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CASE REPORT

Severe Acute Bacterial Skin and Skin Structure Infection Following Fish Spine Injury: A Case Report and Literature Review

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Abstract: This study presents a rare case of severe acute bacterial skin and soft tissue infection (ABSSSI) following freshwater fish spike injury in a 73-year-old man. Within 24 hours of sustaining the wound, the patient developed septic shock and progressive necrotizing fasciitis. Despite early administration of broad-spectrum antibiotics and intensive care, his condition deteriorated, necessitating below-the-elbow amputation on hospital day four. Metagenomic next-generation sequencing (mNGS) identified Aeromonas veronii as the causative agent. Although the patient showed temporary improvement postoperatively, he later developed hospital-acquired pneumonia and succumbed to complications two months later. This case highlights the potential severity of ABSSSIs caused by aquatic pathogens, particularly in immunocompromised individuals. mNGS provides a comprehensive pathogen snapshot within hours of sample receipt, enabling timely refinement of empiric regimens prior to antimicrobial susceptibility testing availability. Early surgical intervention is essential to control infections and improve clinical outcomes.

Keywords: acute bacterial skin and soft tissue infection, necrotizing fasciitis, fish-related human infection, *Aeromonas veronii*

Introduction

Engaging in fish-related activities, including fishing, food preparation, and retail handling, poses the risk of fish spike injuries. Reports indicate that such injuries may introduce bacterial pathogens, leading to acute bacterial skin and soft tissue infections (ABSSSIs).[1,2] Severe cases of ABSSSIs resulting from fish spikes often require hospitalization for proper management.

Bacterial infections caused by fishbone injuries that lead to necrotizing fasciitis (NF), particularly those associated with *Vibrio, Aeromonas*, and *Klebsiella species*, are rare but aggressive forms of necrotizing soft tissue infections (NSSTIs). These infections are characterized by rapidly progressive necrosis of the subcutaneous tissues. The mortality rate of NF ranges from 25.3% to 73%. Early recognition of NF, coupled with emergent surgical debridement and timely administration of appropriate antibiotics, significantly improves survival and clinical outcomes. Notably, empirical antibiotic therapy should be initiated within 24 hours if *Vibrio vulnificus* infection is suspected, as early intervention has been shown to reduce mortality.[3,4] This case report describes the clinical presentation and diagnostic workup of a patient with severe ABSSSI following a fish spike injury. In addition, we explored the optimal empiric antibiotic selection and timely surgical intervention to prevent poor outcomes in such patients. Furthermore, we provide an overview of the classification systems, diagnostic approaches, and therapeutic interventions for severe ABSSSIs caused by community-acquired fish spike injuries.

Case Presentation

A 73-year-old man presented to the emergency department with escalating pain and swelling in his right hand, extending to the elbow, that had developed over the past day. The day before admission, while filleting a freshwater perch at home, he sustained

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a puncture wound on the dorsum of his right hand between the fourth and fifth fingers. Within 24 hours, the wound site became intensely painful and developed blisters.

Due to worsening pain, progressive swelling extending to the elbow, and shortness of breath, the patient was urgently referred to our hospital. His medical history included chronic renal insufficiency, hypertension, and obstructive pulmonary disease.

On admission, he exhibited signs of septic shock, including hypotension (84/54 mmHg) and metabolic acidosis (pH 7.074, BE −19.2, lactate 6.94 mmol/L). Immediate fluid resuscitation was then initiated. The affected limb showed pronounced swelling with large hemorrhagic bullae (see Figure 1). Laboratory tests revealed a white blood cell count of 10.01×10^9/L, platelet count of 50×10^9, C-reactive protein (CRP) of 147.4 mg/L (normal <5 mg/L), and procalcitonin (PCT) of 154 ng/mL (normal <0.05 ng/mL). Cardiac ultrasonography revealed no valvular abnormalities, and arterial Doppler imaging ruled out deep arterial occlusion in the right upper limb (Figure 1).

The patient was intubated and transferred to the emergency intensive care unit (EICU), where empirical antibiotic therapy was initiated with meropenem (1 g IV every 8 h) and vancomycin (0.5 g IV every 12 h) was initiated. Hemodynamic support with norepinephrine and dobutamine was administered guided by invasive intravascular pressure monitoring. Owing to persistent metabolic disturbances and acute kidney injury superimposed on chronic renal insufficiency, continuous venovenous hemodiafiltration was initiated.

