CASE REPORT

Catfish Bite Case Report

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Although catfish are found worldwide and commonly consumed in the southern United States, fatal infections from catfish are rare. *Edwardsiella tarda* is a bacterium known to cause gastrointestinal distress most commonly, but extraintestinal infections are a rarely considered danger for those acquiring, preparing, and consuming aquatic animals. Susceptible to all gram-negative active antibiotics, it is easily treated except in immunocompromised hosts, such as those with malignancy, diabetes, and hepatic dysfunction.

Keywords: *Edwardsiella tarda*, freshwater, infection, mortality, immunocompromised

Introduction

Fishing is a common recreational and sporting activity, and the consumption of fish and other aquatic animals is found throughout the world.¹ Catfish are found worldwide and are a common fish consumed in the southern United States,² including the channel catfish, *Ictalurus punctatus*; blue catfish, *Ictalurus furcatus*; and flathead catfish, *Pylodictis olivaris*.^{3,4} Dangers involved in catfish handling are usually found in the puncture from the barb^{2,3,5} but can also be seen in a bite,⁶ especially by the flathead, which has villiform teeth.⁴ This can lead to potential exposure to pathogens, which, in human infections, can lead to a variety of signs and symptoms ranging from mild to severe.^{6–9} One such organism is *Edwardsiella tarda*, which can be found worldwide.¹⁰ The following case report describes a patient who presented with an extraintestinal infection from *Edwardsiella tarda* that resulted in his hospitalization and eventual death.

Case report

A 58-y-old man presented to the emergency department in the morning, 18 h after receiving a catfish “bite” to the left hand while cleaning fish in early August. The patient did not specify which type of catfish “bit” him, but he presented to an emergency department in the mid-south

of the United States stating he was injured locally, which limits species to the channel, blue, and flathead catfish in freshwater. The patient reported a mild ache and swelling in his hand and decreased appetite and a feeling of being unwell since the incident. The patient had a history of hypertension, chronic obstructive pulmonary disease, seizure disorder, lung cancer, diabetes, tuberculosis, alcohol abuse (~4 710 mL [24 oz] beer per day), cocaine abuse, and tobacco (2 pack per day) abuse. The patient reported an allergy to aspirin and morphine with a questionable sulfa allergy in his chart. On presentation the patient’s vitals were as follows: weight 54 kg, temperature 36.5°C, blood pressure 144/95 mm Hg, pulse 95 beats·min⁻¹, respiration 20 breaths·min⁻¹, and oxygen saturation 98% on room air. The patient appeared in no acute distress, with scleral icterus and edema and erythema to his left hand and forearm; the remainder of the examination was unremarkable. Given the patient’s examination results and history of diabetes and polysubstance abuse, laboratory tests and blood cultures were obtained, as was a radiograph of the hand. The patient was started on vancomycin 1 g every 12 h and Zosyn 3.375 g every 6 h with the first dose given in the emergency department.

Laboratory values are presented in [Table 1](#), Day 1.

Imaging showed dorsal soft-tissue swelling but no acute fracture, dislocation, osseous abnormality, or radiopaque foreign body.

The patient was admitted to the intensive care unit (ICU) and placed on delirium tremens precautions. Within hours, he began to have decreased mentation, hypotension, tachycardia, and tachypnea. The patient

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Submitted for publication January 2019.

Accepted for publication April 2019.

