Closed-space hand infections: diagnostic and treatment considerations

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Abstract

Despite modern diagnostic methods, surgical advances and antibiotics evolution, acute closed-space hand infections still remain a therapeutic challenge. The aim of this review is to present its special clinical features and the current therapeutic management based on the infection site, the type of the infecting pathogen and the host-type. Anatomic pathways facilitate the spread of the infection towards spots of decreased resistance. The accumulation of purulent material subsequently raises the pressure within the closed-space, leading to ischemia and necrosis. These infections are usually attributed to gram-positive cocci and clinicians should also consider the local spread of community-acquired methicillin resistant S. aureus and the host’s comorbidities (immunosuppression, diabetes) before choosing the appropriate antibiotics. Surgical treatment including drainage and irrigation is imperative. The knowledge of anatomy, closed-space pathophysiology and current updates in microbiology and drainage/irrigation techniques are prerequisites for prompt diagnosis and optimal treatment of acute closed-space hand infections.

Introduction

Closed-space hand infections have been considered traditionally as an important concern and have been treated as an emergency. Despite modern diagnostic methods, surgical treatment advances and the evolution of newer antibiotics, hand infections remain a therapeutic challenge.1 Even those authors advocating an evident decrease in the ratio of hand infections, underline the necessity of treatment of infected hands to be treated by experienced staff.2,3

The purpose of this review is to present the special clinical features and the current therapeutic management of closed-space hand infections based on the localization (entry point and spread to adjacent structures), the pathogen and the type of the host. Adequate knowledge of the hand anatomy,1,2 the closed-space pathophysiology and the current updates in microbiology are prerequisites for prompt diagnosis and optimal treatment.

Soft tissue anatomy of the hand: considerations relevant to infection

The differences between the palmar and dorsal structures may explain the different pathways of extension and the different clinical signs of infection between the two sides of the hand. The palmar skin is thicker than the dorsal and is anchored to the underlying structures. It contains many sweat glands but no hair follicles or sebaceous glands, whereas the tough palmar fascia represents a thick and resistant fibrous tissue layer. Both skin and fascia hinder the horizontal spread of pus and even edema to the palm. Pus is rather oriented to the deeper palmar structures while edema is always more prominent at the dorsum of the hand.4 In the deeper structures of the hand the anatomic relationships are established, but variations cannot be excluded.4,5 In Table 1 the special characteristics of all structures of the palmar and dorsal sides of the hand are presented from distal to proximal, in relation to the pathophysiology.4,5

Pathways of pus spread

The true anatomic communications represent pathways of decreased resistance for the spreading pus (Table 2).4,6,9,10 However, in the late stages of purulent infections direct spread into the surrounding tissues through liquefactive necrosis is anticipated.

Pathophysiology

In all closed spaces of the hand, accumulation of purulent material is raising the pressure, compromising the blood flow, and causing ischemia and necrosis. These conditions are further aggravating the infection, establishing a vicious circle.6,11,12 Direct inoculation (epithelium lysis), spread from the adjacent necrotic tissues or through the lymphatic pathway, could result in the establishment of hand infections.

Pathogens

Gram positive cocci and especially S. aureus and β-Hemolytic-Streptococcus group A (pyogenes) are usually involved. This is the result of skin flora inoculation during injury.13 S. aureus is reported to range from 30 to 80% of positive cultures,1,2,5,14-20 although it has been shown that this percentage is decreasing while mixed gram positive and gram negative hand infections are increasing.21,22 An emerging pathogen is the S. aureus that carries the PVL gene; it is usually community-acquired and leads to necrotic lesions.22 At least 3 sets of cultures should be ordered, each one including cultures for bacteria, mycobacteria and fungi. Gram stain provides important information. While cultures are often negative,20,22 recent advances in the detection and identification of bacterial pathogens by molecular methods greatly facilitate the diagnosis and expedite the initiation of treatment.24

Empirical antibiotic treatment is mandatory,1,2,17,18,25 while waiting for the culture results, taking into consideration the hospital flora and the local spread of community-acquired methicillin resistant S. aureus (CA-MRSA). The introduction of antibiotics was a great progress in the early treatment of hand infections and led to the reduction of devastating complications such as amputations.19,26,28

Host type and infection

In immunocompromised patients (underlying malignancy and immunosuppressive dis-
human bite and crush injuries, as well as in diabetes mellitus, intravenous use of drugs and in infection are usually absent. A part of a closed spaces. Finally, systemic signs of infection are likely to occur in these patients, including gram negative bacteria and/or anaerobes and appropriate antibiotics must be administered. The same principles apply to patients with diabetes mellitus, intravenous use of drugs and in human bite and crush injuries, as well as in injuries in highly contaminated environments.

