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]

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,[3,4] 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.[5] In the deeper structures of the hand the anatomic relationships are established, but variations cannot be excluded.[6-8] 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 pathopshysiology.[4,5]

Pathways of pus spread

The true anatomic communications represent pathways of decreased resistance for the spreading pus (Table 2).[3,4,9,10] 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*.[8,11,12] Direct inoculation (epithelium lysis), spread from the adjacent necrotic tissues or through the lymphatic pathway, could result in the establishment of hand infections.

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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,[20,23] 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.[13,26-28]

Host type and infection

In immunocompromised patients (underlying malignancy and immunosuppressive dis-

ease) or patients receiving steroids and anti-tumor necrosis factors, closed-space hand infections present subtly and a high rate of morbidity must be expected if treatment is delayed. Temperature elevation, localized tenderness and erythema are indications for hospitalization while broad spectrum antibiotics and debridement are needed. Discoloration, fluctuation, drainage or, even worse, hypotension were found to be late signs.[2,11,18] Polymicrobial infections and mixed infections 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, rifampin, tetracyclines, and TMP-trimethoprim/sulfamethoxazole. For more serious infections, intravenous vancomycin is recommended. Alternative intravenous therapies include daptomycin, gentamicin, and linezolid.[17,22,29] In crush injuries, injuries in highly contaminated environments or in immunocompromised hosts, gram negative bacteria and/or anaerobes are suspected.[1,6,14,15,18,23,25,30]

Antibiotics are usually required for 7 to 10 days unless complications arise.[18] The route of administration is intravenous for all cases that require hospitalization, until the remission of the acute signs of infection. Subsequently an oral regimen could be administered.

Specific approaches will be presented for the different types of closed-space infections. In all cases a bloodless field is imperative for the evaluation of all potentially infected closed-spaces and for the drainage through safe anatomical paths. Special attention must be given to the avoidance of use of Ershmarch bandage so as to limit the spread of infection. Simple elevation of the hand and forearm is usually adequate for a bloodless field.

Felon

Felon is an infection of the pulp of the distal phalanx. It is usually a primary infection caused by an unnoticed injury in more than

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 horizontal spread	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		Closed space condition
Superficial:		
Web 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. Thenar	1. Between thenar muscle compartment & adductor pollicis muscle	Spread infection into adjacent fascial spaces/bursa
2. Midpalmar	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. Hypothenar		
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 septa, pus or edema have no means of free egress and therefore occlude the blood supply and result in necrosis of the pulp and distal phalange. At the first stage necrosis can spare the epiphysis and distal interphalangeal (DIP) joint,[4-6,14,15,31] but in neglected cases spontaneous expulsion of the necrotic tissue is expected. At this stage the finger pulp is a bluish insensitive pus bag with asinus opening by the nail side. Usually there are no systemic signs of infection.[6]

A lateral incision is preferable since the transverse dissection of the radiating columns of fat and connective tissue results in more efficient drainage and wound closure is by secondary intention. Attention should be drawn to avoid exposure of the healthy DIP joint or the flexor tendon sheath through a proximally extended incision.[5,14] The treatment of coexisting osteomyelitis of the distal phalanx depends on the amount of bone involvement and includes resection of the involved bone or even amputation.

Highlights

Surgical drainage of the finger pulp performed too early, at the stage of a more diffuse inflammation, that some authors call *an early felon*,[15,19] could be an unnecessary and even harmful procedure over a cellulitis,[6] causing diffuse pain and tenderness throughout the whole finger.[4,31] 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 septa and therefore offers a temporary relief.

Tenosynovitis/bursitis

The primary infection is caused by direct inoculation of the tendon sheath/bursa.

In cases of crushing injuries, infection of the tendon sheath is frequent, however in other cases the initial trauma may be even unnoticed.[8] 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.[20]

Exquisite tenderness over the course of the sheath is the most frequent of the cardinal symptoms and signs of Kanavel.[32] 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,[14,23,25,27,29,32-34] while others believe that a limited approach should be used in less severe cases,[20,33] 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.[5,11,14,15,23,30,33,35] The irrigating solution consists of normal saline without antibiotics.[30,34]

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).[5,8] 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.

