Acute Carpal Tunnel Syndrome Due to Pyogenic Flexor Tenosynovitis without Any Antecedent Injury

Hirofumi Yoshida¹, Haruki Imura², Taiga Goto³, Takeharu Nakamata¹, Mohamud R. Daya¹ and Toru Kamiya¹,²

Abstract

We herein report a case of acute carpal tunnel syndrome due to pyogenic flexor tenosynovitis in the absence of any antecedent injury whose rapid progression and course was similar to that seen with necrotizing fasciitis. This potentially disastrous clinical condition must be promptly recognized, since it needs early surgical management to prevent morbidity.

Key words: carpal tunnel syndrome, pyogenic flexor tenosynovitis, necrotizing fasciitis, Staphylococcus aureus

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Introduction

Carpal tunnel syndrome is a common chronic compressive neuropathy of the hand involving the median nerve. Although less common, carpal tunnel syndrome can also occur acutely due to trauma, coagulopathy, inflammation or infection (1). We review the literature and report a case of acute carpal tunnel syndrome that was characterized by no antecedent puncture injury to the hand, the presence of hypoesthesia in all five fingers and a rapidly progressive clinical presentation that resembled necrotizing fasciitis.

Case Report

A previously healthy 48-year-old Japanese woman presented to the emergency department with severe pain in her dominant right hand and wrist. She worked as an office assistant but denied any blunt or penetrating trauma to the hand. She did not take any medications and had no animal contact. Fifteen hours prior, she described the gradual onset of a tingling sensation at the tips of the fingers along with mild clumsiness in her hand. She tried loxoprofen (non-steroidal anti-inflammatory drug) without any improvement in her symptoms.

On an examination, her vital signs were normal, and the hand was noted to be held in a partially flexed position at the wrist (Fig. 1). The skin of the volar aspect of the right wrist appeared red and edematous. The radial and ulnar artery pulses were palpable, and the capillary refill time was normal. A neurological examination revealed subjective hypoesthesia in all five fingers with positive Tinel and Phalen tests. Plain wrist radiographs were unremarkable, but an unenhanced computed tomography (CT) scan of the wrist demonstrated edematous changes around the digital flexor tendons within the wrist (Fig. 2). Laboratory studies were consistent with an acute infectious or inflammatory process with a white blood cell count of 11,000/μL with 87% neutrophils. Over the ensuing two hours, the patient’s pain and edema rapidly worsened, and the erythema began to extend up her arm. Due to the rapid progression and pain out of proportion, an emergent surgical consultation was obtained for possible necrotizing fasciitis. In addition to necrotizing fasciitis, the surgery team was also concerned about pyogenic flexor tenosynovitis, and she was taken promptly to the operating room.

The intraoperative findings revealed a blood-stained purulent exudate as well as edematous changes within the com-
mon flexor tendon sheath (ulnar bursa) with secondary med-ian nerve compression but no evidence of necrotizing fasci-itis. The ulnar bursa was incised, debrided, and irrigated with saline and an external drainage catheter was placed. Two sets of blood cultures failed to grow any organisms, but a Gram stain of the exudate showed the presence of Gram-positive cocci, subsequently identified as penicillin-resistant but methicillin-susceptible Staphylococcus aureus. Preoperatively, she received meropenem, vancomycin and clindamy-cin and the antibiotic coverage was narrowed to first-generation cephalosporin based on the microbiological sus-ceptibility pattern. She had an uncomplicated postoperative course, and antibiotic treatment was continued for 20 days. At her two-month follow-up, she had almost completely re-gained the motor and sensory function in the hand. The final diagnosis was acute carpal tunnel syndrome secondary to pyogenic flexor tenosynovitis.

**Discussion**

Carpal tunnel syndrome is a common chronic compressive neuropathy of the hand involving the median nerve. Although rare, carpal tunnel syndrome can also occur acutely due to trauma, coagulopathy, inflammation or infection (1). To date, there have been 15 cases of acute carpal tunnel syndrome related to bacterial infection reported in the English literature (Table) (2-11). Most of these patients were in good health, although one was immunocompromised due to a prior splenectomy. The disease developed mainly in middle-aged and older patients but across a wide age range, from 10 to 69 years old (median: 52 years old), and was al-most equally observed in men (7/15 cases) as in women (8/ 15 cases). In 11 cases, a preceding injury was thought to be the source of infection, whereas 4 patients had no prior his-tory of antecedent trauma. The initial clinical presentation ranged from an acute course of a few hours to symptoms developing up to eight days following injury. The etiological organism most commonly involved was Staphylococcus aureus (6/15 cases, all of which were methicillin-sensitive) but also included β-hemolytic streptococci, coagulase-negative staphylococci, Enterococcus faecalis, Clostridium histolyticum and Pasteurella multocida. Our case is remark-able in that there was no antecedent puncture injury to the hand, hypoesthesia was present in all five fingers and the rapidly progressive clinical presentation resembled necrotiz-ing fasciitis.