Diagnosis

Because of the deep location of closed space accumulations the typical signs infection are often absent. Throbbing pain, edema and restricted finger motion are the most common sings and symptoms, while specific clinical signs such as Kanavel’s signs (septic tenosynovitis) may be obtained in cases of infections in closed spaces. Finally, systemic signs of infection are usually absent. Apart of a complete laboratory testing (complete blood count, ESR, CRP), imaging studies such as radiographs and ultrasound are helpful in the diagnosis of concomitant skeletal involvement or deep space collections. Culture samples must be obtained before the administration of antibiotics; Gram stain, antibiotic resistance phenotypes, detection of the mec A gene, and molecular typing of the isolates must be requested from the microbiology departments.

Treatment

In deep space hand infections apart of antibiotic coverage and immobilization with hand elevation, the surgical incision and drainage of all potentially communicating spaces and compartments is mandatory, along with intraoperative irrigation and sometimes, continuous postoperative irrigation. Parameters such as the pathway of inoculation, the environment where the initial injury occurred and above all the underlying condition of the host must be taken into consideration for a radical and successful treatment. Finally, postoperative hand therapy must be initiated as soon as the acute signs of infections subside.

Antibiotic treatment is usually initiated with penicillinase-resistant penicillin or cephalosporins. The oral empiric antibiotic treatment expected to be effective against suspected CA-MRSA infections includes ciprofloxacin, clindamycin,(370,209),(996,261)

Table 1. Special anatomical considerations of closed spaces of the hand.

| Anatomic structure     | Special characteristics                       | Pathophysiology                                      |
|------------------------|------------------------------------------------|------------------------------------------------------|
| Finger pulp            | Connective tissue framework                   | Closed space conditions                               |
| Vertical septa          | Fibrous sheaths (tunnels)                     | Anchoring dermis to palmar fascia preventing          |
| Flexor tendon of digit  | Surrounding sheath                            | Closed space conditions                               |
| Flexor tendon of small finger | Possible connection with ulnar bursa       | Pathway of decreased resistance                       |
| Flexor tendon of thumb  | Possible connection with radial bursa         | Pathway of decreased resistance                       |
| Ulnar bursa (level of hand) | Enveloping flexor tendon sheaths of ring/small fingers | Pathway of decreased resistance                       |
| Ulnar bursa (level of wrist) | Possible connection to radial bursa          | Pathway of decreased resistance                       |
| Muscle compartments    | Superficial (thenar, hypothenar), deep (interossei-adductor pollicis muscle) | Closed space condition                               |
| Fascial spaces         | Semi-closed spaces, limited by the palmar vertical septae delineated by the dorsal fascia and skin | Spread infection into subcutaneous distally and dorsally (collar button abscess) |
| Deep                   | 1. Between thenar muscle compartment & adductor pollicis muscle | Spread infection into adjacent fascial spaces/bursa |
|                        | 2. Located deep to the flexor tendons of the long, ring and little fingers and superficial to the 3rd-5th metacarpals. On the radial side it is separated from the thenar space by a very firm partition with a weaker proximal edge and on the ulnar side the space is overlapped by the ulnar bursa | Spread infection into adjacent fascial spaces/bursa |
|                        | 3. Hypothemnlar                                | Pathway of decreased resistance                       |
| Lubrical channel       | Tunnel of fascia surrounding the lubrical muscle communication between web and deep palmar fascial spaces | Pathway of decreased resistance                       |
50% of cases.19 Many authors advocate secondary infection from the extension of a severe or neglected infection around the nail as tissue liquefactive necrosis favors the spread to the surrounding tissues,16,19,31 although the anatomical course of the lymphatics does not support this opinion.4

The typical signs of acute inflammation are present. The initial sticking pain in the distal phalanx transforms to throbbing severe pain, while the initial tenseness of the pulp is replaced by an induration and later by a fluctuating boggy mass. Due to presence of sepsis, pus or edema have no means of free egress. Due to presence of septa, an early felon could be an unnecessary and even harmful procedure over a cellulitis,4 causing diffuse pain and tenderness throughout the whole finger.5,10 Imaging modalities (e.g. ultrasound) can help the differential diagnosis by detecting fluid accumulations.

Decrease of pain sometimes signals an aggravation of the felon as the necrotic process destroys the sepsis and therefore offers a temporary relief.

Tenosynovitis/bursitis

The primary infection is caused by direct inoculation of the tendon sheath/bursa. If left untreated, the secondary infection is carried either through the lymphatics or through pus spreading from adjacent fascial spaces. Sokolow advocated that the primary infection is more rapid and destructive.8

Exquisite tenderness over the course of the sheath is the most frequent of the cardinal symptoms and signs of Kanavel.15 Swelling of the adjacent fingers often supplements the clinical picture, whereas systemic symptoms of infection are unusual.

