arrived at our emergency room (ER) with impaired consciousness accompanied by dyspnea, vomiting, and palpitation. The patient had drunk water boiled with aconitine to alleviate his chronic joint pain before arriving at the ER. Laboratory tests indicate a low partial carbon dioxide pressure (pCO_2) of 18.4 mmol/L in arterial blood gas analysis (ABGa), and respiratory alkalosis with a pH of 7.56. Electrocardiography (ECG) showed ventricular tachycardia (Figure 1A). He was subsequently transferred to the intensive care unit (ICU).

His alkalosis was treated with ventilation therapy at the ICU. He became irritable during ventilation. He was then sedated with midazolam. His consciousness improved 12 hours after ventilation therapy. Left hemiparesis was then observed. He showed dysarthria, and his muscular strength was grade III on the left upper limb and grade III, left lower limb, according to the Medical Research Council (MRC) grading system. Low-density lesions were already observed using brain computed tomography (CT) scan. Tissue plasminogen activator (t-PA) was not used as

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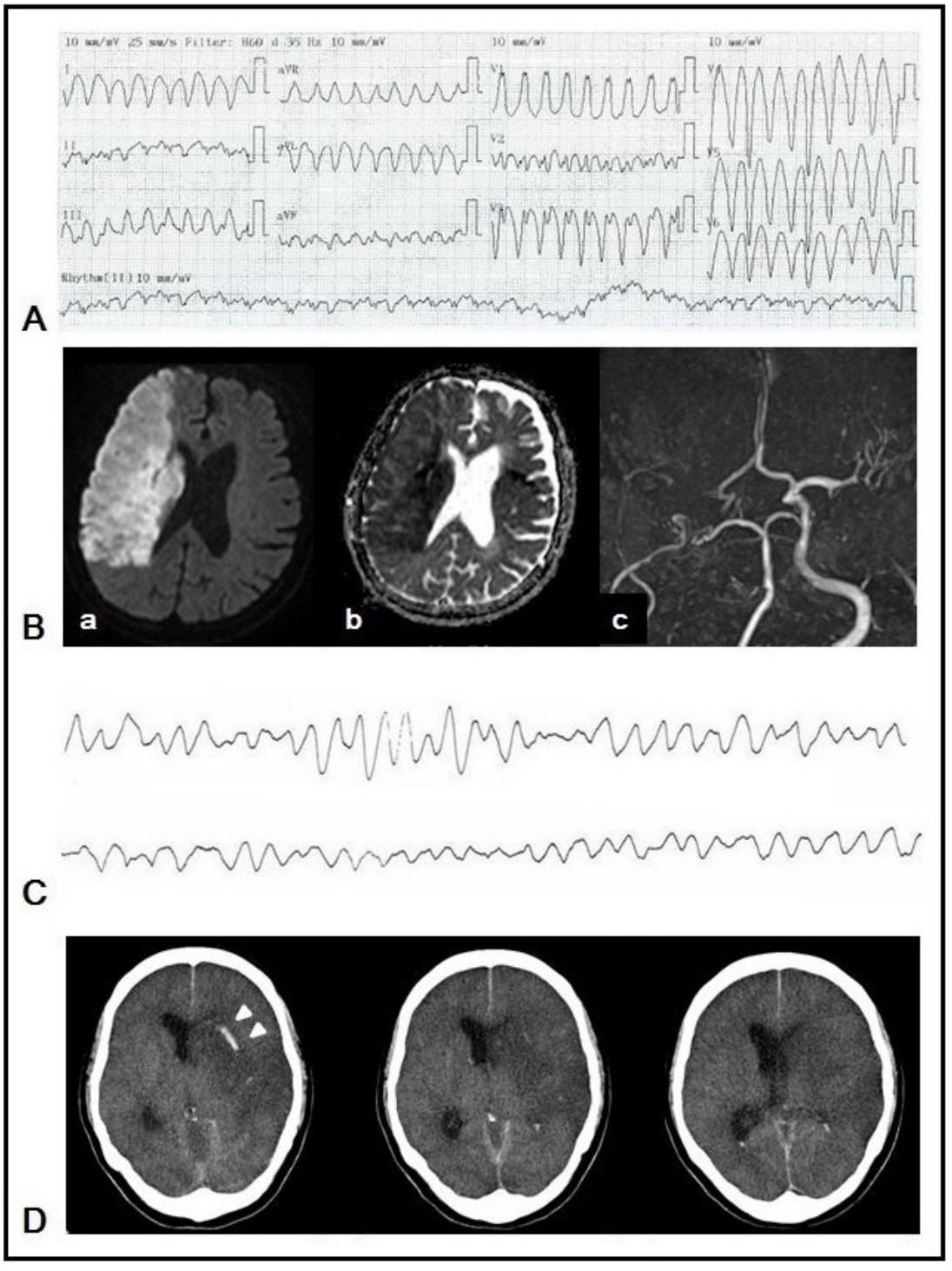

