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Tilapia Handling-Associated *Streptococcus agalactiae* Meningitis: A Case Series of Three Patients

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Abstract: The study reports three adult males (aged 47, 52, and 64 years) who presented with acute bacterial meningitis within 4 to 24 hours after sustaining tilapia-related puncture wounds. Cerebrospinal fluid (CSF) analysis consistently showed neutrophilic pleocytosis, elevated protein, and decreased glucose. All three CSF cultures yielded *Streptococcus agalactiae* (Group B Streptococcus, GBS) sensitive to penicillin. Blood cultures were positive in one patient. Two patients recovered completely with antibiotic therapy; the remaining patient developed post-meningitic hydrocephalus requiring ventriculoperitoneal shunting. These cases illustrate that percutaneous inoculation from fish spines can serve as a direct portal of entry for GBS into the bloodstream, subsequent crossing of the blood-brain barrier, and meningeal infection. Clinicians evaluating community-acquired meningitis in areas where tilapia is commonly handled should inquire about recent fish-related injuries and consider GBS in the differential diagnosis.

Keywords: *Streptococcus agalactiae*; Group B Streptococcus; Meningitis; Tilapia; Fish spine injury; Case report

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1. Introduction

Group B Streptococcus (GBS), or *Streptococcus agalactiae*, ranks among the leading causes of neonatal sepsis and meningitis worldwide [1]. Adult disease was once thought to be confined to those with diabetes, liver cirrhosis, or malignancy [2]. The epidemiological landscape, however, has shifted. In 2015, Singapore documented a large outbreak of invasive GBS sequence type (ST) 283 infections linked to raw freshwater fish consumption, affecting otherwise healthy adults and producing severe outcomes including meningoencephalitis and septic arthritis [3,4]. Phylogenomic analysis confirmed that human and fish ST283 isolates formed a tight clade, establishing the zoonotic potential of this clone [3]. This event reframed GBS as a zoonotic and foodborne threat rather than simply a commensal of the maternal genital tract.

Tilapia (*Oreochromis spp.*) accounts for a substantial share of global aquaculture output, and GBS is endemic in farmed tilapia populations, causing epizootics of septicemia and meningoencephalitis that translate into major economic losses [5]. Human infections acquired through fish-handling injuries typically manifest as skin or soft-tissue infection, septic arthritis, or osteomyelitis [6,7]; meningitis arising directly from such injuries has been reported only sporadically [2,8].

Between August and November 2024, three adult male patients presented to our institution with acute meningitis within hours of sustaining tilapia spine puncture wounds. All CSF cultures grew GBS. We describe their clinical courses and discuss the implications of this transmission route for both clinicians and public health authorities.

2. Case reports

(1) Case 1. A 52-year-old previously healthy man, employed as a chef, sustained a puncture wound to his right index finger from a tilapia dorsal fin while preparing fish on September 5, 2024. The wound was not disinfected. Twenty hours later, he developed fever (39.6 ℃), headache, photophobia, and confusion. On arrival at the emergency department, he was febrile and disoriented. Neck stiffness was present. Non-contrast cranial CT was unremarkable. Lumbar puncture revealed an opening pressure of 420 mmH_2O. CSF analysis showed a white blood cell count of 8,500/μL (90% neutrophils), protein 4.2 g/L, and glucose 0.2 mmol/L. Gram staining revealed gram-positive cocci in chains. Intravenous ceftriaxone 2 g twice daily and vancomycin 1 g twice daily were started empirically, consistent with current guidelines for community-acquired bacterial meningitis [9]. CSF culture grew S. agalactiae, susceptible to penicillin; blood culture was negative. Vancomycin was discontinued on day 5 once susceptibilities were confirmed, and ceftriaxone was continued for a total of 21 days. The patient's mental status normalized by day 3. At discharge, neurological examination was intact, and follow-up at two months showed no residual deficits.