The severe and rapid worsening of pain, edema and erythema raised concerns of possible necrotizing fasciitis; however, the patient’s clinical examination findings were also consistent with pyogenic flexor tenosynovitis due to the presence of Kanavel’s four classic signs (fusiform symmetric swelling of the entire digit, digit held in partial flexion, ten-derness along the flexor tendon sheath, pain along the ten-don with passive digit extension) at the wrist (Fig. 1). While median nerve compression classically causes paresthesia in the first three fingers and the radial portion of the fourth fin-ger, the intermediate bursa connects the radial and ulnar bursa in 80% of individuals (12). This permits a continuous link between the tendon sheaths of the flexor pollicis longus and that of the fifth finger. We suspect that the involvement of ulnar bursa in addition to compression of the median nerve resulted in paresthesia in all five fingers in our case. Although, we performed unenhanced CT on this patient due to our concern for necrotizing fasciitis, another useful imaging technique to consider is a focused point-of-care ultrason-dex of the wrist, since this has been found to be valuable in the early diagnosis of flexor tenosynovitis associated with digits (13). While a physical examination and imaging may help distinguish necrotizing fasciitis from acute carpal tunnel syndrome, an early surgical consultation is a must, since in both instances surgery is an essential part of the definitive diagnosis and therapy.

Initial empiric therapy should be targeted to cover skin organisms, including methicillin-resistant Staphylococcus aureus, given its increasing prevalence and the potentially disastrous nature of acute pyogenic carpal tunnel syndrome.
and prompt operative management is essential.

Second, unlike chronic carpal tunnel syndrome, acute pyogenic carpal tunnel syndrome most likely arises from contiguous spread through visible or invisible wounds rather than hematogenously. While we treated this patient for 20 days, Nourissat et al. suggested that, in the absence of any causative injury, prolonged treatment with systemic antibiotics may be unnecessary following total excision of the infected tissue in a healthy patient (9). We suggest that the duration of therapy should be guided by the culture results and clinical response following surgery.

In conclusion, this case highlights two important clinical teaching points. First, acute carpal tunnel syndrome due to pyogenic flexor tenosynovitis can be a cause of acute onset and rapid progression of wrist pain, even in the absence of injury. Second, unlike chronic carpal tunnel syndrome, acute pyogenic carpal tunnel syndrome can present with paresthesia in all five fingers due to the involvement of the interconnected radial and ulnar bursae. Since acute pyogenic carpal tunnel syndrome can lead to permanent median nerve injury as well as finger and/or hand amputation, early recognition and prompt operative management is essential.

The duration of treatment with prior cases has ranged from one day to five weeks. Neither blood stream infections nor disseminated lesions have been reported, regardless of whether an antecedent injury exists or not. Our patient also showed no systemic symptoms indicative of bacteremia, such as fever or shaking chills, and had neither positive blood culture results nor distant organ involvement. Based on the reported cases, it is reasonable to presume that acute pyogenic carpal tunnel syndrome most likely arises from contiguous spread through visible or invisible wounds rather than hematogenously.

The authors state that they have no Conflict of Interest (COI).

Table. Characteristics of Cases of Acute Pyogenic Carpal Tunnel Syndrome with Bacterial Cause.

| Patient number | Age | Sex | Medical history | Injury | Surgery | Intraoperative culture results | Duration | Reference |
|----------------|-----|-----|-----------------|--------|---------|-------------------------------|----------|-----------|
| 1              | 58  | F   | Gout, Early osteoarthritis | Yes    | Yes     | Coagulate-negative staphylococci | 10 days  | 2         |
| 2              | 53  | F   | Unknown          | Yes    | No growth | β-hemolytic streptococci | Unknown  |           |
| 3              | 68  | F   | Unknown          | No     | No       | β-hemolytic streptococci, Staphylococcus aureus, Streptococcus faecalis | Unknown  | 3         |
| 4              | 52  | M   | Unknown          | Yes    | Yes     | Streptococcus faecalis       | Unknown  | 3         |
| 5              | 50  | F   | Unknown          | Yes    | No growth | Staphylococcus aureus       | Unknown  | 4         |
| 6              | 10  | F   | Unknown          | Yes    | Yes     | Clostridium histolyticum     | 6 weeks  | 5         |
| 7              | 23  | M   | Healthy          | No     | No       | Staphylococcus aureus        | 3 weeks  | 6         |
| 8              | 62  | F   | Subtotal pancreatectomy for chronic pancreatitis, Splenectomy | Yes    | Yes     | Staphylococcus aureus        | 3 weeks  | 6         |
| 9              | 68  | M   | Unknown          | Yes    | Yes     | Coagulate-negative staphylococci | 8 days   | 6         |
| 10             | 59  | M   | Unknown          | Yes    | Yes     | Staphylococcus aureus       | Unknown  | 7         |
| 11             | 43  | M   | Unknown          | Yes    | Yes     | Coagulate-negative staphylococci | Unknown  | 8         |
| 12             | 47  | M   | Unknown          | No     | No       | Staphylococcus aureus       | 1 day    | 9         |
| 13             | 52  | M   | Unremarkable PMH, Penicillin allergy | Yes | Yes | group G streptococci | Unknown  | 10        |
| 14             | 56  | F   | Unknown          | Yes    | Yes     | Pasteurella multocida       | Unknown  | 11        |
| 15             | 48  | F   | Healthy          | No     | No       | Staphylococcus aureus       | 20 days  | Present case |

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