At approximately 24 h post-admission, laboratory results showed a decline in white blood cell count to 2.6×10^9/L and platelet count to 19×10^9/L, while PCT increased to 250 ng/mL and CRP to 166 mg/L. Gram staining of the aspirated blister fluid revealed gram-negative bacilli. Although the patient had a history of exposure to freshwater fish, the potential for cross-contamination between freshwater and marine fish necessitates a high index of suspicion for *Vibrio vulnificus* infection.[5] Based on clinical experience, *V. vulnificus* was initially suspected. Consequently, empirical antimicrobial therapy was adjusted to include ceftriaxone (2 g IV once daily), levofloxacin (0.5 g IV once daily), and minocycline (100 mg orally once daily). However, metagenomic next-generation sequencing (mNGS) of the blister fluid performed

Figure 1 The process of the skin injury.
Notes: Below-elbow amputation of the right arm performed on the 5th day after injury.

48 hours after admission identified *Aeromonas veronii* as the causative pathogen. Blood mNGS was performed using the BGISEQ-500 platform (BGI, China). Following extraction of microbial nucleic acids from 3mL EDTA-blood, libraries were sequenced to a depth of 20M reads. Pathogens were identified using BGI's Dr.Tom pipeline with RefSeq/GenBank databases, requiring >10 RPM for bacteria/fungi and >5 RPM for viruses. Negative controls showed no contamination.

Given that many aerobic bacteria produce β-lactamase and may exhibit multidrug resistance,[6–9] anti-infective therapy was continued with meropenem (1 g every 8 hours), levofloxacin (0.5 g once daily), and minocycline (100 mg once daily). Three days later, *Aeromonas veronii* was identified from blister cultures by matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS). Antimicrobial susceptibility testing was performed using both Kirby-Bauer disk diffusion and broth microdilution methods (following CLSI M45 guidelines). The isolate demonstrated susceptibility to piperacillin-tazobactam and gentamicin, as well as to fluoroquinolones, carbapenems, aztreonam, and cefotaxime. Antibiotic therapy was promptly adjusted based on susceptibility results. CRP levels decreased to approximately 87 mg/l and PCT dropped to 122.9ng/mL on the 3rd day after admission.

Despite appropriate antimicrobial therapy, the affected limb remained swollen, pulseless, and cold. Advanced imaging, including arterial enhancement and 3D reconstruction, demonstrated poor visualization of the radial and ulnar arteries. Rapidly progressing NF and tissue necrosis necessitated below-the-elbow amputation on hospital day four (post-injury day five). Following surgery, the antibiotic regimen was continued. The patient's respiratory function improved, septic shock resolved, CRP declined to 18 mg/L, and PCT decreased to 22.5 ng/mL. The surgical site healed well after the amputation. Unfortunately, the patient later developed severe hospital-acquired pneumonia and succumbed to complications two months later.

Methods

For our review of the English-language literature on fish spine injuries complicated by bacterial infections since 1980, we used MEDLINE and Index Medicus to identify publications concerning fish spine and bacterial infections cases complicated by infection. No restrictions were placed on water salinity; cases from marine, freshwater, and estuarine environments were all included. A bibliographic search of pertinent articles was also used.

Results and Discussion

Bacterial infections following fish spine injuries are common and occur in freshwater and saltwater environments.[3,4] The severity of the infection depends on the bacterial species involved. In severe cases, early antibiotic administration combined with surgical intervention significantly improves patient prognosis.

In immunocompetent individuals, common pathogens associated with minor aquatic injuries include *Staphylococcus aureus, Streptococcus spp., Erysipelothrix rhusiopathiae*, and *Pseudomonas aeruginosa*. Severe infections are often caused by *Aeromonas hydrophila, Vibrio vulnificus, Edwardsiella tarda, Chromobacterium violaceum*, and *Photobacterium damsela*.[3,4,10] Less frequently encountered emerging pathogens include community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) and group A *Streptococcus* species (Table 1). Effective management of ABSSSIs related to fish-sting injuries requires prompt empirical antibiotic therapy and surgical intervention in many cases.

In this case, the patient presented with a history of a freshwater fish bone injury. Initial differential diagnosis included *Aeromonas hydrophila, Streptococcus iniae*, and *Chromobacterium violaceum*. Although *Streptococcus* and *Staphylococcus aureus* are not typically associated with aquatic exposure, they were also considered and empirical antimicrobial therapy with meropenem and vancomycin was initiated.

By hospital day 2, the patient developed an increased number of tense pustules on the dorsum of the right hand, along with rising inflammatory markers. Despite the reported exposure being to freshwater fish, cross-contamination with marine species could not be excluded. Given the patient's history of aquatic exposure, rapid clinical deterioration, hemorrhagic bullae, skin necrosis, and sepsis, ceftriaxone, levofloxacin, and minocycline were added to cover *V. vulnificus*.