(2) Case 2. A 64-year-old man with type 2 diabetes mellitus, controlled on oral metformin, worked at a seafood market. On August 27, 2024, he sustained a tilapia spine laceration to his left thumb. No first aid was applied. Twenty-four hours later, his family found him feverish, rigored, and unable to respond verbally. In the emergency department, his temperature was 39.8 ℃, and his Glasgow Coma Scale score was 10 (E3V2M5). Cranial CT showed no acute intracranial abnormality. CSF opening pressure was markedly elevated at 600 mmH_2O. The CSF contained 12,500 white blood cells/μL (95% neutrophils), protein 6.5 g/L, and glucose 0.1 mmol/L. Gram-positive cocci in chains were seen on stain. Empiric ceftriaxone plus vancomycin was initiated [9]. Both CSF and blood cultures returned positive for penicillin-sensitive S. agalactiae. Antibiotics were narrowed to ceftriaxone alone after day 5, continued for 28 days, given the severity of the initial presentation and development of communicating hydrocephalus on follow-up cranial MRI at two weeks. The patient underwent ventriculoperitoneal shunt placement on day 22. At the three-month follow-up, he ambulated independently but exhibited mild cognitive slowing.

(3) Case 3. A 47-year-old man, otherwise healthy, was injured by a tilapia spine on the right middle finger on November 10, 2024, while cleaning fish at home. The wound was rinsed with tap water but not otherwise treated. Approximately 24 hours later, he presented with fever (38.9 ℃), headache, nausea, vomiting, and drowsiness. Meningeal signs were positive. Cranial CT was normal. Lumbar puncture showed an opening pressure of 330 mmH_2O, CSF white cell count 1,850/μL (80% neutrophils), protein 2.8 g/L, and glucose 1.2 mmol/L. Gram stain again demonstrated gram-positive cocci in chains. Ceftriaxone and vancomycin were started empirically [9]. CSF culture grew S. agalactiae; blood culture was negative. Once susceptibility was confirmed, penicillin sensitivity was confirmed, and vancomycin was stopped; ceftriaxone was continued for 21 days. Fever resolved within 72 hours, and the patient was discharged neurologically intact.

Table 1. Clinical and laboratory features of three patients with tilapia spine injury-associated GBS meningitis

Characteristic	Case 1	Case 2	Case 3
Age (years) / Sex	52 / Male	64 / Male	47 / Male
Underlying condition	None	Type 2 diabetes	None
Injury site	Right index finger	Left thumb	Right middle finger
Injury to symptoms (h)	20	24	24

Characteristic	Case 1	Case 2	Case 3
Presenting symptoms	Fever, headache, photophobia, confusion	Fever, rigors, headache, confusion	Fever, headache, vomiting, drowsiness
CSF opening pressure (mmH_2O)	420	600	330
CSF WBC (/μL, % neutrophils)	8,500 (90%)	12,500 (95%)	1,850 (80%)
CSF protein (g/L) / glucose (mmol/L)	4.2 / 0.2	6.5 / 0.1	2.8 / 1.2
Blood culture	Negative	Positive (GBS)	Negative
Outcome	Full recovery	Hydrocephalus (shunted), mild cognitive impairment	Full recovery

3. Discussion

The three patients described here share a striking clinical pattern: a minor percutaneous wound from a tilapia spine, followed within a single day by fulminant meningitis caused by S. agalactiae. This temporal proximity between inoculation and central nervous system involvement points to rapid hematogenous dissemination rather than the indolent course typical of opportunistic GBS infection in chronically ill hosts. Notably, two of the three patients had no identifiable immunocompromising condition, a finding consistent with the Singapore ST283 outbreak in which healthy younger adults developed severe invasive disease [3]. A systematic review of adult GBS meningitis by van Kassel and colleagues identified predisposing conditions in most cases, yet also noted that a subset of patients lacked any recognized risk factor [10].

The mechanism by which GBS reaches the meninges after percutaneous exposure merits consideration. In the classic model, bacteria colonize mucosal surfaces, enter the bloodstream, and subsequently traverse the blood-brain barrier. Joyce et al. recently demonstrated that GBS synthesizes surface glycolipids, including glucosyl-diacylglycerol, mediated by the iagB gene, which promote bacterial survival in blood by protecting against neutrophil clearance and antimicrobial peptides [11]. An alternative pathway involves direct neural invasion along olfactory or trigeminal nerve routes when epithelial barriers are breached, a route demonstrated in murine models [12]. In the present series, bacteremia was documented in Case 2 and strongly suspected in the others, given the speed of symptom onset, favoring hematogenous spread as the dominant mechanism, though concurrent neural invasion cannot be excluded, given the neurotropism of GBS [12].

