Infectious flexor hand tenosynovitis: State of knowledge. A study of 120 cases

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ABSTRACT

Introduction: Since Kanavel in 1905, knowledge of phlegmon of flexor tendon sheaths of the fingers have evolved over the twentieth century. This serious infection is 20% of infections of the hand and may have adverse consequences for the function of the finger and even beyond, of the hand. Amputation is always a risk. Frequently face this type of infection, we conducted a retrospective study and made an inventory of knowledge in order to consolidate and improve the overall care.

Materials & Methods: The study was retrospective and cross, focused on 120 patients operated on at Hand Surgery Unit, during 4 years. Inclusion criteria were primary or secondary infection of the sheath of the flexor tendons of the fingers. The evaluation focused on clinical and paraclinical perioperative parameters. At last follow, digital mobility (Total Active Motion), the functional score of QuickDASH and the socio-professional consequences were evaluated.

Results: The mean age was 40 years, with a male predominance. The hospital stay was 17 days on average (3 days to 80 days). From the classification of Michon, as amended by Sokolow, we found 60 Stage I, 48 stage II, 12 stage III. The Total Active Motion was respectively 240°, 140°, 40°. QuickDASH scores were respectively 20, 56 and 90 out of 100. The time for return to work was 1 month for stage I, 4 months for stage II and 12 months for stage III.

Discussion: The long-term functional outcome was generally poor, with stiffness or digital amputation. The poor prognostic factors were: the initial advanced stage of infection, infection beta-haemolytic Streptococcus group A, and delayed surgical management. Smoking was identified as a new risk factor in this disease, as well as diabetes or immunodeficiency. This study confirmed the predominance of Staphylococcus, and scalability of the infection depending on the mode of contamination, and / or type of germ that is to say, scalability schedule for β-hemolytic streptococci group A chronic and scalability for intracellular bacteria (mycobacteria).

Conclusion: Any suspicion of flexor hand tenosynovitis should lead to an emergency surgical exploration, not primary antibiotics prescription!

1. Introduction

Since the initial description by Kanavel in 1905 [1], data collection and modes of management for phlegmons of flexor tendon sheaths have continued to evolve following medical advancements and the advent of antibiotics. A phlegmon is a diffuse infection without a local collection, which is the opposite of an abscess. The word “phlegmon” has its origins in the Greek word *phlégō*, which means *I burn*.

A phlegmon of the flexor tendon sheaths refers to an infection of the

flexor tendons of the fingers, which is caused by infection of the synovial sheets. This severe infection represents 20% of hand infections and may result in complications that compromise the function of the finger, and even that of the hand, if it is not treated quickly. Furthermore, there is always a risk that amputation may be required.

The SOS Hand Unit frequently manages this type of infection. We hereby present a report, discussing the current state of knowledge on phlegmons of tendon sheaths, in order to codify and improve patient management, implement prevention strategies and add to the body of

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Fig. 1. Flexor tendon sheath.

medical knowledge in this area.

We make a distinction between the common synovial sheath of the flexor tendons, which runs from the carpal tunnel to the palm of the hand, the synovial sheet of the long flexor tendon of the little finger, and the digital synovial sheets of the four long fingers [2] (Fig. 1).

The synovial structure, which is also called the synovial sheath of the flexor tendons, is made up of two leaflets: a parietal leaflet and a visceral leaflet, which surround the tendon like a sleeve. The small space thus created between the two leaflets plays an important role in the mechanics of flexor tendon action.

A true communication may exist between these two digitocarpal sheaths at the level of the wrist. Gosselin found this in only 1.67% of cases, but other studies have reported a communication rate of 60%. This variation is the basis for the so-called “*per continuitatem*” phlegmon [3–5]. With respect to vascularisation, Zbrodowski and Guimberteau [6,7], describe the intrinsic vascularisation of the synovial sheaths and flexor tendons.

Three modes of contamination in phlegmon pathophysiology have been identified: direct inoculation, diffusion (a nearby focus of infection) and haematogenous spread.

During history-taking, the following should be elicited from patients: the circumstances of the accident, any predisposing condition (such as diabetes, immunodeficiency, chronic alcoholism, drug abuse), tobacco addiction, anti-tetanus vaccination status, and the usual functional signs (e.g. constant, acute and severe or slowly evolving pain along the length of the sheath of the flexor tendons of the fingers, and the flexed attitude of the finger).

