Renal Failure and Hepatitis Following Ingestion of Raw Grass Carp Gallbladder: A Case Report

Lina Zhou

Wenzhou Medical University

Shaoshao Dong

Wenzhou Medical University

Shengze Zhang

Wenzhou Medical University

Wen Huang (✉ qq2627897841@126.com)

hospital

Case Report

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Abstract

Background: Fish gallbladder has long been used as folk remedy in Asian countries. Multiple organ damage after fish gallbladder ingestion resulting in near mortality has been known to occur. Here, we describe a case of grass carp gallbladder poisoning and review the literature.

Case Presentation: A previously healthy, 50-year-old woman was admitted to our hospital with a 2-day history of generalized abdominal pain and repeated vomiting following ingestion of 2 raw, grass carp gallbladders in an attempt to alleviate her cough. She developed anuria on day 4 with markedly elevated serum creatinine, urea, bilirubin, alanine aminotransferase, and aspartate aminotransferase. Based on thorough evaluation of her history and prompt biochemical investigations, we diagnosed her with acute renal failure and hepatitis secondary to fish gallbladder poisoning. Her renal biopsy revealed acute tubular necrosis. And she underwent six sessions of conventional hemodialysis due to renal failure. Supportive treatment, like gastric mucosal protectant and liver protectant were administered for targeted organ protection. The patient's liver function gradually recovered and serum creatinine was 164 $\mu\text{mol/l}$ at discharge on day 24. Over a period of 2-week follow-up, her renal function completely recovered.

Conclusion: The purpose of this paper is to highlight the need for physicians to be mindful of the toxic complications of raw grass carp gallbladder ingestion and to promote its awareness among the people to help reduce this incidence of food poisoning.

Background

Fish gallbladder has long been used as folk remedy in Asian countries. In some rural areas of southeastern and southern provinces in China, including Hong Kong and Taiwan,¹ the habit of swallowing fish gallbladder to cure diseases such as asthma, arthritis, and cough, enhance visual acuity, relieve pain, and perform detoxification is prevalent. Multiple organ damage after fish gallbladder ingestion resulting in near mortality has been known to occur. Cases of poisoning have also been reported in Cambodia, Japan, Korea, India, and Vietnam.^{2–4} This practice poses a serious health concern from the standpoint of food safety. With improved development in health care in China, we now have effective treatment for patients with fish gallbladder poisoning. The purpose of this paper is to raise the awareness regarding food poisoning secondary to ingestion of raw cyprinid fish.

Case Presentation

A 50-year-old woman consumed 2 raw grass carp gallbladders (approximately 4–6 ml) for alleviation of her cough 2 days prior to presenting at our hospital. She developed diffuse abdominal pain and profuse vomiting six hours after its ingestion accompanied by sweating, dry mouth, heaviness of the chest, and dizziness. She had no history of hypertension, diabetes, cardiac disease, cerebrovascular disease, allergies, surgical interventions, and no relevant family history. She presented to a nearby health clinic ten hours after ingestion, where her laboratory investigations showed decreased leukocytes ($2.1 \times 10^9/\text{L}$), neutrophils ($1.27 \times 10^9/\text{L}$), and lymphocytes ($0.65 \times 10^9/\text{L}$), erythrocytes and platelets were normal. With a provisional diagnosis of acute gastroenteritis, she received empirical antiemetics (Metoclopramide 10 mg IV daily), proton pump inhibitors (Omeprazole 40 mg IV daily), and intravenous saline for 2 days without improvement. She was then referred to the gastroenterology department of our center for further treatment on day 3. At admission, her temperature was 37.1°C , pulse rate 46/min bpm, respiratory rate 20/min, and blood pressure 108/74 mmHg. She had upper abdominal tenderness, with no rebound pain, hyperactive bowel sounds of 5–6/min, and no pain on renal percussion. The rest of her systemic examination was unremarkable. Her ECG, (shown in Fig. 1) revealed sinus bradycardia of new onset, with heart rate (HR) of 46/min at that time. Her serum creatinine was $344 \mu\text{mol/l}$, urea 15.88 mmol/L (normal range being 2.5–6.4 mmol/L), and she was referred to the Nephrology department for further management.