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Table 1 Pathogen, Clinical Features, and Suggested Therapeutic in ABSSSI by Fish Spikes

Pathogen	Common Severity	Clinical Manifestations	Antimicrobial Choices	Common Types of Fish Injuries
Vibrio spp.	Severe infection	Cellulitis, hemorrhagic bullae, ulcers, necrotizing fasciitis; septicemia with high fatality	Prior choice: ciprofloxacin or levofloxacin Additional options: co-trimoxazole, or third-generation or fourth-generation cephalosporins	Marine fish
Aeromonas spp.	Severe infection	Cellulitis, pyodermasfuruncles, necrotizing infections	Prior choice: ciprofloxacin or levofloxacin Additional options: co-trimoxazole, or third-generation or fourth-generation cephalosporins	Marine and Freshwater fish
Edwardsiella tarda	Severe infection	Pyodermas, necrotizing infections, myonecrosis	Prior choice: ampicillin or cephalosporins (cefazolin or cefazidime). Additional options: aminoglycosides or fluoroquinolones	Marine and Freshwater fish
Photobacterium damselae subsp. Damselae	Severe infection	Soft tissue infections, especially necrotizing fasciitis	Prior choice: high-dose ceftazidime (or cefotaxime) and tetracycline. Additional options: fluoroquinolone	Marine fish
MRSA	Mild to severe infection	Cellulitis, abscesses, necrotizing fasciitis	Prior choice: vancomycin, linezolid, daptomycin. Additional options: teicoplanin, clindamycin, minocycline, co-trimoxazole (trimethoprim/ sulfamethoxazole)	Marine and Freshwater fish
Group A streptococci	Mild to severe infection	Erysipelas, cellulitis, or necrotizing fasciitis. Typical not life-threatening; 80% of patients recover	Prior choice: for NF, high-dose penicillin G intravenously and clindamycin, or cephalosporin Additional options: clindamycin, vancomycin, linezolid, daptomycin	Marine and Freshwater fish
Streptococcus iniae	Mild to severe infection	Impetigo and cellulitis	Prior choice: penicillin, Additional options: cephalosporins, macrolides and trimethoprim-sulfamethoxazole	Marine and Freshwater fish
Chromobacterium violaceum	Mild to severe infection	Cellulitis, pustules, ulcers with black necrotic bases and bluish purulent discharges	Prior choice: aminoglycoside or fluoroquinolone or tetracyclines or trimethoprim-sulfamethoxazole. Additional options: imipenem, aminoglycosides	Marine and Freshwater fish
Erysipelothrix rhusiopathiae	Mild to severe infection	Erysipelas, usually resolves within three to four weeks	Prior choice: penicillin G, or amoxicillin. Additional options: parenteral third-generation cephalosporins, fluoroquinolones	Marine fish
Pseudomonas aeruginosa	Mild to severe infection	Rashes with necrotic areas, edema beyond the visible rash.	Prior choice: antipseudomonal beta-lactam, combined with aminoglycoside. Additional options: antipseudomonal beta-lactam with fluoroquinolone	Freshwater fish

Abbreviations: MRSA, methicillin-resistant *Staphylococcus aureus*; NF, necrotizing fasciitis.

Pathogen-Specific Clinical Manifestations and Antibiotic Therapy

Aeromonas hydrophila

Species of the genus *Aeromonas* are facultative anaerobic gram-negative bacilli that are widely distributed in soil and various natural water systems.[11] Although these organisms are found in brackish water, saltwater exposure is not a typical source of *Aeromonas*-related skin infections. *Aeromonas* infection was also found in fish.[12–14] Most reported cases of *Aeromonas* infection originate in tropical and subtropical regions, with relatively fewer cases documented in temperate zones.[6]

Clinically significant species associated with human infections include *Aeromonas hydrophila, Aeromonas caviae*, and *Aeromonas veronii*.[6,15] Soft tissue infections caused by *Aeromonas* species typically occur following superficial wounds or injuries inflicted by aquatic organisms.[12–14]

Aeromonas-associated cellulitis presents with intense erythema and induration at the site of the injury. It is the most common form of *Aeromonas*-induced soft tissue infection and frequently progresses to suppuration.[16] In more severe cases, infections may escalate to NF, with systemic symptoms emerging within 8–48 hours, particularly in immunocompromised individuals.[17] Delayed recognition and treatment of NF can lead to severe complications including sepsis, limb amputation, or death.[17–22] Necrotizing soft tissue infections caused by *Aeromonas* have a reported mortality rate of 12.5%, which decreases to 7.5% in patients undergoing early intervention.[20]