During physical examination, the following should be looked out for: digital oedema, pain with passive extension, pain with palpation of the cul-de-sac of the sheath, epitrochlear or axillary lymphadenopathy, lymphangitis, and general signs (such as fever, chills, malaise). These general signs are not always present and point to severe disease.

No complementary examination is useful for making a diagnosis of a phlegmon of the flexor sheaths - the diagnosis is clinical. However, complementary examinations are useful in the context of diagnostic

Fig. 2. Sheath washing.

uncertainty, in the search for a lesion, for pre-operative evaluation, and to identify a microorganism.

Phlegmons evolve in three distinct phases, which form the basis for Sokolow's modification of Michon's classification [3,4]:

- **Stage I:** Exudative serositis, which distends the sheath; the fluid is clear or turbid and the synovium is simply hyperaemic.
- **Stage IIa:** The synovium is subnormal. There are some localized pathologic areas; there is no justification for a complete synovectomy, but a partial synovectomy (limited to the pathologic areas) may be performed.
- **Stage IIb:** The synovium is pathologic (oedematous, hypertrophic and granulomatous) throughout the length of the digital tunnel; there may also be infiltration of the subcutaneous tissue, which may have septic areas; a complete synovectomy is justified in this situation.
- **Stage III:** Necrosis, with greater or lesser tendon involvement (Fig. 2).

2. Materials and methods

2.1. Materials

This retrospective cross-sectional study was carried out at the orthopaedic and trauma surgery department of the SOS Hand Unit, involving patients who had an operation for treating a phlegmon of the flexor tendon sheaths of the fingers, between 2004 and 2008.

The exclusion criteria in our study were infections of the hand that did not involve the flexor tendon sheaths and non-infectious tenosynovitis. All of the patients were examined in person, except when this was not logistically possible (for example due to distant relocation). In these cases, patients were contacted by telephone.

The preoperative variables studied were: age, handedness, profession, leisure activities, consumption of tobacco or alcohol, past medical history, drug history, allergies, history of the illness (such as the type of wound), the circumstances, fingers affected, time interval before onset of symptoms, mode of management (use of initial antibiotic therapy, NSAIDs (non-steroidal anti-inflammatory drugs) etc.), functional signs (pain, flexion attitude of the finger), local physical signs (pain over the cul-de-sac, pain with extension, neurologic testing, vascularisation), regional physical signs (lymphadenopathy, lymphangitis), general signs (fever, chills, malaise), biological data, bacteriologic data and imaging.

For the purpose of follow-up research, the variables that were studied were: finger sensitivity and mobility (total active motion for the long fingers and Kapandji score [26] for the little finger), the Quick-Dash functional score [27], an abridged version of the DASH (*disability of the arm, shoulder and hand*), and the time interval before return to work or leisure activities.

Fig. 3. Flexor hand necrosis.

2.2. Methods

All of the patients that underwent surgery had standard hospital admission, with timely management (< 6 h). All of the interventions were carried out using a magnifying loupe, with the patient in the *dorsal decubitus* position, under either general anaesthesia or plexus block, on a table under upper extremity regional anaesthesia, with an inflatable pneumatic tourniquet at the origin of the upper limb.

Careful debridement of all wounds was carried out. On the basis of the anatomic and clinical stage, exploration was carried out using a palmar zigzag incision and several manoeuvres could be performed.

In the case of Stage I, if the sheath was closed it was opened up using a scalpel, then a counter-incision was made on the proximal or distal end of the sheath. The sheath was washed with physiologic saline, going from “clean to dirty”, after putting in place a catheter with a large-enough diameter (Cathlon Pink, 20 G x 1-1/4”. Smiths Medical, Luton, UK.) (Fig. 3).

In the case of Stage II, a tenosynovectomy was performed on the entire finger to the greatest extent possible, preserving the pulleys as much as was feasible.

In the case of Stage III, necrotic tissue was excised (tendons and pulleys), and finger amputation was performed when necessary.