On day 4, her urine output reduced drastically (less than 100 ml over 24 hours). Serum creatinine rose markedly to $505 \mu\text{mol/L}$. Liver function revealed increased ALT, AST, total bilirubin and direct bilirubin, as shown in Table 1. She was diagnosed to have acute renal failure (ARF) and hepatitis. She was started 3–4 hour sessions of hemodialysis on day 4 due to renal failure, which was then maintained every 2 days. Gastric mucosal protectant omeprazole 40 mg IV daily for four days and liver protectant reduced glutathione 1.2 g IV daily for 14 days were administered. Her urine output increased to 1400 ml/24 hours on day 12, liver function improved and dialysis was withdrawn on day 19. After exclusion of contraindications, she consented for a renal biopsy to identify the cause of her ARF on day 13, and it revealed acute tubular necrosis (shown in Fig. 2). She was discharged on day 24, with serum creatinine having decreased to $164 \mu\text{mol/l}$. After discharge, she was followed up with weekly serum biochemical tests in the nephrology outpatient department and her renal function completely recovered after 2 weeks.

Table 1
Selected indicators after admission

	Day3	Day4*	Day5	Day6	Day7	Day10	Day12&	Day14	Day17	Day19§	Day22	Day24#	Day31
liver function													
ALT		2739.00	1674.00	1052.00	684.00	264.00		86.00	61.00	39.00			
AST		1399.00	416.00	155.00	76.00	37.00		21.00	23.00	21.00			
glutamyl transpetidase		212.00	178.00	162.00	141.00	104.00		78.00	89.00	80.00			
total bile acid		/	32.30	14.80	17.00	/		/	/	/			
glucocholic acid		/	29.85	13.10	11.60	/		/	/	/			
total bilirubin		49.70	32.70	25.30	17.30	12.70		15.30	15.70	12.50			
direct bilirubin		43.50	26.60	19.20	12.70	8.10		8.30	8.90	7.40			
albumin		42.90	38.70	35.90	35.20	36.50		36.00	49.10	48.50			
renal function													
serum creatinine	344.00	505.00	521.00	551.00	/	589.00		590.00	580.00	458.00	242.00	164.00	104
urea	15.88	19.60	17.30	15.00	/	10.20		11.40	14.80	18.30	15.80	12.20	6.54
uric acid	647.00	739.80	550.50	500.30	/	454.80		391.30	406.50	427.10	375.10		
uronoscopy													
urine protein	/	2+(1)	/	/	/	1+ (0.5)	/	/	-	/	/		
urine chlorine								187.8					
urine sodium								221.9					
urine potassium								19.85					
urine uric acid								0.7					
24 h urine protein								303					
urine-NAG)		22.9											
urine IgG		149.0											
urine β2-microglobulin		3.44											
urine α1-microglobulin		34.0											
urine transferrin		55.0											
urine amylase		542.00											
routine blood test													
leukocyte	5.7	/	5.60	/	7.00	6.90	6.40	8.50	/	6.90	6.40		5.9
neutrophils	4.7	/	4.60	/	5.50	5.10	4.90	7.10	/	4.80	4.30		3.9

IgG = immunoglobulin G; HAV = hepatitis A virus, HBV = hepatitis B virus, HCV = hepatitis C virus, HEV = hepatitis E virus;ANA = antinuclear antibodies; anti-GBI basement membrane antibody; ANCA = antineutrophil cytoplasmic antibodies; IgE = immunoglobulin E; IgA = immunoglobulin A; IgM = immunoglobulin M.

Day4*:convention hemodialysis was performed; Day 12&: patient's urine output increased to 1400 ml; Day 19§: liver function improved and withdrawal dialy; patient discharged with serum creatinine 164 umol/l.