Table 1. Laboratory values

Laboratory test	Day					
	1	2	3	4	5	6
Chemistry						
Sodium (mmol·L ⁻¹)	132	131	134	131	132	129
Potassium (mmol·L ⁻¹)	4.3	4.3	5.8	6.1	6.7	6.0
Chloride (mmol·L ⁻¹)	96	101	100	98	95	95
Carbon dioxide (mmol·L ⁻¹)	12	11	6	9	8	7
Urea nitrogen (mg·dL ⁻¹)	12	18	11	6	6	5
Glucose, serum (mg·dL ⁻¹)	46	65	17	112	161	107
Calcium (mg·dL ⁻¹)	7.7	6.0	6.7	7.3	6.9	7.8
Creatinine (mg·dL ⁻¹)	1.27	2.25	2.05	1.56	1.48	1.36
Albumin (g·dL ⁻¹)	2.0	1.5	1.9	1.9	2.3	3.5
Total bilirubin (mg·dL ⁻¹)	4.0	5.6	7.2	10.4	8.8	11.1
Alkaline phosphatase (unit·L ⁻¹)	190	49	60	177	174	534
AST (SGOT) (unit·L ⁻¹)	251	766	7524	8850	6852	15,108
ALT (SGPT) (unit·L ⁻¹)	99	160	978	1357	846	1,686
Anion gap	>20	19	>20	>20	>20	>20
Glomerular filtration rate (GFR) (mL·min ⁻¹ ·1.73 m ⁻²)	>60	36	41	56	59	>60
Creatine kinase (CK) (unit·L ⁻¹)	2458	1979			91,212	187,140
Lactic acid (mmol·L ⁻¹)	16.3	12.2	20.3	16.4	21.2	20.8
International normalized ratio (INR)	2.4	3.9	4.4	5.2	>10.0	4.0
Hematology						
White blood cells (thou·μL ⁻¹)	1.7	4.3	2.8	3.3	3.4	2.7
Hemoglobin (g·dL ⁻¹)	11.4	9.0	6.0	8.9	5.4	10.1
Hematocrit (%)	35.3	28.0	19.8	27.8	17.6	32.6
Platelet count (thou·μL ⁻¹)	36	40	18	43	12	69
Band neutrophil (%)	21			6		2
Segmented neutrophil (%)	62.0	73.0	66.3	52.0	41.0	40.0
Lymphocyte (%)	9.0	4.0	21.5	26.0	48.0	21.0
Monocyte (%)	4.0	6.0	11.2	11.0	10.0	28.0
Eosinophils (%)	2	10	1	1	1	9
Blood gas						
I-Stat pH, arterial	7.29	7.13	7.14	7.23	7.10	6.95
I-Stat PCO ₂ , arterial (mm Hg)	22	25	23	27	22	18
I-Stat PO ₂ , arterial (mm Hg)	122	151	149	132	81	107
I-Stat HCO ₃ , arterial (mmol·L ⁻¹)	11	8	8	11	7	4
I-Stat base excess (mmol·L ⁻¹)	-16	-21	-21	-16	-23	-28
I-Stat O ₂ saturation (%)	98	99	99	98	91	94
I-Stat F _I O ₂ (%)		40	40	40	40	50

AST, aspartate aminotransferase; SGOT, serum glutamic oxaloacetic transaminase; ALT, alanine aminotransferase; SGPT, serum glutamic-pyruvic transaminase; PCO₂, partial pressure of carbon dioxide; PO₂, partial pressure of oxygen; HCO₃, bicarbonate; O₂, oxygen; F_IO₂, fraction of inspired oxygen.

was intubated with an arterial blood gas presented in Table 1. Clindamycin was added and 3 vasopressors were started by the afternoon of day 2. Gram-negative rods were grown in culture by that evening.

Infectious disease was consulted and changed the patient's antibiotics to meropenem and ciprofloxacin owing to the increased risk for *Aeromonas* with the patient's apparent liver disease resulting from ethanol abuse and hepatitis C antibody-positive status. Vancomycin was changed to linezolid for gram-positive

coverage to attempt to lessen nephrotoxicity, and clindamycin was stopped. Acyclovir was also started for concern for disseminated herpes simplex virus when he began to show blistering and sloughing of the skin of his left upper extremity by the early morning after admission.

Orthopedics was consulted to assess for compartment syndrome and potential necrotizing fasciitis. Compartment pressures obtained in the hand were 17 to 18 mm Hg, and muscle biopsy was negative. The patient later developed bilateral knee effusions, which were concerning for septic

arthritis. Aspiration for each knee showed 878 to 5317 white blood cells·hpf⁻¹ with 8 to 52% segmented neutrophils and 48 to 88% lymphocytes.