In severe or aggravating infections, drainage through a wide or limited approach, in a bloodless field, is necessary. The wide approach is either lateral or palmar (zig-zag) whereas the limited consists of two small incisions over the two edges (A1 and A5 pulleys) of the infected sheath. Surgeons advocating the limited approach claim a better final range of motion,14,21,25,27,29,33,34 while others believe that a limited approach should be used in less severe cases.20,30 although there are no level I studies comparing the type of incision. All authors agree that intra-operative irrigation is useful or at least of no harm. In addition, many authors advocate that post-operative sheath irrigation until the resolution of the acute inflammation.5,11,14,15,21,28,31,35

Ulnar bursitis is often difficult to be diagnosed due to its deep location. It is characterized by the development of hand edema, especially upon the dorsum. A general fullness is seen in the palm, but the palmar concavity is not lost at first. The wall of the bursa often becomes necrotic before the extensive formation of exudate, and this permits the trespass of the accumulation to the surrounding closed spaces. There is exquisite tenderness and the wrist becomes fixed, whereas the little finger and sometimes the ring finger show tenderness to palpation and pain on passive extension. Extension to the radial bursa is observed up to 85% of cases.4,9

Radial bursitis is diagnosed by the swelling and tenderness in the thenar eminence and along the radial bursa.

The treatment in cases of purulent accumulation consists of surgical drainage, debridement of abscesses through a palmar incision over the infected area followed by intraoperative irrigation with normal saline. Care should be taken not to injure the branch of the median nerve, which supplies the thenar muscles as it passes across the radial bursa, approximately 1 cm distal to the transverse ligament of the wrist.

In both tenosynovitis and bursitis the surgical wounds can be closed by secondary intention unless there is continuous postoperative irrigation.36 Passive assisted and active exercises to restore range of motion start with the remission of acute inflammation and after the removal of postoperative irrigation system (after 24-48 hours).13 However Nemoto et al. in 1991 suggested mobilization to start even if the irrigation system is in place and advocates the use of the system for a week postoperatively.
**Deep space infections of the hand**

These are infections of the midpalmar space, the thenar space and compartment, the hypothenar space and compartment and the subaponeurotic space of the dorsum of the hand. They are either primary infections via direct bacterial inoculation or secondary infections through extension from adjacent tendon synovium/bursae or fascial spaces. An aggressive treatment may prevent devastating complications from the adjacent sheaths, nerves, bones and joints. Close observation and antibiotics are always necessary. Drainage, debridement and intraoperative irrigation are the subsequent steps, whereas the decision for continuous postoperative irrigation is based on intraoperative findings. The wound can be closed later or by secondary intention if continuous postoperative irrigation is not used. Rehabilitation with passive assisted or active range of motion exercises begins after the remission of the acute inflammation.

In a mid-palmar infection the concavity of the palm is usually lost. There is an exquisite tenderness over the infected area which is palil and sometimes red. Fingers are held rigidly flexed, with decreasing rigidity from the little to the index finger. However, finger rigidity in case of mid-palmar infections is less severe than the rigidity of septic tenosynovitis. There is usually a pitting edema in the remaining palm (over the thenar) and at the dorsum of the hand, while in the case of pus extension along the cubital channel, swelling of the web space is also observed. The midpalmar space is drained and debrided through a palmar incision (transverse, longitudinal or one of the cubital channels) taking care to avoid injury of the neurovascular bundle or contamination of the surrounding tissues (the ulnar bursa in particular).

In the thenar compartment and thenar space infections a painful swelling of the thenar eminence is observed. The concavity of the palm is lost and often soft edema on the dorsum of the hand is present. The 1st metacarpal and the thumb are abducted and distal phalanx flexion becomes more marked. In extensive infections, a double incision over the volar and dorsal side is necessary for drainage of the infected spaces/compartments.

In hypothenar compartment and space infections the abscess is more localized. There is often limited swelling in the palm but always painful swelling of the hypothenar eminence. The incision is located over the hypothenar with care to avoid injury to the neurovascular bundle.

In subaponeurotic space infections the dorsal edema is more soft and the ischemia of the skin less severe than in cases of subcutaneous pus accumulation. Attempts of finger extension may be limited by pain. The dorsal incisions for the drainage of the subaponeurotic spaces are longitudinal, over the 2nd and between 4th-5th metacarpals.

**Web space infections**

These are either primary infections from direct inoculation or secondary extending from the adjacent anatomical structures. The diagnosis is clinical and based on an abscess forming at the distal edge of the palm separating the adjacent fingers. The attachment of the palmar fascia to the skin results to the spread of the pus into the dorsal subcutaneous space with the typical picture of a collar-button abscess. Surgical treatment includes drainage of the often multi-loculated abscess, through a palmar and/or a dorsal incision. The incision should never cross the edge of the web to avoid the formation of dysfunctional scars.

**Highlights**

- Because of the intimate relationship of the midpalmar and the thenar spaces, any infection persisting for more than 48 hours extends in the majority of cases to the adjacent space. When both spaces are involved, a considerable abscess is formed under the flexor tendons.
- These are infections of the midpalmar space, the thenar space and compartment, the hypothenar space and compartment and the subaponeurotic space of the dorsum of the hand.
- Rehabilitation with passive assisted or active range of motion exercises begins after the remission of the acute inflammation.

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