International Journal of Biology and Life Sciences
ISSN: 2957-9511 | Vol. 3, No. 1, 2023

Acute Traditional Chinese Medicine Fuzi Poisoning: A Case Report

Jingyang Sun [1], Juanjuan Guo [2, *]

[1] Graduate school, Changzhi Medical College, Changzhi, Shanxi 046000, China
[2] Department of Geriatric Medicine, Heping Hospital Affiliated to Changzhi Medical College, Changzhi, Shanxi 046000, China
*** Corresponding author:** Juanjuan Guo (Email: 1127492473@qq.com)

Abstract: Fuzi is lateral root of aconite crowfoot plants of aconite root of processed products, its medicinal sheen, sweet, hot, toxic, authority and by the kidney, spleen, for "back to Yang inverse first to save medicine", there are back to Yang Yang to save inverse, fire, cold analgesic effect, modern pharmacology study anti-inflammatory analgesic, cardiac, anti-aging and other functions, is the doctor of traditional Chinese medicine clinical commonly used drugs, often used in the treatment of cardiovascular diseases, diseases such as rheumatoid arthritis. According to different machining processing technology can be divided into salted aconite root, Heishun tablet, Baishun tablets, Light monkshood tablet, etc. Clinical use of Fuzi is often after processing and attenuation, but poisoning still occurs from time to time. A case of poisoning caused by taking Fuzi in our hospital is reported as follows.

Keywords: Fuzi; Aconitum Alkaloids; Aconitine Poisoning; Arrhythmia.

1. Case Report

The patient, an 82-year-old male, was admitted to our department at 13: 44 on December 6, 2022 because of "dizziness for 6 hours". The patient suffered from dizziness, fatigue, chest tightness, shortness of breath, blurred vision, numbness of the lips and tongue tip, no chest pain, sweating, nausea, vomiting, abdominal pain, syncope, hemiplegia, aphasia and unconsciousness after taking the decoction of traditional Chinese medicine (including Fuzi) orally 6 hours ago. Self-measured blood pressure at home was 76/64mmHg(1mmHg=0.133kPa), so he went to our hospital for emergency treatment. The patient had a history of coronary stent implantation for 6 years, and stopped atorvastatin calcium tablets and aspirin for one month. The history of hypertension is 10 years, and the highest blood pressure is 180/100mmHg. He usually takes nifedipine sustained-release tablets 10mg/ time, twice a day, and the blood pressure control is ideal, about 130/80mmHg. [1]

Physical examination on admission: body temperature: 36.8℃; Pulse: 91 beats/min, irregular pulse. ; Breathing: 20 times/minute; Blood pressure: 70/46 mmHg; Conscious, skin wet and cold, answer to the point, jugular vein not bulging, bilateral pupils equicrural, about 2.5mm in diameter, soft neck, no resistance, clear breathing sounds in both lungs, no obvious dry and wet rales, heart rate 71 minutes, irregular rhythm, no obvious murmurs in all valve areas of the heart, flat and soft abdomen, no tenderness and rebound pain, no touch under the liver and spleen ribs, no edema and lower limbs. Auxiliary examination: biochemistry: serum albumin (ALB) 34.4 $g \cdot L^{-1}$, serum cholinesterase (CHE)6130 $U \cdot L^{-1}$, glucose (Glu) 7.75 $mmol \cdot L^{-1}$, Na 136.0 $mmol \cdot L^{-1}$and Ca 2.15$mmol \cdot L^{-1}$. Blood gas analysis: PH 7.416, PCO2 34.2 mmHg, PO2 76.9 mmHg. Liver function, renal function and thyroid function are normal. Electrocardiogram: complete right bundle branch block (see Figure 1). Perfect cardiac color Doppler showed that the inner diameter of ascending aorta and aortic sinus widened, aortic valve insufficiency (calcification), left ventricular diastolic compliance decreased, left ventricular ejection fraction was 63%, second, tricuspid regurgitation (light) and aortic valve regurgitation (light+). MRI examination of the head: ischemic white matter lesion under the left frontal cortex, and A1 segment occlusion of the right anterior cerebral artery. 24-hour dynamic electrocardiogram showed sinus rhythm, frequent supraventricular premature beats, paroxysmal atrial tachycardia, ventricular premature beats occasionally in pairs (19:37), partial T wave changes, and heart rate variability (HRV)SDNN 147 ms (see Figure 2). Admission diagnosis: aconitine poisoning, polymorphic arrhythmia, drug-induced hypotension, coronary heart disease after stent implantation, hypertension level 3 very high-risk group. After admission, ECG monitoring was given to monitor blood pressure and blood oxygen saturation, continuous pumping of norepinephrine bitartrate into vein to boost blood pressure, intravenous drip of esmomeprazole to inhibit acid and protect stomach, oral Wenxin Granule and plenty of fluid replacement to promote symptomatic treatment of drug metabolism.