	Day3	Day4*	Day5	Day6	Day7	Day10	Day12&	Day14	Day17	Day19§	Day22	Day24#	Day31
lymphocyte	0.56	/	0.60	/	0.90	1.00	0.90	0.90	/	1.40	1.40		1.5
hemoglobin	125	/	116.00	/	114.00	105.00	93.00	92.00	/	89.00	91.00		83
platelet	156	/	128.00	/	116.00	122.00	98.00	84.00	/	111.00	156.00		161
inflammatory indicators													
c-reactive protein	1.4			12.90		6.10							
procalcitonin					3.31						0.12		
serum amyloid A		89.60									7.60		
erythrocyte sedimentation rate		9											
coagulation function													
prothrombin time		12.40			11.20		11.50						
activated partial thromboplastin time		25.50					27.70						
thrombin time		17.20					14.60						
D-dimer		7.20			1.69		4.84						
blood gas analysis													
PH		7.35											
PaCO ₂		29.4											
PaO ₂		81.9											
actual bicarbonate radical		16											
standard bicarbonate radical		18											
electrolytes													
K ⁺	4.60	4.40	4.20	3.80	3.80	3.50	/	3.40	3.80	3.80	4.00	4.00	
Na ⁺	138.00	133.00	135.00	137.00	137.00	137.00	/	136.00	137.00	139.00	136.00	138.00	
CL ⁻	103.00	100.00	99.00	99.00	101.00	99.00	/	95.00	90.00	95.00	94.00	98.00	
P	/	1.50	1.34	1.64	1.65	1.30	/	1.22	/	2.04	1.64		
Ca ²⁺	2.29	2.22	2.18	2.21	2.10	2.17	/	2.10	/	2.31	2.49		
other indicates													
serum ferritin										770.7			

IgG = immunoglobulin G; HAV = hepatitis A virus, HBV = hepatitis B virus, HCV = hepatitis C virus, HEV = hepatitis E virus;ANA = antinuclear antibodies; anti-GBM = anti-glomerular basement membrane antibody; ANCA = antineutrophil cytoplasmic antibodies; IgE = immunoglobulin E; IgA = immunoglobulin A; IgM = immunoglobulin M.

Day4*:convention hemodialysis was performed; Day 12&: patient's urine output increased to 1400 ml; Day 19§: liver function improved and withdrawal dialysis; patient discharged with serum creatinine 164 umol/l.

	Day3	Day4*	Day5	Day6	Day7	Day10	Day12&	Day14	Day17	Day19§	Day22	Day24#	Day31
transferrin saturation										18			
folic acid										11.4			
vitamin B12										453			
erythropoietin										3.8			
parathyroid hormone		75.10								151.40			
serumβ2-microglobulin		7.32											
serum amylase	159.00	137.00	/	/		205.00							
heart function	creatine kinase and Troponin normal range												
hepatophilic virus	HAV, HBV, HCV, HEV, TORCH were negative												
immunization indicators	ANA,anti-GBM and ANCA negative; IgE 212 IU/ml (< 100 IU/ml), C3 0.57 (0.9–1.8 g/L), C4 0.1 (0.1–0.4 g/L), C1q 101 (159–233 mg/L). IgA were normal;Widal's test negative												
IgG = immunoglobulin G; HAV = hepatitis A virus, HBV = hepatitis B virus, HCV = hepatitis C virus, HEV = hepatitis E virus;ANA = antinuclear antibodies; anti-GBM basement membrane antibody; ANCA = antineutrophil cytoplasmic antibodies; IgE = immunoglobulin E; IgA = immunoglobulin A; IgM = immunoglobulin M.													
Day4*:convention hemodialysis was performed; Day 12&: patient's urine output increased to 1400 ml; Day 19§: liver function improved and withdrawal dialy; patient discharged with serum creatinine 164 umol/l.													

Repeat ECG revealed persistence of sinus bradycardia (HR 52 bpm). Trends of indicators are shown in Table 1. Cardiac ultrasound, pulmonary and abdominal CT scans were normal. Renal biopsy is shown in Fig. 2.

Discussion

Wu HL, et al. reviewed fishes with poisonous gallbladders fishes in China.² There are currently 12 known species of fish with poisonous bile in China, including the crucian carp (*Carassius auratus*), black carp (*Mylopharyngodon piceus*), silver carp(*Hypophthalmichthys molitrix*,Cyprinus carpio and big head carp (*Aristichthys nobilis*); all of which belong to the freshwater fish family Cyprinidae. Poisoning caused by grass carp gallbladder accounts for more than 80% of all cases, because it has a relatively big gallbladder.