Table 2. Pathways of pus spread.

From	Through	To
Index flexor tendon sheath	1. Proximal edge 2. Lubrical channel	Thenar space (vice versa)
Middle and ring flexor tendon sheath	1. Proximal edge 2. Lubrical channel	Midpalmar space (vice versa)
Web space	Lubrical channel	Deep palmar spaces (vice versa)
Flexor policis longus tendon sheath	Proximal edge	Radial bursa (vice versa)
Small finger flexor tendon sheath	1. Proximal edge 2. Lubrical channel	1. Ulnar bursa (vice vesa) 2. Midpalmar space
Ulnar bursa	1. Radial edge 2. Communication distal to the wrist 3. Proximal edge	1. Midpalmar space (vice versa) 2. Radial bursa (more often vice versa) 3. Parona's space of wrist
Radial bursa	1. Ulnar edge 2. Proximal edge	1. Thenar space (vice versa) 2. Parona's space of wrist
Hand dorsal subaponeurotic space	Narrow connection at MCP level	Finger dorsal subaponeurotic spaces

Highlights

In the presence of multiple painful fingers, careful evaluation will reveal the infected one from its marked painful rigidity.

Decrease of tenderness to a considerable degree does not necessarily mean a definite improvement; it may be only a temporary relief due to the rupture of the infected sheath at its proximal edge and therefore of extension of the pus to more proximal structures. Differential diagnosis should include acute bleeding into the tendon sheath in patients under anticoagulation therapy and also tenosynovitis developing in 2/3 of patients with gonococcal infection.[5,19]

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 a 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.[14,36] 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 pallid 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,[14,15] while in the case of pus extension along the lubrical channel, swelling of the web space is also observed. The midpalmar space is drained and debrided through a palmar incision (transverse, longitudinal or along one of the lubrical channels) taking care to avoid injury of the neurovascular bundle or contamination of the surrounding tissues (the ulnar bursa in particular).[4,28,31]

In *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.[14,15,28,31,36]

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.[15]

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.[4,28] Attempts of finger extension may be limited by pain. The dorsal incisions for the drainage of the subaponeurotic space are longitudinal, over the 2nd and between 4th-5th metacarpals.[14]

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.

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.[15,36] 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

The web is a significant crossroad in hand infections.

