Cardiac Arrests after Eating Raw Grass Carp Gall Bladders: A Case Report

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Case report

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Abstract

BACKGROUND: Cases of poisoning by eating raw fish gallbladder occur mainly in Asia. These cases have been reported mainly with acute liver and kidney damage. We found that the ingestion of carp gallbladder also caused myocardial damage and lead to frequent cardiac arrest in patients with subclinical long QT interval syndrome.

Patient concerns: We report a 57-year-old woman with an unexplained Long QT syndrome history who developed arrhythmia sudden death two times following ingestion of raw fish gall bladder.Fish bile poisoning, toxic hepatitis, acute renal failure, cardiac arrest, postoperative cardiopulmonary resuscitation, long QT syndrome were diagnosed. As in this case, myocardial injury caused by fish bile poisoning further leads to cardiac arrest related to LQTS.The patient underwent continued renal replacement therapy (CRRT) and other supportive treatments to manage symptoms, promote detoxing, protect liver and myocardium in our hospital. Then she completed a cardiac pacemaker placement in local hospital.Her liver enzymes profile returned to normal after 18 days, and kidney injury index returned to normal after more than one month. On follow up after three months, her renal as well as the liver parameters were found normal and she never attack malignant arrhythmia again.

Conclusion: Eating fish bile can lead to MODS, not merely to liver and kidney damage. Some of the LQTS patients usually have no clinical symptoms. Clinicians should be aware of changes in low-risk LQTS patients when something special happens to them.

Core Tip

The majority of cases of fish gallbladder poisoning mainly lead to liver and kidney damage. We report a woman with myocardial injury caused by fish gallbladder poisoning. It is thought that the damage to the heart muscle caused her asymptomatic long QT interval syndrome to become a cardiac arrest. For patients with subclinical long QT syndrome the high risk of sudden death from severe disease should be taken into account.

1. Introduction

In some countries and regions of Southeast Asia, local residents believe that eating raw fish gall bladder can cure their chronic diseases and boost immunity and it is also a signification of courage. Some research shows that a kind of toxin bile salt which is called 5 alpha-cyprinol sulphate with hepatotoxic and nephrotoxic is contained in fish gall bladder especially in carp [1].

Long QT syndrome (LQTS) is a syndrome of unknown etiology which characterized by prolonged QT interval in the electrocardiogram and accompanied by abnormal T wave and/or U wave. Some of the LQTS patients have no clinical symptoms when it does not occur. Congenital LQTS is usually genetically related and these patients are more likely to experience syncope and even sudden death than others [2].

2. Case Presentation

Chief complaints

A 57-year-old woman came to the emergency department in our hospital after taking fish gallbladder for six hours.

History of present illness

She ate 4 fresh grass carp gall bladders as a folk remedy for stomachache. Her husband took her to hospital at once, while she has no specific clinical symptoms or signs. As she strongly refused to get a gastric lavage and any other inspection or treatment, the doctor let them go. After 6 hours of ingestion of gall bladder, she developed dizziness accompanied by visual rotation, nausea and vomiting. She also had diarrhea three times during this period. So she came to our emergency department at the second time.

History of past illness

The patient did not provide any previous medical history except a stomachache without clinical diagnosed.

History of family illness

She and her family had a free family history.

Physical examination

The patient characteristics, measured vitals on admission are depicted in Table 1. All physical examinations about cardiopulmonary revealed no abnormality. There was neither lymphadenopathy nor hepatosplenomegaly. Neurological examinations were normal too.

Table 1
Patient Profile

Age	Gender	Temper	Pulse	Respiratory Rate	Blood Pressure	chronic diseases	allergic history
57	Female	36.2℃	74 bpm	19 bpm	93 / 64 mmHg	None	None

Laboratory examinations

Electrocardiogram(ECG)showed a QT interval of 518 ms.Her creatine kinase isoenzyme and creatine phosphokinase increased while he had a normal cardiac troponin I at that time. Table 2 is some of the biochemical values.

Course records and treatment

As she still refused to get neither nether gastric lavage nor hemodialysis, doctors adopted other supportive treatments to manage symptoms, protect liver and promote detoxing at that night.

Her indicators of liver enzymes and kidney damage increased significantly and her urine output decreased since the next day. Continued renal replacement therapy (CRRT) was received on the second to fourth days then urination improved and renal function got normalized. Drugs such as glutathione continue to be used for liver protection. Symptoms and indicators gradually improvedAfter seven days, she consulted with her family to stop hemodialysis. After that, the biomarkers of kidney damage increased, but urine volume was almost normal. Liver enzymes profile returned to normal after 18 days, and kidney injury index returned to normal after more than one month.

