OPEN

CASE REPORT
Hand/Peripheral Nerve

An Unusual Cause of Flexor Tenosynovitis: *Streptococcus mitis*

Ugur Anil Bingol, MD*
Cağatay Ulucay, MD†
Turhan Ozler, MD†

Summary: *Streptococcus mitis* is a commensal organism of the human oropharynx that rarely causes infection in healthy individuals. Herein, we describe a previously healthy 35-year-old woman who presented with acute pyogenic flexor tenosynovitis of the left index finger due to *S. mitis* infection. The patient's infection was treated successfully via surgical and medical interventions, and during follow-up, it was determined that she was complement component C3 deficient. Tenosynovitis is an emergent clinical syndrome that can result in permanent disability or amputation. To the best of our knowledge, this case report is the first to describe tenosynovitis due to *S. mitis*; in addition, it highlights the importance of initiating therapy with antibiotics that are effective against this rare pathogen. *(Plast Reconstr Surg Glob Open 2014;2:e263; doi: 10.1097/GOX.0000000000000236; Published online 9 December 2014.)*

Acute pyogenic flexor tenosynovitis is a serious infection of the hand that can occur following penetrating trauma to a digit. It is associated with considerable morbidity and can result in permanent functional impairment of the digit. The most common etiology of tenosynovitis is infection, and the majority of cases are due to gram-positive organisms. *Staphylococcus aureus* is the most commonly isolated organism in patients with tenosynovitis, accounting for 80% of all cases.[1] It is recommended that surgical treatment be initiated immediately in most cases of suspected tenosynovitis, whereas in certain otherwise healthy patients, antibiotic therapy alone can be used for the first 24 hours and the need for surgery can be reassessed based on the response to antibiotic treatment.[2] Herein, we present a previously healthy 35-year-old woman who developed tenosynovitis of the index finger due to *Streptococcus mitis* infection.

*From the *Department of Plastic, Reconstructive and Aesthetic Surgery, Yeditepe Medical School, Yeditepe University, İstanbul, Turkey; and †Department of Orthopedics and Traumatology, Yeditepe Medical School, Yeditepe University, İstanbul, Turkey.*

Received for publication October 10, 2014; accepted October 22, 2014.

DOI: 10.1097/GOX.0000000000000236

CASE REPORT

A 35-year-old woman was presented to the emergency department with left index finger pain and edema. She reported that a piece of a broken compact disc punctured her finger the day before, which prompted a visit to the emergency department of another institution; there, the wound was debrided and sutured, and she was discharged. At the time of presentation, the patient was afebrile and reported that she did not have fever or chills during the preceding 24 hours. Physical examination showed Kanavel's 4 signs of tenosynovitis, and purulent drainage was expressed from the wound, but the fluid was not foul smelling.

Baseline laboratory values for markers of inflammation were normal. The erythrocyte sedimentation rate was 14 mm/h (normal range, <25 mm/h), C-reactive protein was 1.3 mg/L (normal range,

Disclosure: *The authors have no financial interest to declare in relation to the content of this article. The Article Processing Charge was paid for by the authors.*

0.1–5 mg/L), and the leukocyte count was 6.13 L^{-1} (normal range, 4–11 L^{-1}) with 43% neutrophils (normal range, 36–66%). All other hematologic and biochemical parameters were normal. The patient was admitted to the hospital with the initial diagnosis of acute flexor tenosynovitis, and surgery was performed. An incision was made on the ulnar side of the left index finger, and drainage and debridement of the tendon sheath were performed via tenosynovectomy. Upon making the initial incision, a volume of purulent drainage was expelled suddenly. Massive necrosis was observed over the entire area between the base of the nail and the A2 pulley (Fig. 1). Samples of tissue and drainage fluid were sent to the microbiology department for analysis and culture, and *S. mitis* was isolated.