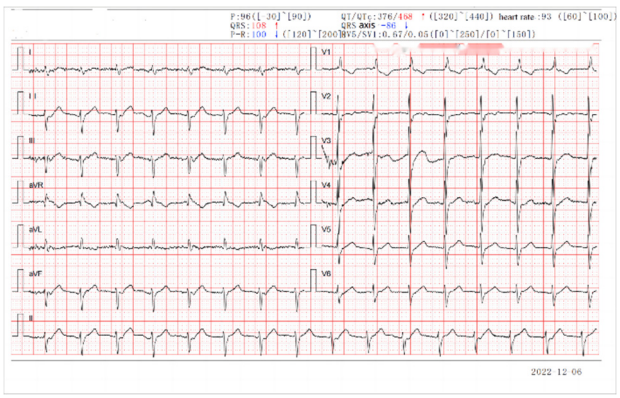

Figure 1. Electrocardiogram

On the second day of hospitalization, the patient's numbness around the mouth and the tip of the tongue was slightly less than before, but he still complained of dizziness, and his blood pressure fluctuated between 100-120/60-80

mmHg (pumped with norepinephrine). On the third day of hospitalization, the patient's blood pressure was stable, the numbness around the mouth and the tip of the tongue was significantly reduced, and dizziness improved. Metoprolol tartrate tablets were added to control the heart rhythm, and the patient and his family asked to leave the hospital. Telephone follow-up on the fifth day after discharge showed that the patient had no dizziness, shortness of breath, numbness around the mouth and the tip of the tongue, and his self-rated blood pressure returned to normal, with occasional palpitations and discomfort.

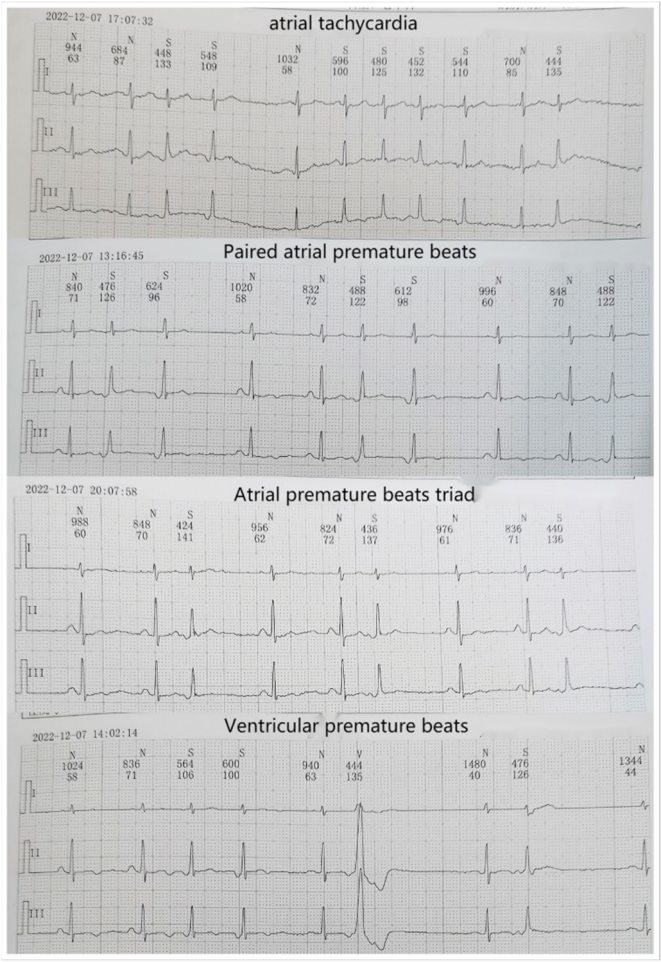

Figure 2. Dynamic electrocardiogram (I, II, III leads)

2. Discussion

Aconitum carmichaeli is a toxic Chinese herbal medicine, and its toxicity comes from aconitine alkaloids. Oral aconitine 0.2mg can be poisoned, and 3-5mg will be fatal [2]. According to their pharmacological activities, it can be divided into diester-diterpenoid alkaloids (DDAs), monoester-diterpenoid alkaloids (MDAs) and unesterified-diterpenoid alkaloids (UDAs) [3]. Diester alkaloids are the main sources of aconitine toxicity, including aconitine (AC), neoaconitine (MA) and hypaconitine (HA). More and more studies show that aconite can cause multiple organ injuries, especially cardiotoxicity and neurotoxicity [4]. The Chinese pharmacopoeia suggested that aconite should be decocted first and then decocted for a long time, so as to hydrolyze the diester alkaloids with high toxicity into monoester alkaloids and aminoalcohol aconitine with low toxicity, thus reducing the risk of poisoning. This case decocted traditional Chinese medicine by itself, and the decoction time was insufficient, which was the main reason for its poisoning.