That GBS causes meningoencephalitis in tilapia is well established. Mian et al. reported that Brazilian Nile tilapia isolates required as few as 90 colony-forming units to produce lethal meningoencephalitis in experimental infections, and disease expression was temperature dependent, occurring above 26 °C [5]. A comprehensive review by Abdallah et al. further elaborated on the clinical signs, pathogenesis, and risk factors of GBS infection in cultured Oreochromis niloticus, highlighting its global impact on aquaculture [13]. Virulence gene profiling of aquatic GBS isolates has identified cfb, hylB, lmb, and cylE at high prevalence, with the presence of scpB, a gene classically associated with human GBS disease, in Thai aquatic strains, supporting the hypothesis that fish-derived strains retain pathogenic potential for human hosts [14]. The rapid progression from skin puncture to meningitis observed in our patients is compatible with infection by a strain bearing these neuroinvasive determinants.

From a diagnostic standpoint, each patient in this series presented with the typical CSF profile of pyogenic meningitis: marked neutrophilic pleocytosis, elevated protein, and low glucose. Opening pressures were notably high (330 to 600 mmH_2O), exceeding the range commonly reported for pneumococcal or meningococcal meningitis, and may warrant more aggressive intracranial pressure management [2]. CSF Gram stain showed gram-positive cocci in chains in all three cases, which, together with the occupational context, should prompt empiric coverage for GBS in addition to the usual empiric regimen of third-generation cephalosporin plus vancomycin. Culture confirmation remains the gold standard, though

culture-negative GBS meningitis is increasingly recognized [8]. Metagenomic next-generation sequencing (mNGS) has proven useful in such settings; He et al. reported a case of GBS meningitis in a diabetic patient diagnosed by mNGS after negative cultures [15]. Similarly, FilmArray meningitis/encephalitis panel detected GBS in a culture-negative case reported by Ide et al. [8].

All isolates in this series were susceptible to penicillin, allowing narrowing of the empiric regimen once susceptibilities returned. Ceftriaxone monotherapy was effective in two patients; the third, whose presentation was the most severe, received 28 days of treatment on account of hydrocephalus. Current guidelines recommend penicillin G or a third-generation cephalosporin for two to three weeks for GBS meningitis in adults, with longer courses considered in the setting of complications such as ventriculitis or brain abscess [9,16]. The development of hydrocephalus in Case 2 echoes a similar complication described by Akahara et al., who reported a patient with GBS meningitis, ventriculitis, and endocarditis [16]. Despite appropriate antimicrobial therapy, GBS meningitis can produce lasting structural sequelae, likely related to the intense inflammatory response triggered in the subarachnoid space.

Yang et al. recently described a case of severe soft-tissue infection following a freshwater fish spike injury, underscoring that fish spine injuries can introduce pathogenic bacteria into the human host beyond the skin compartment [17]. Our cases extend this observation by demonstrating that the central nervous system is also at risk, particularly when the inoculated organism possesses the invasive determinants documented in tilapia-associated GBS strains.

Several limitations apply to this case series. The retrospective design and single-center setting limit generalizability. We did not perform whole-genome sequencing on the GBS isolates, so we cannot confirm sequence type or demonstrate clonal relatedness to tilapia strains. Environmental sampling of fish or pond water from the sources named by patients was not conducted. These gaps could be addressed in future prospective studies.

Percutaneous injuries from tilapia spines represent an underappreciated route of GBS acquisition that can produce rapidly progressive, life-threatening meningitis in adults, including those without traditional risk factors. Emergency physicians and neurologists in regions where tilapia is farmed or sold should routinely ask about recent fish-handling injuries when evaluating patients with community-acquired bacterial meningitis, as early recognition of this exposure may guide empiric antimicrobial choices and improve outcomes.

4. Patient consent

Written informed consent was obtained from all patients for publication of this case series and any accompanying images.

Disclosure statement

The authors declare no conflict of interest.

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