Prompt diagnosis and timely therapeutic intervention are critical to improving outcomes.[7,23,24] Most *Aeromonas* strains exhibit intrinsic resistance to Penicillins (penicillin, ampicillin, carbenicillin, ticarcillin), First-generation cephalosporins (eg, cefazolin, cephalexin), Second-generation cephalosporins (eg, cefuroxime, cefaclor). This resistance is primarily mediated by chromosomal AmpC β-lactamases and efflux pumps. In contrast, they remain broadly susceptible to Fluoroquinolones (ciprofloxacin, levofloxacin), Third- and fourth-generation cephalosporins (cefotaxime, ceftazidime, cefepime), Aminoglycosides (gentamicin, amikacin), Carbapenems (imipenem, meropenem), Tetracyclines (doxycycline).[22] These organisms commonly produce inducible chromosomal β-lactamases, which may evade detection by standard commercial susceptibility assays. Three main classes of β-lactamases have been identified: class C cephalosporinases, class D penicillinases, and class B metallo-β-lactamases (CphA). Given the evolving resistance patterns, antimicrobial susceptibility testing is essential to inform targeted therapy.[23] While awaiting definitive results, empiric treatment with fluoroquinolones, third-generation cephalosporins, and/or TMP-SMX is generally appropriate. In high-resistance settings, third-generation cephalosporins and/or aminoglycosides may be preferred.

In our case, mNGS of blister fluid performed 48 hours after admission identified *Aeromonas* veronii as the causative pathogen. Given the known β-lactamase production and potential multidrug resistance of *Aeromonas* spp, antimicrobial therapy was continued with meropenem, levofloxacin, and minocycline. Three days later, blister fluid cultures confirmed *A. veronii*, showing susceptibility to piperacillin-tazobactam, gentamicin, fluoroquinolones, carbapenems, aztreonam, and cefotaxime. The antibiotic regimen was optimized following susceptibility testing. But this case exhibits several distinctive features that augment the current literature on fish spine injuries. First, the unprecedented rapidity of disease progression—from puncture to septic shock within 24 hours and limb necrosis necessitating amputation by day 5. Second, the pathogen's paradoxical behavior is notable: despite *A. veronii*'s in vitro susceptibility to carbapenems and fluoroquinolones, clinical deterioration progressed relentlessly under targeted therapy, suggesting unrecognized virulence mechanisms (eg, biofilm formation or toxin persistence).

The mainstay of management of NF includes broad-spectrum antibiotic therapy and urgent surgical debridement. Patients suspected of having NF should undergo immediate surgical consultation as early intervention may prevent the need for amputation. In our case, the affected limb was swollen, pulseless, and cold. Arterial contrast imaging and 3D reconstruction showed poor visualization of the radial and ulnar arteries, indicating vascular compromise. Rapidly progressing NF and tissue necrosis necessitated below-the-elbow amputation on hospital day four. This highlights the necessity of timely surgical intervention in severe ABSSSIs following fish spine injuries. Early recognition, prompt empirical antibiotics, and immediate surgical evaluation are essential to prevent poor outcomes, including limb loss. In this case, despite fulfilling necrotizing fasciitis diagnostic criteria (hemorrhagic bullae + septic shock + leukopenia) at admission, definitive debridement was delayed until day 4. Literature indicates intervention within <12h of shock onset reduces amputation risk by 67%-a window missed due to prioritization of hemodynamic stabilization over limb-salvage surgery.

Vibrio vulnificus

Vibrio vulnificus is primarily found in coastal or estuarine environments with water temperatures above 18°C and salinity levels ranging from 15 to 25 parts per thousand (ppt).[10] Skin and soft tissue infections caused by *V. vulnificus* typically arise from direct exposure to seawater, handling of contaminated seafood, or wounds sustained by marine shellfish.[25,26] Interestingly, reports from Israel describe cases of *V. vulnificus* infection linked to the handling and processing of freshwater fish such as tilapia and carp, suggesting that these species may serve as reservoirs for the pathogen.

Cutaneous *V. vulnificus* infections typically manifest as hemorrhagic bullae.[27,28] The pathogen employs flagellar-driven chemotaxis toward host-derived nutrient gradients (eg, L-serine, oxygen) to penetrate deep soft tissues, evading superficial immune defenses-a virulence mechanism validated by >90% reduced tissue invasion and necrotic spread in murine models using mcpΔ or ΔfliM mutants.[29] This infection follows a fulminant course, rapidly progressing to septic shock with >50% mortality within days.[30,31] Patients with predisposing conditions, such as chronic liver disease, diabetes

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mellitus, or immunosuppression, are at an increased risk of developing severe complications, including NF, septic shock, and death.[4,32] In a matched-pair cohort study (n=154) comparing *V. vulnificus* (n=77) versus non-*Vibrio* (n=77) necrotizing soft tissue Infection, mortality in cirrhotic patients with *V. vulnificus* infection reached 68.2% (versus 28.6% in non-cirrhotics),[32] attributable to hepatic failure impairing bacterial clearance and exacerbating endotoxemia.

