POISONING BY ACONITE; CLINICAL FEATURES OF ACONITE POISONING

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Introduction: Aconite is a well-known toxic plant and contains Aconitum alkaloids, including aconitines (aconitine, mesaconitine, hypaconitine, and jesaconitine), which are the substances that cause aconite poisoning. Various toxic symptoms are observed in aconite poisoning cases, and aconite poisoning often results in death from refractory ventricular fibrillation.
Objective: The aim of this study was to describe the clinical features of aconite poisoning, and the relationship between the arrhythmias and the serum concentration of aconitines in aconite poisoning cases.
Method: The subjects of this study were 30 patients with aconite poisoning who were admitted to the Iwate Medical University Hospital between 1984 and 2011. The medical charts were reviewed to obtain information about the background characteristics of the patients. The serum concentration of aconitines was determined by liquid chromatography mass-spectrometry.
Results: The subjects comprised 22 males and 8 females, ranging in age from 5 to 78 years (mean age, 48.3±17.0 years). The causes of the intoxication were suicide attempt (13 cases), suicide (2 cases), and mistaken ingestion of the poisonous plant in place of edible wild plants (15 cases). The symptoms of the intoxication were as follows: dizziness (9 cases), nausea/vomiting (24 cases), circumoral paraesthesia (23 cases), numbness of the extremities (23 cases), palpitation (19 cases), hypotension (18 cases), chest pain/chest discomfort (17 cases), and arrhythmias (25 cases). The arrhythmias varied in type, and included ventricular fibrillation, torsade de pointes, ventricular tachycardia, non-sustained ventricular tachycardia, premature ventricular contractions, and premature atrial contractions. Antiarrhythmic agents such as lidocaine and mexitil etc. were used to treat the tachyarrhythmias. Of the six cases with refractory ventricular fibrillation, two died and four survived with percutaneous cardiopulmonary support (PCPS) (3 cases) or cardiopulmonary bypass (1 case). The serum concentration of aconitines at the point of disappearance of the arrhythmia in five cases was 0.83±0.27 ng/mL.
Conclusion: Patients with aconite poisoning invariably manifested nausea/vomiting, numbness, and arrhythmias. The main symptom of the intoxication was arrhythmia. Although many cases received treatments for tachyarrhythmias, the effect of the antiarrhythmic agents on the tachyarrhythmia was unclear. PCPS seems to be effective for patients who became haemodynamically unstable as a result of arrhythmia refractory to treatment with antiarrhythmic agents and/or electrical cardioversion. Thus, the serum concentration of aconitines could serve as an indicator for the disappearance of arrhythmia in aconite poisoning patients. These results may represent information useful for the treatment of aconite poisoning in the future.