Figure 1: (A) and (B): Patient 1, (C) and (D): Patient 2.
(A) Ventricular tachycardia on electrocardiography (ECG) during hospital admission. (B) Three days after admission, acute infarction in the right middle cerebral artery (MCA) territory and occlusion from the proximal portion of the internal carotid artery were observed on brain magnetic resonance (MR) imaging and angiography. (a: diffuse weighted image, b: apparent diffusion coefficient image, c: MR angiography) (C) Ventricular tachycardia on electrocardiography (ECG) during the hospital admission. (D) The day after admission, acute infarction in the left MCA territory accompanied by intracerebral hemorrhage (arrow head) was observed.

the onset of the neurological deterioration was not clearly determined.[7]

Three days after ICU admission, the patient underwent brain magnetic resonance (MR) imaging and angiography (Figure 1B). An occlusion was observed from the proximal portion of the internal carotid artery. The acute infarction expanded as observed on the CT scan with no severe edematous change around the lesions. The patient regained his consciousness completely, and could breathe without ventilator after day 5.

Patient 2

A 61-year-old woman, non-smoker/non-drinker, with no special medical disorder, visited our ER for chest pain and impaired consciousness. She had neuralgic pain in both feet. She drank water boiled with aconitine 30 minutes before coming to the hospital to control her pain. Pulseless ventricular tachycardia was found when her blood pressure dropped at the ER. Cardio-pulmonary resuscitation (CPR) was performed. After five minutes, spontaneous circulation was restored. The ABGa showed that the pCO_2 was low at 15.6 mmHg, and HCO_3, 13.1 mmol/L. She also showed respiratory alkalosis with a pH of 7.530. The ECG showed ventricular fibrillation (Figure 1C).

Her alkalosis was treated with ventilation therapy. Extra-corporeal membrane oxygenation (ECMO) was performed. Although hematological abnormal findings improved the following day, her stuporous mental state remained, and she did not regain her consciousness. Her muscular strength on the right upper limb motor decreased to MRC grade II, and lower limb motor, MRC grade II. CT brain showed diffuse low-density lesions accompanied by intracerebral hemorrhage in the entire left hemisphere (Figure 1D). Angiography could not be conducted because her creatinine clearance was very low at 12.3 mL/min.

In the next three days, her consciousness worsened and craniectomy was considered. Anisocoria and Cheyne-Stokes respiration suggestive of transtentorial herniation were noted. As her vital signs were unstable, surgery could not be performed. After five days, she developed sudden cardiac arrest. She succumbed and died despite cardio-pulmonary resuscitation.

DISCUSSION

Aconitine is an active component extracted from *Chuanwu* (草烏). It is commonly used as a painkiller for arthritis and neuralgia in several Asian countries including South Korea and China. Traditional medicine practitioners in South Korea use this herb infrequently. Because of the potential toxic effects of this herb, although it is recommended to only supply the herb with a prescription from a traditional medicine doctor, the general public can still obtain the herb almost without any restriction in South Korea, where traditional medicines and folk remedies are commonly used. Consequently, aconitine poisoning still occasionally occurs.

Aconitum rootstocks are extracted through soaking or boiling them in water. This boiling process leads to the hydrolysis of aconitine alkaloids into less-toxic derivatives like aconines.[8] Severe poisoning cases have been reported after an ingestion of as little as 0.2 mg of pure aconitine or after the consumption of decoctions prepared from prescriptions containing 6 g of cured Aconitum rootstocks.[8] Toxicity usually occurs within 10 minutes to an hour after ingestion. Aconitine and its related alkaloids can bind with a high affinity to the open state of sodium channels, causing their persistent activation.[4] The signs and symptoms were manifestations of gastrointestinal, cardiovascular, and neurological toxicity. Cardiovascular effects are the main toxic effects of aconitine, and can occasionally be fatal.

Our two patients demonstrate that neurological signs and symptoms can occur after taking aconitine. Both patients had cardiac arrhythmias, with large cerebral infarctions. We believe the cerebral infarcts were from cardiogenic embolism secondary to the cardiac arrhymias. This complication has not been previously reported in the medical literature.

In conclusion, arrhythmias caused by aconitine intoxication can result in cardiogenic embolism, which may cause secondary cerebral infarction.

DISCLOSURE

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Conflicts of interest: None.

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