Ichthyogallotoxin has been studied extensively in grass carp bile.⁵ The compound 5α-cyprinol sulfate (5α-cholestane-3α, 7α, 12α, 26, 27-pentol 26-sulfate) has been implicated in cases of human poisoning and has been isolated from carp bile. The molecular formula of the water soluble and thermostable 5α-cyprinol sulfate is C₂₇H₄₈O₈S. Toxicity is dose-dependent, depending on quantity of bile and size of the ingested fish gall bladder.⁶ Mohri et al.⁷ reported that essentially all of the bile toxicity could be explained by the properties of cyprinol sulfate, based on the LD₅₀ values. The LD₅₀ of 2.5 mg / 20 g mouse was reported. After day 5, some mice died for every 24 h with a dose of 1.0 mg intraperitoneal administration⁷.

Damage to multiple organs after ingestion of fish gallbladder such as,the gastrointestinal, renal, hepatic, cardiac, and neurological systems have been reported previously⁸. The habit of swallowing fish gallbladder to cure chronic diseases exists in the rural areas of southeastern and southern China, including Hong Kong and Taiwan. Due to differences in socio-cultural beliefs³carp fish (Labeo rohita), known as Rohu in India, is commonly consumed as aphrodisiac. Our observations from this patient were similar to that of previous reports. The patient swallowed raw bile of grass carp in the belief that her cough would be alleviated and developed anuria due to ARF instead. The initial symptoms⁸ were similar to that of gastroenteritis, such as abdominal pain, nausea, vomiting and watery diarrhea, occurring 0.5-4 h after consumption of the offending agent. Edema, oliguria, or anuria may occur within 2–3 days after poisoning, sometimes even as late as 3–6 days, as reported by some authors⁸. Remarkable increase in urea, serum creatinine, urine-NAG with non-glomerular proteinuria was observed in our patients. ARF caused by fish gall bladder consumption have been reported in several case series,^{9,10} with a morbidity rate of 55–100% and overall mortality rate of 91.7% in all gall bladder consumers. Although some reports have showed improvement of renal function within 2–3 weeks¹¹, the recovery time was 5 weeks in our patient. Raised liver enzymes or jaundice occurs in 75–87% of patients.⁹ Jaundice usually occurs on day 2 to 3, but can occur as late as day 5. Liver and kidney damage often occur simultaneously¹² and mild hepatitis caused by carp gallbladder is usually self-limited. Our patient's liver function recovered on day 19, which was consistent with previous reports. These patients can also die of fulminant hepatic failure.³ Other manifestations include cardiac complications (palpitations, hypertension, and myocardial damage) and neurological involvement (convulsion and coma), which varies individually.

The exact mechanisms of the toxic effects are not clear. However, necrosis of the renal proximal tubules (PT) may play an important role in the development of ARF.¹³ Degeneration of the renal tubular epithelial cells, dilatation of some tubular lumen, epithelial shedding, and segmental naked basement membrane formation was revealed by renal biopsy (shown in Fig. 2). The pathological severity correlated with clinical symptoms of sudden oliguria or anuria as well as increased urine NAG, a marker of tubular injury.¹⁴ In an animal study,¹³ glomerular filtration rate (GFR) decreased 24 h after ingestion of 0.3 mL of grass carp bile, suggesting that nephrotoxic substances might cause cellular damages by inhibiting the cytochrome oxidase, promoting calcium influx, and inducing

lysosome membrane instability.⁹ In a porcine study,¹⁵ Choi K, et al. suggested that nitric oxide (NO) generation and the phospholipase C pathway affect the release of renal dipeptidase (RDPase) from the PT which may be involved in the development of ARF in vivo after carp bile ingestion. The significance of the interstitial edema and inflammation characterized by infiltration of lymphocytes and monocytes observed in the renal biopsy can be explained by the serious inflammatory response to cell necrosis induced by increased cell membrane permeability and the resultant enzyme release. In addition to renal toxicity, fish bile may cause a series of inflammatory responses evidenced by increased inflammatory mediators and cytokines, eventually leading to pathological changes of cell degeneration and necrosis. Another study¹⁶ showed that plasma endothelin levels correlated with the severity of carp gallbladder poisoning. In our patient, c-reactive protein, procalcitonin and serum amyloid A were increased. However, the role of inflammation in carp gallbladder toxicity requires further elucidation.