Aconitum alkaloids poisoning can involve nervous system, cardiovascular system, digestive system, respiratory system, etc., among which the damage of nervous system, digestive system and cardiovascular system is the most important [5]. Patients may suffer from numbness of limbs, tongue and mouth, convulsion, dizziness, confusion, mental disorder, nausea, vomiting, abdominal pain, abdominal distension, diarrhea, abnormal liver function, palpitation, decreased or increased blood pressure, and arrhythmia. In severe cases, nausea, arrhythmia and cardiac arrest may be life-threatening. Cardiotoxicity is the main feature of aconitine poisoning and the most common cause of death, which can cause many types of arrhythmia, among which ventricular arrhythmia is the most common [6,7]. One or more kinds of arrhythmia can occur in the same patient, and the types of arrhythmia may change during the poisoning process. Its mechanism may be related to vagus nerve excitation, inhibition of sinoatrial node, and action on ventricular muscle to increase the excitability of ectopic rhythm points [8]. From the electrophysiological point of view, the interaction between aconitine and cellular ion channels and the destruction of intracellular ion homeostasis are the key mechanisms of aconitine-induced arrhythmia. Wei Zhou [9] and Peng F [10] found that aconitine induced intracellular NA^+-Ca^+ overload by affecting Na^+channels, Na^+-K^+-ATPase, Ca^+channels, K^+channels and connexin 43(Cx43) in myocardial cells, and induced arrhythmia, at the same time, it affected mitochondrial apoptosis and oxidative stress damage in myocardial cells.

The diagnosis of acute aconitine poisoning is based on: (1) a history of taking or coming into contact with aconitum herbs and their products; (2) There is a disorder of arrhythmia; (3) Clear toxicological detection. If both (1) and (2) are satisfied, clinical diagnosis can be made. In addition, combined with the characteristics of the patient's arrhythmia, it is necessary to identify the poisoning from poisonous mushrooms, digitalis, oleander and other plants [11]. The patient had a clear history of taking aconite. The emergency electrocardiogram showed complete right bundle branch block, and the Holter electrocardiogram showed pleomorphic arrhythmia 24 hours after admission, which could be diagnosed as acute aconite alkaloid poisoning.

Bedside electrocardiogram (12-lead or 18-lead) was performed as soon as possible at the first diagnosis to observe patients for arrhythmias, circulatory failure, respiratory failure, and unconsciousness. At present, there is no specific antidote for aconitine poisoning. The key to treatment is to reduce the residual drug concentration in the body as soon as possible. Gastric lavage and emesis should be carried out as soon as possible, and a large amount of rehydration, diuresis, drainage and other measures should be carried out to reduce the absorption of poison. Gastric lavage is generally the best effect within 6 hours, and the gastric lavage time window can be appropriately relaxed for patients with large intake and serious illness. For critically ill patients, hemoperfusion can be started early under comprehensive treatment, and can be repeated according to the patient's condition [12]. Patients with hemodynamic instability due to aconitine poisoning may be given vasoactive drugs such as norepinephrine. Respiratory failure, heart failure can be given appropriate treatment. Zeng [13] and Dao[14] et al. believed that atropine could counteract the overexcitation of the vagus nerve of the heart, improve the excitability of the sinoatrial node, and thus terminate arrhythmias. Atropine could be used as the first-choice drug for arrhythmias caused by aconitine poisoning,

and it was suitable for both chronic and tachyarrhythmia. For tachyarrhythmias with stable hemodynamics, lidocaine, a class I antiarrhythmic drug, can be selected, which can rapidly promote K+ outflow and reduce myocardial automatoriality. Liu et al. [15] concluded that amiodarone is better than lidocaine in controlling tachyarrhythmias caused by monitonine poisoning. Therefore, when the use of lidocaine is not effective, Beta blockers and amiodarone are also available. Patients with hemodynamic disorder and severe ventricular arrhythmia should be given electric cardioversion therapy in time [13]. Hypotension occurred in the early stage of this case, and the electrocardiogram showed complete right bundle branch block. The initial doctor gave it booster and fluid replacement treatment, and polymorphic arrhythmia gradually appeared after admission. After giving a lot of fluid replacement to promote poison metabolism and applying vasoactive drugs, the vital signs recovered smoothly.