Cardiology was consulted on admission to the ICU for suspected cardiogenic shock management secondary to sepsis. Bedside echocardiogram showed an ejection fraction of 15 to 20%, which improved to 36 to 40% after dobutamine initiation. There was moderate global hypokinesis, normal diastolic function, and no vegetations identified on the echocardiogram.

Nephrology was consulted for acute renal insufficiency, and the patient was started on a bicarbonate drip and continuous renal replacement therapy.

Neurology was consulted because the ICU team noted seizure-like activity in the patient. An electroencephalogram was obtained, showing moderate to severe generalized slowing consistent with a generalized disturbance of cerebral function as seen in toxic, metabolic, postanoxic, multifocal, or diffuse structural abnormalities. No electrographic seizures or nonconvulsive status epilepticus were seen.

During the course of the admission, the patient was noted to be hypothermic to 29°C with inability to maintain blood pressure despite increased vasopressor use. Examination showed continued edema of the upper and lower extremities. The patient also noted sloughing to the extremities with flaccid bullae formation and ischemia in the fingers and toes.

Before the patient died, laboratory tests were obtained; the results are presented in Table 1, Day 6. Blood cultures and knee aspirate showed *Edwardsiella tarda*, which were pansensitive.

With worsening septic shock, the patient was made do not resuscitate by the family and died 6 d after his initial injury.

Discussion

Edwardsiella tarda is a facultative anaerobic gram-negative bacillus in the Enterobacteriaceae family.¹¹ It can be found in channel fish, eels, flounder, largemouth bass, and rainbow trout and infects fish, amphibians, reptiles, and mammals.^{9,12} Found in a variety of fresh or brackish water, mud, and the intestines of fish and other marine animals, *Edwardsiella tarda* is spread through feces.^{11,12} Reports of fatal infections in freshwater catfish in Arkansas have been reported with extraintestinal manifestations such as emphysematous putrefactive disease.¹³ In humans it is known to cause a variety of illnesses, including gastroenteritis most commonly and rarely extraintestinal manifestations,^{11,12} which usually occur in immunocompromised hosts such as those with malignancy, diabetes mellitus, or hepatic dysfunction.^{8,10,14}

Extraintestinal symptoms can range from mild to severe, depending on which organs are infected. Reports have included cellulitis, cholecystitis, intra- and extrauterine pelvic infections, liver abscess, meningitis, mycotic aneurysms, osteomyelitis, peritonitis, and salpingitis.^{4,10–12,14} Given the wide range of infections possible, there is no set of signs or symptoms that are particularly associated with *E tarda*.

E tarda is the most common gram-negative bacteria causing secondary infection in catfish injuries.⁶ In >80% of cases caused by the bacteria, it is cultured from stool and associated with gastrointestinal illnesses in healthy and immunocompromised individuals who have consumed contaminated fish. Unfortunately, the precise role is not known because it is also found in the stool of those without gastrointestinal symptoms and with other known pathogens.¹¹ *E tarda* is uniformly susceptible to all antibiotics with gram-negative activity,¹² which is consistent with our culture and sensitivity.

In a literature review from 1968 to 2013, the mean age of patients was 61 y (ranging from 2 d to 101 y); 61% of patients were male.¹⁰ Cancer was present in 38% of patients, with hepatobiliary cancer in 21% and liver cirrhosis in 17%. The overall mortality rate was 45%⁶ despite appropriate antimicrobial therapy but increased to 61% among those with soft-tissue infections.¹⁰ There was no significant difference between those who survived and those who did not when looking at age, sex, complications, geographic area, or season of onset. Liver cirrhosis, including alcoholic cirrhosis, was considered an independent risk factor for death. The period from onset of disease to death was a median of 8 d (20 h–61 d).¹⁰