Compared to *Aeromonas*-induced soft tissue infections, *V. vulnificus* is associated with higher rates of acute respiratory failure (39.3% vs 3.8%; $p = 0.002$) and intensive care unit admission (50.0% vs 7.7%; $p < 0.001$).[33] Additionally, *V. vulnificus* accelerates the onset of septic shock (median: 24 hours vs 72 hours for MRSA; $p < 0.01$) and necessitates limb amputation in 32% of cases due to rapid necrotic tissue destruction. In contrast, MRSA infections more frequently complicate as necrotizing pneumonia (17% incidence).[34] These findings collectively underscore the uniquely aggressive pathophysiology of *V. vulnificus* relative to both *Aeromonas* and MRSA pathogens.[32,35]

Given the fulminant progression and high mortality of *V. vulnificus* infections, clinicians must maintain heightened suspicion in patients with seawater exposure.[36,37] Antibiotic initiation delays exceeding 6 hours doubled mortality rates (52.9% vs 25.0%).[32] The pathogen demonstrates in vitro susceptibility to a broad range of antimicrobials, including third-generation cephalosporins, beta-lactam/beta-lactamase inhibitor combinations, carbapenems, tetracyclines, aminoglycosides, fluoroquinolones, and other agents such as trimethoprim-sulfamethoxazole or chloramphenicol.[5] Combination antibiotic therapy is recommended in patients with severe infections. Aggressive surgical debridement is crucial to excise necrotic tissue and reduce bacterial burden,[38] as each 1-hour delay in intervention elevated mortality risk by 18%.[32]

Edwardsiella tarda

Edwardsiella tarda is a gram-negative bacillus commonly found in aquatic environments, particularly freshwater ecosystems.[39] HtpG is a biologically active protein that is required for E. tarda to cope with various stress conditions especially that encountered in vivo the host system during infection.[40]

Although extraintestinal infections caused by *E. tarda* are rare, they often manifest as wound abscesses following exposure to contaminated water including saltwater. Those who engage in fishing or handling fish, severe soft-tissue infection caused by *E. tarda*, specifically focusing on myonecrosis and other extraintestinal infections.In case reports of catfish injuries, the most common gram-negative bacteria causing secondary infection was *E. tarda*.[41] A patient sustained a catfish spine puncture in an aquatic setting, resulting in severe soft-tissue infection with local redness, swelling, and pain. The infection progressed to sepsis and necrosis. *E. tarda* was identified as the pathogen.[42]

Clinically, *E. tarda* infections are similar to those caused by *Aeromonas spp., Vibrio vulnificus*, and even typhoid fever.[43] This pathogen frequently induces abscess formation at sites of traumatic wound infections. Hepatic dysfunction has been identified as a significant risk factor for severe complications including extensive myonecrosis and fatal septic shock. *E. tarda* has also been recognized as a potential causative agent of type 3 NF, a rapidly progressive and life-threatening infection with a mortality rate increased to 61.1% (11 of 18) in a meta analysis.[39]

The fatality rate associated with *E. tarda* infections is comparable to that of *V. vulnificus* and severe *Aeromonas* infections, with death occurring within 48 hours in some cases.

Management of severe *E. tarda* infections requires a combination of broad-spectrum antimicrobial therapy and urgent surgical intervention. The pathogen is generally susceptible to a wide range of antibiotics commonly used for gram-negative infections, including beta-lactam agents (such as cephalosporins), aminoglycosides, and fluoroquinolones. However, in cases of NF or septicemia, aggressive surgical debridement is essential to control infection and prevent systemic dissemination. Prompt recognition and early intervention are critical to improve patient outcomes.

Photobacterium damselae subsp. damselae

Photobacterium damselae subsp. damselae is a facultative anaerobic, halophilic, gram-negative rod that belongs to the *Vibrionaceae* family. Initially classified as *Vibrio damselae*, this species was reassigned to the genus *Photobacterium* in 1991, based on phenotypic and genetic characteristics. *P. damselae* subspecies *damsalea* is the most prevalent pathogen between marine fishes.[44,45]

Human infections caused by *P. damselae* primarily result from wound exposure to saltwater or brackish water, particularly during handling of fish and contaminated tools.[46,47] The first documented case of NF caused by *P. damselae* occurred in a 53-year-old male following a stingray tail barb injury to his lower limb in Florida coastal waters. The clinical progression observed includes cellulitis developing within 12 hours post-injury, rapidly progressing to NF within 48 hours, and complicated by septic shock.[48]

Notably, NF associated with *P. damselae* tends to be more severe and has a higher mortality rate than infection caused by *V. vulnificus*.[49] Unlike *V. vulnificus*, which predominantly affects individuals with underlying conditions such as chronic liver disease and diabetes mellitus, *P. damselae* has been reported to cause NF, even in immunocompetent hosts.[48,50] Despite aggressive antibiotic therapy and surgical intervention, some patients with *P. damselae* infection progress to multiple organ failure within hours of symptom onset.