In our patient, we also found decreased leukocytes, neutrophils, lymphocytes, hemoglobin and platelets; and sinus bradycardia in the ECG, which to our knowledge has not been previously reported. In 1993, Lim PS, et al.¹⁷ reported a case of sinus bradycardia that persisted for 2 days even after atropine was administered for symptomatic relief. Some authors have demonstrated experimentally that bile acids can affect the cardiovascular functions of animals both *in vivo* and *in vitro*, inducing bradycardia and systemic hypotension.¹⁸ It was also found¹⁸, *in vitro*, that the bile salts caused a potent hemolysis. In our patient, the decreased values of leukocytes, neutrophils, lymphocytes, and platelets resolved spontaneously, though anemia lasted for more than 3 weeks. Sinus bradycardia persisted for 2 days. We hypothesize that there may be an undiscovered hematotoxin or cardiotoxin in fish gallbladder, which needs further investigations.

There is no specific antidote for carp gallbladder poisoning. However, gastric lavage is necessary in the early 72 h using 1–5% soda or warm water. Protecting the gastric mucosa by administering raw eggs and cattle milk may be effective¹¹. Conventional hemodialysis is an effective temporary treatment for anuria or oliguria. Liu Z, et al.¹⁹ revealed continuous venovenous hemodiafiltration could effectively remove inflammatory mediators, metabolites, bilirubin and toxins in severe cases while maintaining homeostasis. Supportive treatment, like gastric mucosal protectant and liver protectant are equally important for targeted organ protection. Our patient did not receive gastric lavage like 1–5% soda or warm water due to the earlier misdiagnosis of gastroenteritis and delayed presentation at our center after poisoning. Nutritional support was provided appropriately and was tolerated in our patient, which aided her clinical recovery.

Conclusion

In summary, the diagnosis of this disease is primarily clinical and dependent on the history, since there are no special laboratory tests to confirm the disease. General practitioners, emergency physicians, and gastroenterologists should be mindful of the toxic complications of raw grass carp gallbladder. Educating the public regarding the toxic effects of raw gall bladder consumption could be helpful in reducing the incidence of its toxicity, especially in the rural areas.

Abbreviations

IV = intravenous injection; ECG = electrocardiogram; HR = heart rate; ALT = alanineaminotransferase; AST = aspartate aminotransferase; ARF = acute renal failure; CT = computer tomography; NAG = N-acetyl- β -D-glucosaminidase; PT = proximal tubules; GFR = glomerular filtration rate; NO = nitric oxide; RDPase = renal dipeptidase.

Declarations

Ethics approval and consent to participate

The Ethics Committee of Wenzhou People's Hospital has approved the publication of this report. The patient's informed consent has been obtained.

Consent for publication

This study has been approved by the patient for publication.

Availability of data and materials

All data analysed during this study are included in this published article and its supplementary information files.

Competing interests

None

Funding

None

Authors' contributions

LNZ wrote the article. SSD performed renal biopsy and conventional hemodialysis on the patient. SZZ collected the data. WH approved the final version of the manuscript.

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Figures

Figure 1

The initial ECG 25mm/s, 10mm/mv showing sinus bradycardia .(HR 46bpm) ; QRS:84ms, QTC:389ms, Q-T:445ms, P-R:166ms.

Figure 2

Light microscopy HE(×200) of the renal biopsy showing: A (white arrow) degeneration of renal tubular epithelial cells, dilatation of some tubular lumen, epithelial shedding, segmental naked basement membrane formation; B (black arrow) renal interstitial edema with a small amount of inflammatory cell infiltration; C (blue arrow) intact glomerular capsule wall, no obvious thickening of cystic wall, mesangial cells and matrix hyperplasia, capillary loops open.