3. Conclusion

To sum up, for patients with dizziness, blurred vision, numbness of limbs and mouth, nausea and vomiting, palpitation, chest tightness, arrhythmia, and decreased blood pressure, it is necessary to ask in detail whether there is any history of consumption and contact with drugs containing aconitine alkaloids, be highly alert to whether it is aconitine poisoning, and give gastric lavage, drainage and other treatments as soon as possible after diagnosis to reduce the absorption of poisons. Patients with arrhythmia and hemodynamic disorder should be given hemoperfusion and antiarrhythmic treatment as soon as possible to improve the success rate of treatment. At the same time, it is necessary to strengthen public health education, guide the rational and safe use of drugs, and avoid poisoning incidents.

References

[1] Xu Xin, Li Gangmin, Sun Chen, et al. Research progress on water-soluble alkaloids of Radix Aconiti Lateralis and their pharmacological effects [J]. Pharmacology and Clinical of Traditional Chinese Medicine, 2021,37(05):213-219.

[2] Wang Dan, Jia Dexian, Li Zhenzhen, et al. Discussion on safety evaluation and risk control measures of Aconitum kusnezoffii [J]. China Journal of Traditional Chinese Medicine, 2018, 43 (15): 3093-3100.

[3] Wu J J, Guo Z Z, Zhu Y F, et al. A systematic review of pharmacokinetic studies on herbal drug Fuzi: Implications for Fuzi as personalized medicine[J]. Phytomedicine, 2018,44: 187-203.

[4] Wang M, Hu W J, Zhou X, et al. Ethnopharmacological use, pharmacology, toxicology, phytochemistry, and progress in Chinese crude drug processing of the lateral root of Aconitum carmichaelii Debeaux. (Fuzi): A review[J]. J Ethnopharmacol, 2023, 301:115838.

[5] He Jian, Wu Ping, Dong Yu, et al. Analysis of adverse reactions of Radix Aconiti Lateralis and mechanism prediction of cardiotoxicity caused by network pharmacology [J]. China Journal of Traditional Chinese Medicine,2019,44(05):1010-1018.

[6] Ye Q, Liu H, Fang C, et al. Cardiotoxicity evaluation and comparison of diterpene alkaloids on zebrafish[J].Drug Chem Toxicol, 2021,44(3):294-301.

[7] Liu Mingyan. Electrocardiogram and clinical examination analysis of 43 cases of acute aconitine poisoning [J]. International Journal of Laboratory Medicine, 2014,35 (17): 2296-2297.

[8] Li Chaorong, Peng Wenjing. A case of second-degree atrioventricular block caused by taking conventional dose of accessory poisoning [J]. Primary Medical Forum, 2020,24 (10): 1467-1468.

[9] Zhou W, Liu H, Qiu L Z, et al. Cardiac efficacy and toxicity of aconitine: A new frontier for the ancient poison[J]. Med Res Rev, 2021,41(3):1798-1811.

[10] Peng F, Zhang N, Wang C, et al. Aconitine induces cardiomyocyte damage by mitigating BNIP3-dependent mitophagy and the TNFalpha-NLRP3 signalling axis[J]. Cell Prolif, 2020,53(1): e12701.

[11] Emergency Physician Branch of Chinese Medical Association, Emergency Recovery and Disaster Medicine Committee of Chinese Medical Association, China Emergency Medical Association, et al. Expert Consensus on Diagnosis and Treatment of Acute Aconitum Alkaloid Poisoning [J]. Chinese Journal of Emergency Medicine, 2022,31(03):291-296.

[12] Peng Hao, Tang Jiang, Yang Hance, et al. Investigation and analysis of a case of poisoning caused by self-made medicinal wine in a gathering of residents [J]. Practical Preventive Medicine, 2020,27(03):374-375.

[13] Zeng Huan-chao, Quan Zheng-lin, Fang Zhi-cheng, et al. Report on a case of sudden cardiac arrest caused by poison in Wutou alkali and recovery to success [J]. Miscellany of Chinese Medicine and Industry, 2021,34(02):119-121.

[14] Dongyuan Dao. Analysis of emergency rescue methods for severe arrhythmia caused by aconitine poisoning [J]. World Compound Medicine, 2021,7(01):43-45.

[15] LiuWei. Comparison between amiodarone hydrochloride and lidocaine injection in the treatment of ventricular arrhythmia caused by aconitine poisoning [J]. Journal of Clinical Rational Drug Use,2020,13(23):47-48.