Catfish injury treatment depends on injury type and severity. The injury site should be examined for abrasions, lacerations, and puncture wounds, and a neurovascular examination should be performed. Injuries can be caused both by the fish and environmental hazards such as hooks, bacteria and viruses in the water and fish, metal, and vegetation. Evaluation for foreign bodies such as spines and hooks should be undertaken with radiographs, initially x-rays. Flushing the wound with normal saline and debridement of necrotic tissue should be undertaken with the administration of antibiotics. Any foreign bodies found should be surgically removed, and wounds should undergo closure by secondary intention or delayed primary closure. Tetanus prophylaxis, if indicated, should be administered. Wounds in freshwater should be treated with empiric antibiotic coverage because of the prevalence of both gram-positive and gram-negative bacteria. Sites of envenomation from catfish spines should also be immediately immersed in hot water (45°C) for 30 to 90 min because toxins tend to be heat-labile.^{1,4,8}

Acknowledgments: I thank Dr. Brian Hawkins for his assistance both with the patient and review of this paper.

Financial/Material Support: None.

Disclosures: None.

References

1. Ajmal N, Nanney LB, Wolfort SF. Catfish spine envenomation: a case of delayed presentation. *Wilderness Environ Med.* 2003;14(2):101–5.
2. Thalaspitiya SP, Wijerathne BT, Siriwardhana NP. Fresh-water catfish envenoming in a tropical country. *Wilderness Environ Med.* 2017;28(3):259–66.
3. Blomkalns AL, Otten EJ. Catfish spine envenomation: a case report and literature review. *Wilderness Environ Med.* 1999;10(4):242–6.
4. Karr CR, Plikaitis C, Germino KW, Nakanishi AK. Catfish noodling forearm injury requiring urgent surgical treatment. *Wilderness Environ Med.* 2014;25(1):75–9.
5. Taylor DM, Ashby K, Winkel KD. An analysis of marine animal injuries presenting to emergency departments in Victoria, Australia. *Wilderness Environ Med.* 2002;13:106–12.
6. Kaar CR, Nakanishi AK. Recreational and commercial catfishing injuries: a review of the literature. *Wilderness Environ Med.* 2017;28(4):348–54.
7. Haddad V, de Souza RA, Auerbach PS. Marine catfish sting causing fatal perforation in a fisherman. *Wilderness Environ Med.* 2008;19(2):114–8.
8. McKinstry DM. Catfish stings in the United States: case report and review. *J Wilderness Med.* 1993;4(3):293–303.
9. Marsh PK, Gorbach SL. Invasive enterocolitis caused by *Edwardsiella tarda*. *Gastroenterology.* 1982;82(2):336–8.
10. Hirai Y, Asahata-Tago S, Ainoda Y, Fujita T, Kikuchi K. *Edwardsiella tarda* bacteremia. A rare but fatal water- and foodborne infection: review of the literature and clinical cases from a single centre. *Can J Infect Dis Med Microbiol.* 2015;26(6):313–8.
11. Slaven EM, Lopez FA, Hart SM, Sanders CV. Myonecrosis caused by *Edwardsiella tarda*: a case report and case series of entristestinal *E. tarda* infections. *Clin Infect Dis.* 2001;32(10):1430–3.
12. Ebisawa KF, Nishimura S, Yamamoto S, Ohji G, Iwata K. Mycotic aneurysm caused by *Edwardsiella tarda* successfully treated with stenting and suppressive antibiotic therapy: a case report and systematic review. *Ann Clin Microbiol Antimicrob.* 2018;17(1):21.
13. Meyer FP, Bullock GL. *Edwardsiella tarda*, a new pathogen of channel catfish (*Ictalurus punctatus*). *Appl Microbiol.* 1973;25(1):155–6.
14. Yamanoi K, Yasumoto K, Ogura J, Hirayama T, Suginami K. A case of pelvic abscess caused by *Edwardsiella tarda* followed by laproscopic resection of a hematoma derived from caesarean section. *Case Rep Infect Dis.* 2018;2018:1–6.