On day 8, the patient developed chest distress and asthma, and computerized tomography showed massive pleural effusion on both sides. She underwent bilateral thoracic close drainage.

Table 2
Some of Serum Biochemical Values during Hospitalization

Serum Biochemical Values	1st day	2nd day	4th day	7th day	9th day	12th day	18th day	23th day (after CPR)	25th day	28th day	38th day	45th day
urea-nitrogen review(2.5–6.1 mmol/L)	6.41	9.43	2.23	4.96	10.05	11.55	6.59	9.39	10.62	9.17	7.91	7.02
serum creatinine(46-92umol/L)	53.4	187.8	79.5	193.7	391.3	388.9	188.1	162.7	243.7	169.7	83.8	68.3
creatine phosphokinase(30-135U/L)	163	55			30			184	515	93		
creatine kinase isoenzyme(0-25U/L)	225	17.8			4.7			32.4	3.2	3.2		
cardiac troponin I (0-0.01 ng/ml)	< 0.01	0.06			0.05	0.03	< 0.01	0.84	0.14	< 0.01		
total bilirubin (3.4-17.1umol/L)		21.8	44.1	21.8	17.3	18.1	16.8	9.5		9.4	8.6	
direct bilirubin (0.1-3.4umol/L)		12.3	30.5	8.1	5.8	5.0	4.1	2.5		2.4	1.7	
alanine aminotransferase(5-35U/L)		4829	1407	493	225	102	27	60		33	18	
aspartate aminotransferase(8-40U/L)		9922	554	54	29	21	14	109		32	21	
lactate dehydrogenase (109-245U/L)		8321	261	238	204	228	253	431		312	183	
total bile acid (0.1-9.67umol/L)		155.1	169.2	4.7	1.5	1.9	1.2	2.3		2.7	3.4	

In the early hours of the 23rd day, her relatives found her gasping for breath and unconscious. She was asphygmia when doctors arrived. We quickly gave her cardiopulmonary resuscitation and endotracheal intubation. She regained her autonomic rhythm in about half an hour. On day 25, her ECG monitor recorded a brief ventricular fibrillation and then return to sinus rhythm rapidly. Metoprolol and lidocaine were used to control the arrhythmia. She recovered well and on day 26 she had the endotracheal tube removed and was transferred to the observation ward. At this time, the ECG showed that a QT interval of 550 ms was accompanied by abnormal T waves. On the 43rd day, the patient had another sudden cardiac arrest when talking with others in the morning. After cardiopulmonary resuscitation for about 10 minutes, she regained consciousness and was hospitalized for observation. Considering her long QT interval, the possibility of arrhythmia or even sudden death can happen at any time, we suggests the installation of a pacemaker. After consultation with her family, she decided to return to the local hospital to complete the cardiac pacemaker placement. On follow up after three months, her renal as well as the liver parameters were found normal.

3. Discussion

Jervell and Lange-Nielsen form and Romano-Ward forms are two kinds of congenital LQTS. The former is also known as heart and auditory (deafness) syndrome [3, 4]. Even a sizeable proportion of low-risk, adult LQTS patients with no cardiac symptoms

ECGs were generally normal except for the slight extension QT interval and follow-up studies have shown no impact on their survival [5]. Our patient used to be one of them. As we later learned, she had only fainted a few times in the past fifty years. Only the QT interval was prolonged in her previous ECGs without T wave or U wave changes. Although it does not affect the treatment but it reflects our lack of awareness in early stages of the disease.

4. Conclusion

There is nothing special about the treatment of this case, but the following experience can be provided. We should also pay attention to the protection of other organ functions when we pay attention to liver and kidney function damage in patients with fish bile poisoning. Since myocardial damage from eating fish gall bladder has rarely been reported. In this case, fish bile poisoning should be the cause of ventricular fibrillation and cardiac arrest. Clinicians should be aware of changes in low-risk LQTS patients when something special happens to them.

Declarations

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The authors declare that no funding was recieved during the study.

Availability of data and materials

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Contributions

Han J Y, Zhuo L,Li F,Huang S Q and Chen D M were the patient's bedside physician, reviewed the literature and wrote manuscript drafting; Sun Y and Chen X B performed the microbiological analyses and interpretation and contributed to manuscript drafting ; Sun Y and Luo J Y, deputy chief physicians, participated in the diagnosis and treatment of the patient; Wang Y L and Li X M were responsible for the revision of the manuscript for important intellectual content. The authors have no conflicts of interest to declare.

Statement of Ethics

The authors declare that subject has given her written informed consent to publish this case.

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Competing interests

The authors declare that they have no competing interests.

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