Empiric antibiotic treatment against all gram-positive organisms was initiated using teicoplanin 1 × 400 mg; additional treatment consisted of local wound care, including twice-daily dressing changes and mini debridements. The wound was left open and allowed to heal by secondary intention. The patient's symptoms resolved steadily during 12 days of hospitalization, during which time she remained afebrile and repeated blood samples never showed an elevated leukocyte count or acute-phase reactants. Tests for HIV antibodies and HIV RNA were negative. Further investigations showed a low level of blood complement component C3. She was referred to the immunology department with a presumptive diagnosis of a complement deficiency syndrome. The patient's medical history included 3 episodes of pneumonia in which *Streptococcus pneumoniae* was identified as the causative agent before age 10, hospitalization due to *S. aureus* sepsis 9 years earlier, and an allergic reaction to penicillin that resulted in anaphylaxis.

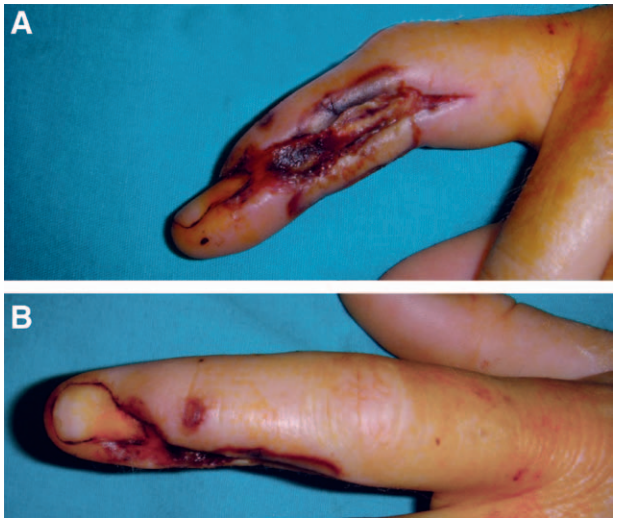

Fig. 1. Tenosynovitis with necrosis. A, ulnar and B, dorsal views.

DISCUSSION

Acute pyogenic flexor tenosynovitis is a surgical emergency that can occur following penetrating trauma to a digit. It is associated with high rates of long-term complications, including stiffness, persistent infection, boutonniere deformity, and amputation; 1 study reported a complication rate as high as 38%, with 17% of cases leading to eventual amputation of a digit.[3] Kanavel's 4 cardinal signs of tenosynovitis can be used for diagnosis: symmetric swelling of the entire digit; profound tenderness along the length of the digit; semiflexed position of the digit; and severe pain with passive extension of the affected digit.[4]

The most common etiology of tenosynovitis is introduction of bacteria through a wound, although hematogenous seeding of the tendon sheath has been described.[5] *S. aureus*, a normal commensal of the skin with notorious pathogenic potential, is the most frequently isolated organism in patients with tenosynovitis, accounting for up to 80% of cases; however, the number of cases due to methicillin-resistant *S. aureus* strains is increasing. *Streptococcus* species and *Pseudomonas aeruginosa* are isolated in the majority of the remaining cases.[1] Atypical organisms, including gram-negative rods, mixed flora, *Eikenella corrodens*, and *Pasteurella multocida*, can be the causative agent in immunocompromised patients and in victims of human and animal bites.[5] The wide variety of pathogenic agents, including methicillin-resistant *S. aureus*, that can cause tenosynovitis necessitates the use of broad-spectrum antibiotics for empiric therapy until culture and sensitivity results are obtained.

To the best of our knowledge, the present case report is the first to describe tenosynovitis due to *S. mitis*, which is a member of the *Streptococcus viridans* family and is typically a nonpathogenic commensal of the human oropharynx. Although *S. mitis* is of low virulence and pathogenicity, infection by this organism has been observed. In most cases of *S. mitis* infection, the host is elderly, immunocompromised, or undergoing cytotoxic anticancer treatment; however, *S. mitis* has been implicated in some oral infections and certain cases of infective endocarditis, meningitis, and bacteremia, even in immunocompetent hosts. *S. mitis* is particularly worrisome, as the strains identified in pathologic settings tend to be resistant to commonly used antibiotics.[6] Interestingly, *S. mitis* is the most antibiotic-resistant species of the *S. viridans* family.[7] *S. mitis* is naturally able to ac-

quire antibiotic resistance genes from other bacteria and to donate resistance genes to its closest genetic relative, *S. pneumoniae*.[6] The genome of *S. mitis* contains many of the virulence factors implicated in the pathogenicity of *S. pneumoniae*, and the cause of the large difference in pathogenicity between the 2 species is currently being investigated.[8]