In all cases, a local focus of infection (e.g. paronychia, arthritis, osteitis) was treated and multiple samples (at least three) were taken for bacteriology and histology.

The associated lesions (tendinous or neurovascular) were treated simultaneously.

The tourniquet was released before the end of the intervention in order to appreciate the viability of the finger. Skin incisions were usually closed at the level of the articulations. A short period of immobilization was routinely observed, especially in cases that involved tendon or neurovascular repair.

Antibiotic therapy was empirical, with the most frequently used being a combination of amoxicillin and clavulanic acid. This was subsequently adjusted depending on the bacteriologic results. Retraining was started early (beginning from the first dressing). Postoperatively, signs of local (ischemia, recurrence of sepsis), regional (proximal spread of infection) and general (severe sepsis, wound decompensation) complications were watched for - the development of which would lead to a repeat operation on the patient within 48 h.

3. Results

This study included 120 cases of phlegmons of flexor tendon sheaths, operated on at the orthopaedic surgery department of the HEGP, from 2004 to 2008. The mean age of patients was 40 years [17–75 years], with 84 men (70%) and 36 women (30%). The number

of manual workers (63 cases) was similar to the number of non-manual workers (57 cases).

52 patients presented with chronic immunosuppression, which was linked to diabetes (25 cases), cancer (10 cases), HIV (eight cases) and long-term corticosteroid therapy (nine cases). 74 patients were cigarette smokers (62%), with more than five packet years.

Among secondary causes of phlegmons of flexor tendon sheaths, there were two cases of recent A1 pulley surgery (trigger finger), nine cases of exploration of a palmar wound that had been tagged as a “clean wound”, five cases of flexor tendon sutures, seven cases of excision of paronychia and four cases of Dupuytren contracture surgery (minimal-access fasciotomy or open palm technique).

At the level of the hand, the index finger and the middle finger were affected in two out of three cases. Among the medications taken before specialized care, the use of antibiotics and NSAIDs were identified. Antibiotics were prescribed in 70 cases (58%): by the attending physician (54 cases), or by the emergency doctor (13 cases), or by self-medication (three cases). There was a history of ingestion of NSAIDs in 36 cases (30%), more frequently by self-medication (60% of cases).

The time interval between the accident and consultation at a specialist centre was on average 2.5 days [4 h - 4 months]. The average duration of hospital admission was 17 days [1–80 days]. There were 8 cases of intensive care admission, for an average duration of 11 days. The indications for admission to the intensive care unit were severe sepsis with multiple organ failure (six cases), and anaphylactic shock in response to antibiotics (two cases).

From an anatomical and pathological perspective, 60 cases with Stage I (50%), 48 cases with Stage II (40%), and 12 cases with Stage III (10%) disease were identified. Among Stage III cases, there were six cases of partial finger amputation and four cases of total finger amputation.

The number of early repeat surgical interventions (< 5 days) was 15%, regardless of stage. They included extensive tenosynovectomy (eight cases), treatment of another finger during a pre-existing phlegmon per continuitatem (three cases), secondary excision of necrotic tissues up to the wrist (two cases), partial or total amputation for salvage (four cases), covering with an inguinal flap (one case). Repeat surgical interventions were performed in a total of 90% of cases, with Stages II and III managed essentially with tenoarthrolysis (18 cases).

The average duration of post-operative antibiotic therapy was three weeks [8 days - 6 months] and depended on the initial severity, clinical response and the isolated microorganisms.

The average values for “total active motion” were: 240° for Stage I, 140° for Stage II, 50° for Stage III. The QuickDash score was a function of the initial stage of the infection, according to Sokolow’s modification of Michon’s classification. This was assessed in 91 patients. The mean QuickDash score was 42/100 regardless of stage. This corresponded to 20/100 for Stage I, 56/100 for Stage II and 90/100 for Stage III. The time interval to return to work was also classified according to the stage, and on average was one month for Stage I, four months for Stage II and 12 months for Stage III.

Microbial populations were obtained from bacteriologic samples taken preoperatively (blood culture) and peri-operatively (pus, synovial fluid, synovium, necrotic tissue). In 15% of cases no microorganism was found. The most frequently isolated bacterium was *Staphylococcus* (41 cases). *Staphylococcus aureus* accounted for two out of three staphylococcal infections. The other third were infections with *Staphylococcus epidermidis*.