References

1. Simmen HP, Giovanoli P, Battaglia H, et al. Soft tissue infections of the upper extremities with special consideration of abscesses in parenteral drug abusers. A prospective study. J Hand Surg Br 1995;20:797-800.
2. Stevenson J, Anderson IW. Hand infections: an audit of 160 infections treated in an accident and emergency department. J Hand Surg Br 1993;18:115-8.
3. Best R. An anatomical and clinical study of infections of the hand. Ann Surg 1929;89:359-78.
4. Kanavel AB. Infections of the Hand. 4th ed. Philadelphia: Lea, Febiger; 1921. p 16.
5. Spann M, Talmor M, Nolan W. Hand infections: basic principles and management. Surg Infect 2004;5:210-20.
6. Clark DC. Common acute hand infections. Am Fam Physician 2003;68:2167-76.
7. Jamieson JG. The fascial spaces of the palm with special reference to their significance in infections of the hand. Brit J Surg 1950;28:193-9.
8. Monstrey SJ, Van der Werken C, Kauer JM, Goris RG. Tendon sheath infections of the hand. Neth J Surg 1985;37:174-8.
9. Beye HL. Deep palmar hand infections. Ann Surg 1917;66:24-42.
10. Hoon LW, Ross GJ. Infections of the hand: a review of 90 cases. Ann Surg 1913;57:561-8.
11. Rigopoulos N, Dailiana ZH, Varitimidis S, et al. Compartmental infections of the hand. Scand J Plast Reconstr Surg Hand Surg 2008;42:38-42.
12. Schnall SB, Vu-Rose T, Holtom P, et al. Tissue pressures in pyogenic flexor tenosynovitis of the finger. J Bone Joint Surg Br 1996;78:793-5.
13. Petinaki E, Malizos KN. Bacterail ecosystem of the upper extremity. In: Malizos KN, Soucacos PN. Infections of the Hand & Upper Extremity. 1st ed. Athens: PMP (Paschalidis Medical Publications); 2007. p 19.
14. Abrams RA, Botte MJ. Hand infections: treatment recommendations for specific types. J Am Acad Orthop Surg 1996;4:219-30.
15. Brown MB, Young LV. Hand infections. South Med J 1993;86:56-66.
16. Chong AKS, Puchaindran ME, Lim AYT, Looi KP. Common bacterial infections of the hand. Singapore Med J 2006;47:340-5.
17. Dailiana ZH, Rigopoulos N, Varitimidis SE, et al. Clinical and epidemiological features of upper-extremity infections caused by Staphylococcus aureus carrying the PVL gene: a four-year study in Greece. Med Sci Monit 2008;14:511-4.
18. Dellinger EP, Wertz MJ, Miller SD, Coyle MB. Hand infections. Bacteriology and treatment: a prospective study. Arch Surg 1988;123:745-9.
19. Gaar E. Occupational hand infections. Clin Occup Environ Med 2006;5:369-80.
20. Sokolow C, Dabos N, Lemerle JP, Vilain R. Bacterial flexor tenosynovitis in the hand. A series of 68 cases. Ann Chir Main 1987;6:181-8.
21. Stomberg BV. Retreatment of previously treated hand infections. J Trauma 1985;25:163-4.
22. Weinweig N, Gonzalez M. Surgical infections of the hand and upper extremity: A county hospital experience. Ann Plast Surg

2002;49:621-7.
23. Dailiana ZH, Rigopoulos N, Varitimidis S, et al. Purulent flexor tenosynovitis: factors influencing the functional outcome. J Hand Surg Eur 2008;33:280-5.
24. Post JC, Preston RA, Aul JJ, et al. Molecular analysis of bacterial pathogens in otitis media with effusion. JAMA 1995; 273:1598-604.
25. Spiegel JD, Szabo RM. A protocol for the treatment of severe infections of the hand. J Hand Surg Am 1988;13:254-9.
26. Ellis M. Infections of the hand. Br Med J 1965;4:1353-54.
27. Phipps AR, Blanshard J. A review of in-patient hand infections. Arch Emerg Med 1992;9:299-305.
28. Shamblin WR. The diagnosis and treatment of acute infections of the hand. South Med J 1969;62:209-12.
29. Marcotte AL, Trzeciak MA. Community-acquired methicillin-resistant Staphylococcus aureus: an emerging pathogen in orthopaedics. J Am Acad Orthop Surg 2008; 16:98-106.
30. Gosain AK, Markison RE. Catheter irrigation for treatment of pyogenic closed space infections of the hand. Br J Plastic Surg 1991;44:270-3.
31. Robins RHC. Infections of the hand. J Bone Joint Surg Br 1952;34:567-80.
32. Di Felice A, Seiler JG, Whitesides TE. The compartments of the hand: An anatomic study. J Hand Surg Am 1998;23:682-6.
33. Gutowski KA, Ochoa O, Adams W. Closed-catheter irrigation is as effective as open drainage for treatment of pyogenic flexor tenosynovitis. Ann Plast Surg 2002;49:350-4.
34. Nevasier RJ. Acute infections. In: Green DP, Hotchkiss RN, Pederson WC. Green's Operative Hand Surgery, 4th ed. USA; Churchill Livingstone; 1999. p 1033.
35. Nemoto K, Yanagida M, Nemoto T. Closed continuous irrigation as a treatment for infection of the hand. J Hand Surg Br 1991;18:783-9.
36. Jebson PL. Deep subfascial space infections. Hand Clin 1998;14:557-66.