Given its aggressive clinical course, *P. damselae* infections should be treated with empirical broad-spectrum antimicrobial therapy, similar to that used for *V. vulnificus* infections. Most *P. damselae* strains are susceptible to the antibiotics commonly used against *V. vulnificus*. However, antimicrobial therapy alone is often insufficient and early surgical debridement is essential for optimal outcomes.[51] Delayed surgical intervention may significantly increase morbidity and mortality, underscoring the need for the prompt recognition and aggressive management of *P. damselae*-associated infections.

Chromobacterium violaceum

Chromobacterium violaceum is a gram-negative bacillus predominantly found in tropical and subtropical regions, where it exists freely in soil and freshwater environments.[52] Human infections caused by this pathogen are rare and often overlooked in clinical practice, unless patients present with rapidly progressive sepsis, typically characterized by distinctive purplish skin lesions and growth of pigmented colonies in culture.

The primary route of infection involves percutaneous inoculation through skin injuries,[53] such as lacerations followed by exposure to brackish or stagnant water.[54] Fish bites (eg, Tilapia) are a critical route of percutaneous inoculation, as evidenced by cases of bacteremic cellulitis in high-risk patients (eg, 64-year-old diabetic in Taiwan).[55] A review of 132 human C. violaceum cases (1953–2020) revealed predominantly male patients (median age 17.5 years, IQR 5.0–40.0), with 33% having comorbidities or immunodeficiency. Primary entry sites were lower limbs (28.0%), torso (8.5%), and upper limbs (6.8%). Infection also occurred via oral or inhalational exposure to contaminated environments. Median incubation was 4.0 days (IQR 2.0–8.0); disease duration was 17.5 days (IQR 8.0–30.8).[52] The initial manifestations include an ulcerative skin lesion with bluish purulent discharge at the site of injury accompanied by localized swelling, usually affecting the extremities. If left untreated, infection can progress to invasive septicemia within days,[56] particularly in immunocompromised individuals.

Systemic dissemination is often marked by high fever and the appearance of macular skin lesions that can evolve into multiple abscesses, with secondary involvement of deep tissues, such as bones and the liver.[55] Literature reports an incidence of abscesses in internal organs (36.4%).[52]

Chromobacterium violaceum is susceptible to aminoglycosides, fluoroquinolones, tetracyclines, imipenem, and trimethoprim–sulfamethoxazole but exhibits intrinsic resistance to penicillins and cephalosporins. Although uncommon, certain strains have shown resistance to imipenem and aminoglycosides, further complicating treatment. Given the high mortality rate associated with C. violaceum infections, early diagnosis and prompt initiation of appropriate antimicrobial therapy are crucial. Management should include a combination of intravenous antibiotics and aggressive surgical debridement or drainage of purulent abscess collections, to prevent systemic complications.[53,56]

Erysipelothrix rhusiopathiae

Erysipelothrix rhusiopathiae is a facultative anaerobic gram-positive rod that is environmentally resilient and capable of surviving in a variety of conditions.[57] Human infections are primarily associated with occupational exposure, particularly in individuals handling fresh fish or animal products, where minor skin injuries serve as the primary entry point.

Human infection can take one of three forms: a mild cutaneous infection known as erysipeloid, a diffuse cutaneous form and a serious although rare systemic complication with septicaemia and endocarditis. The most common clinical presentation is erysipeloid, a localized cutaneous infection with an incubation period of one–two days. Lesions typically appear on fingers or hands as well-demarcated, tender, violaceous, and edematous plaques. Peripheral extension may occur; however, central desquamation and ulceration are rare. Unlike other bacterial skin infections, suppuration is uncommon, although a vesicular formation may develop. Systemic symptoms are generally absent; however, approximately 10% of patients experience mild constitutional symptoms such as arthralgia and low-grade fever. If untreated, erysipeloid lesions typically resolve spontaneously within three four weeks.[58,59]

Severe or invasive infections, including generalized cutaneous and systemic infections, are rare but clinically significant. These infections most commonly arise as complications of infective endocarditis, which can lead to septicemia, meningitis, arthritis, pneumonia, osteomyelitis, or toxic shock syndrome. Of the 49 reported cases of serious *E. rhusiopathiae* infection, 90% have been associated with presumed or confirmed endocarditis.[60] This infection typically presents with a characteristic erysipeloid cutaneous lesion in 40% of cases and is linked to a high mortality rate (38%).[60] *E. rhusiopathiae* particularly tends to affect structurally damaged, yet native, left-sided valves. Unlike other types of endocarditis, *E. rhusiopathiae* infections have not been linked to prosthetic valves or intravenous drug use. Early recognition and appropriate antimicrobial therapy are crucial to prevent systemic complications.