The biological effects of complement include promotion of chemotaxis and anaphylaxis as well as opsonization and phagocytosis in microorganisms. C3 is activated by both the classic and alternative pathways and plays a central role in the complement system. Patients with C3 deficiency develop severe episodes of recurrent pneumonia, meningitis, peritonitis, and sepsis, beginning at an early age. The most common pathogens that cause infection in these patients are *S. pneumoniae*, *Neisseria meningitidis*, *Haemophilus influenzae*, and *S. aureus*.[9] Complement deficiency could account for the presented patient's vulnerability to severe gram-positive bacterial infections; however, additional research is needed to more clearly understand the pathophysiological mechanism/s involved in bacterial infections in individuals with this type of complement deficiency.

Because of the rapidly progressing clinical course of tenosynovitis and its ability to cause permanent disability, timely initiation of empiric antibiotic therapy with broad-spectrum antibiotics is critically important.[2] Identification of *S. mitis* as the causative bacterium in the presented case highlights the importance of administering antibiotics that are effective against atypical pathogens until culture and sensitivity results are obtained.

CONCLUSION

The considerable morbidity associated with acute flexor tenosynovitis and the present study's identification of a novel causative agent with great capacity for antibiotic resistance underscore the importance of treating patients empirically with broad-spectrum antibiotics that are effective against *S. mitis*. We also think that the present findings concerning *S. mitis* should form the basis of additional research on the pathogenic nature of *S. mitis*. Lastly, the presented patient's history of multiple serious infections with other gram-positive organisms and her current complement deficiency syndrome suggest a unique vulnerability, indicating that more studies on the role of complement in gram-positive infections are necessary.

Ugur Anil Bingol, MD
Department of Plastic
Reconstructive and Aesthetic Surgery
Yeditepe Medical School
Yeditepe University
34752 Kozyatağı
Istanbul, Turkey
E-mail: ugur.bingol@yeditepe.edu.tr

REFERENCES

1. Hausman MR, Lisser SP. Hand infections. *Orthop Clin North Am.* 1992;23:171–185.
2. McDonald LS, Bavaro MF, Hofmeister EP, et al. Hand infections. *J Hand Surg Am.* 2011;36:1403–1412.
3. Pang HN, Teoh LC, Yam AK, et al. Factors affecting the prognosis of pyogenic flexor tenosynovitis. *J Bone Joint Surg Am.* 2007;89:1742–1748.
4. Kanavel AB. The symptoms, signs, and diagnosis of tenosynovitis and major fascial space abscesses. In: *Kanavel AB, ed. Infections of the Hand.* 6th ed. Philadelphia, Pa.: Lea and Febiger; 1933:364–395.
5. Draeger RW, Bynum DK Jr. Flexor tendon sheath infections of the hand. *J Am Acad Orthop Surg.* 2012;20:373–382.
6. Mitchell J. *Streptococcus mitis*: walking the line between commensalism and pathogenesis. *Mol Oral Microbiol.* 2011;26:89–98.
7. Doern GV, Ferraro MJ, Brueggemann AB, et al. Emergence of high rates of antimicrobial resistance among viridans group streptococci in the United States. *Antimicrob Agents Chemother.* 1996;40:891–894.
8. Johnston C, Hinds J, Smith A, et al. Detection of large numbers of pneumococcal virulence genes in streptococci of the mitis group. *J Clin Microbiol.* 2010;48:2762–2769.
9. Figueroa JE, Densen P. Infectious diseases associated with complement deficiencies. *Clin Microbiol Rev.* 1991;4:359–395.