The treatment of localized *E. rhusiopathiae* infections is typically straightforward, with penicillin-based antibiotics such as penicillin G, ampicillin, amoxicillin/clavulanate, and cloxacillin proving highly effective. *E. rhusiopathiae* is highly sensitive to penicillin but shows resistance to vancomycin. Given that vancomycin is often used in empiric treatment for suspected endocarditis, it is crucial to promptly distinguish *E. rhusiopathiae* from other gram-positive bacteria. This ensures that the appropriate antibiotic therapy can be initiated without delay.

Streptococcus iniae

Streptococcus iniae, a gram-positive β-hemolytic *streptococcus*, is a significant pathogen in farmed fish (eg, tilapia, yellowtail, rainbow trout, and coho salmon) and an emerging zoonotic agent. While primarily causing disease in aquatic species, it also infects captive and wild marine mammals, including Amazon river dolphins, bottlenose dolphins, and Australian dolphin populations.[61] Human infections occur through percutaneous inoculation during handling or processing of fresh fish, leading to invasive clinical presentations. One of the most significant outbreaks occurred in Toronto in 1996, where 11 cases of *S. iniae* invasive infections were linked to handling or preparing farmed fish.[62] Eight patients developed cellulitis after sustaining minor injuries, and four had underlying chronic conditions. The infections were confirmed by culture and matched to *S. iniae* strains isolated from infected tilapia at local aquaculture farms.

Although *S. iniae* infections are relatively rare in humans, they can be clinically significant and severe, particularly in high-risk populations. The most severe cases have been observed predominantly in individuals of Asian descent, often the elderly, with predisposing conditions, such as diabetes mellitus, chronic rheumatic heart disease, or cirrhosis.[61] Soft-tissue infections caused by *S. iniae* frequently present as bacteremic cellulitis, which may progress to endocarditis, meningitis, septic arthritis, sepsis, pneumonia, osteomyelitis, or toxic shock syndrome. Taiwan reported 666 invasive *Streptococcus iniae* infections in adults (2014–2020),[63] revealing aquaculture as a critical exposure route—especially for occupational groups engaged in fish handling. Although mortality rates were not formally calculated, the data highlight elevated lethality when treatment was delayed, disproportionately affecting patients aged ≥65 years, those with underlying conditions (eg, diabetes or cirrhosis), or subjects receiving surgery >24 hours after symptom emergence.

Antimicrobial susceptibility studies have indicated that *S. iniae* is sensitive to a broad spectrum of antibiotics, including aminoglycosides, cephalosporins, macrolides, penicillins, and trimethoprim-sulfamethoxazole.[64] Penicillin remains the treatment of choice for *S. iniae* infections, although empirical antibiotic selection should consider local resistance patterns and the severity of the clinical presentation. Early recognition and appropriate antimicrobial therapy are essential for preventing complications in severe cases.

Group A Streptococcus

Streptococcus pyogenes, a *Group A streptococcus* (GAS), is a well-recognized pathogen capable of causing rapidly progressive skin and soft tissue infections including erysipelas, cellulitis, and NF. Streptococcal NF can develop even after minor skin trauma, with fish fin injury being a notable predisposing factor.[65]

Although erysipelas is a significant clinical entity, it is generally not life-threatening, with approximately 80% of patients achieving favorable outcomes with appropriate antibiotic therapy65. However, in approximately 10% of cases, the infection extends into deeper soft tissue layers, and 2–5% of patients develop systemic complications, such as bacteremia. A 65-year-old previously healthy man sustained an injury to his left hand from a fish bone.[66] Two days later, he developed fever, swelling, and pain, with clinical signs of NF spreading to his upper limb, armpit, chest, and abdomen. Despite aggressive treatment, gangrene developed, leading to amputation of the left arm at the shoulder. After surgery and debridement, the patient fully recovered. GAS was isolated from the tissues.

NF caused by GAS is classified as a type 2, a monomicrobial form of the disease, in which S. pyogenes is the predominant pathogen.[67–69] This infection may have a gradual onset from hours to days following trauma. However, in some cases, it deteriorates rapidly, leading to extensive tissue necrosis and systemic toxicity. Often referred to as "flesh-eating disease", GAS-associated NF can be fatal within 24 h without timely intervention. The mortality rate was significantly higher in patients who developed streptococcal toxic shock syndrome (STSS) (38%) and septic shock (45%).[70] Since delay in recognition and effective treatment of NF caused by invasive group A streptococcus increases the mortality and disability, the early diagnosis and management of this disease are essential for a better outcome. We presented a patient with a severe form of streptococcal NF of the left upper limb in whom amputation was performed as a life saving procedure.

Antimicrobial therapy must be directed at pathogens and used in appropriate doses. After having been repeated, operative procedures are no longer needed when a patient demonstrates obvious clinical improvement, and no fever for 48–72.[71,72] Linezolid and clindamycin, alone or in combination with penicillin, may optimize the treatment of GAS infections by reducing bacterial burden and exotoxin release.[70]

Community-Acquired Methicillin-Resistant Staphylococcus aureus (CA-MRSA)

CA-MRSA has emerged as a major cause of skin and soft tissue infections, including spinal injuries in fish.[73] In a 2-year prospective study at Middlemore Hospital (Auckland, NZ), 60 patients with upper limb fish spike injuries underwent microbial sampling. MRSA was isolated in 8% (4/50) of adequate specimens.[73] Infections typically present as furuncles and are characterized by necrotic follicular infections with subcutaneous tissue involvement.[74–76] Infection may progress to cellulitis or, more commonly, to abscess formation.[77] Clinically, distinguishing CA-MRSA-induced cellulitis from *S. pyogenes*-induced cellulitis can be challenging because of the overlapping presentations.

CA-MRSA is an increasingly recognized cause of NF. In some cases, the infection prognecrotizing fasciitisresses rapidly over several hours, leading to extensive tissue destruction.[78–80] A review of 53 cases in Taiwan found that 38% were caused by S. aureus and 60% were due to MRSA.[34]

A retrospective analysis at Denver Health Medical Center (where CA-MRSA prevalence exceeds 50% among community S. aureus isolates) revealed extremity involvement in all 5 MRSA-positive NF cases identified among 30 NF patients during the study period.[81]

Given the potential severity of CA-MRSA infections, clinicians should maintain a low threshold for expanding antibiotic coverage in cases where the initial empirical therapy is ineffective or if patients develop sepsis. Empirical antimicrobial therapy for any skin or soft tissue infection should include coverage against MRSA (eg, vancomycin or alternative agents with activity against MRSA) as part of the initial regimen. Additionally, cases demonstrating non-susceptibility to glycopeptides (such as vancomycin) have been reported,[82] and novel antibiotics may be utilized for these non-susceptible pathogens.[83] Early surgical evaluation and debridement remain critical management components in patients with necrotizing infections.[84]

Other Bacteria

Group B beta-haemolytic streptococcus (GBS) is increasingly being reported as a causative agent of necrotising soft tissue infections. Reports have documented that the bacteria can also cause invasive disease, including NF and streptococcal toxic shock-like syndrome.[85] GBS is the most important tilapia pathogen in Asia.[86] GBS ST283 strains have been detected in freshwater and marine fish.[87] In humans, handling infected fish can lead to cellulitis, endocarditis, meningitis, and even severe systemic infections.[88]

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Another case of *Plesiomonas shigelloides* bacteremia following a catfish barb injury represents an unusual presentation of a common infection.[89] A healthy male in his early 40s developed sepsis and cellulitis after being injured by a catfish at a freshwater lake. Blood cultures identified *P. shigelloides*, leading to a change from broad-spectrum antibiotics to ciprofloxacin. Although *P. shigelloides* bacteremia typically occurs in immunocompromised individuals due to translocation from the bowel, the venom from the catfish barb exacerbated the severity of the infection.

Given their propensity for chronic manifestations, infections caused by *nontuberculous mycobacteria* and *Shewanella* species were excluded from the present investigation.

Conclusion

Severe ABSSSIs following fish spike injuries can be life-threatening. Clinicians should recognize that only a limited number of common pathogens are responsible for these infections. Although differentiation based on clinical presentation alone is challenging, a thorough history and initial findings, such as impetigo, erysipelas, cellulitis, pyodermas, and necrotizing soft tissue infections, may guide empirical antibiotic selection. In particular, infections caused by *V. vulnificus* warrant a high level of suspicion because of their rapid progression and high mortality.

Early empirical antibiotic therapy that targets the most likely pathogens is critical. Deep infections should prompt immediate surgical evaluations. Aggressive debridement of necrotic tissue until viable tissue is encountered remains the cornerstone for effective NF management. In this case, an earlier surgical intervention might have prevented amputation. Therefore, timely diagnosis and appropriate therapeutic strategies are essential for achieving favorable outcomes.

Ethics and Consent Statements

This retrospective case report was conducted in the Infectious Diseases Department of the First Affiliated Hospital of Guangzhou Medical University. The study protocol was approved by the hospital's ethics committee (Approval No. ES-2025-K125-01), and informed consent for the publication of case details was obtained from the patient's family. Specifically, consent was provided by the patient's son.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis, and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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