Streptococcus pyogenes, which is also called Group A β -haemolytic *Streptococcus*, was present in 15% of cases (15 cases).

Bacterial co-infection was present in 30% of cases (30 cases). These co-infections were of gram-positive cocci (*Staphylococci* and *Streptococci*) (20 cases) or mixed infections (10 cases) with gram-positive cocci and gram-negative bacilli (*E. coli*, *Proteus mirabilis*).

The other bacterial populations isolated were: *Fusobacterium* (one case), *Pasteurella multocida* (seven cases), *Corynebacterium xerosis* (one

case), *Micrococcus* (one case).

Intracellular microorganisms were identified in this study in six cases: *Mycobacterium tuberculosis* (four cases) and *Mycobacterium marinum* (two cases).

The proportion of smokers in this study was 62% (74 cases). A smoker was defined as a patient with more than five packet years. Within the group of smokers, a higher rate of Stage II compared to Stage I disease was found (37 patients with Stage II or 50% of cases, and 30 patients with Stage I disease, or 40% of cases), using Sokolow's modification of Michon's classification. By considering only Stage I and II, it was noted that delayed wound healing was present in smokers, with an average of 27 days compared to 18 days in non-smokers. The rate of early repeat surgical intervention was also doubled in the smokers group when only Stages I and II were considered (12 versus six cases). Additionally, the average QuickDash functional score in patients who smoked was 10% lower than the QuickDash score of non-smoking patients (67 versus 75/100). However, the microorganisms that were found in the smokers' group were similar to those found in non-smokers.

Among the diabetic and immunodeficient population, the peri-operative assessment found 20% of patients with Stage I disease (10 cases), 60% of patients with Stage II disease (31 cases) and 20% of patients with Stage III disease (11 cases). Regardless of stage, the QuickDash functional score was 20% lower in patients with comorbidities compared to the group without (55 versus 69/100). Among the diabetic patients, there was a higher rate of polymicrobial infections (two out of three, or 35 cases). Among the immunodeficient patients (with HIV, cancer or long-term corticosteroid therapy), there was a higher rate of slow-growing organisms: *Mycobacterium tuberculosis* (three cases), *Fusobacterium* (one case).

4. Discussion

Among the causes of primary phlegmons of the flexor sheaths, direct inoculation was the most frequent cause identified [8,9]. The penetrating agent was frequently metal (65% of cases), followed by plant matter (17% of cases), then agents from aquatic and animal environments [19].

Amongst the causes of secondary phlegmons of the synovial sheaths (secondary to surgery) were 1) surgery of the flexor tendons or their pulleys, 2) surgery of soft tissue paronychia (increasing the risk of phlegmons of the flexor sheaths, which required special attention during excision) 3) Dupuytren contracture surgery requiring flap cover.

Additionally, 9 wounds which were identified as "clean" resulted in secondary flexor sheath phlegmons. This suggests the possibility of invisible micro-lesions of the flexor sheaths, which would have the same consequences as an open wound. Therefore, in the case of a palmar wound over the flexor tendons, we may therefore consider it acceptable to wash the flexor tendon sheath even when there are no visible lesions.

Studies by Zbrodowski and Guimberteau have demonstrated the presence of extensive vascularisation of flexor sheaths and flexor tendons [6,7]. We suggest that the disruption of this process of vascularisation leads to amplification of the infection, which may result in necrosis. Two mechanisms could result in the disruption of vascularisation. In the first stage, bacterial multiplication and the action of their toxins lead to septic micro-thromboses. In the second stage, the equivalent of a "compartment syndrome of the finger" occurs, secondary to elevated intra-digital pressure, resulting in spasms of the micro-vasculature [10]. The need for emergency surgery can therefore be appreciated, with an attempt at decompression required.

It should be noted that tobacco consumption increases the severity of the identified infection and delays wound healing. We consider two tobacco-linked pathophysiological hypotheses: digital microangiopathy and immune dysfunction [11,12] through disorganisation of lymphocytes.

A fulminant clinical picture leads to a clinical diagnosis of phlegmon

of the flexor sheaths of the flexor tendons. However, in cases of chronic infection (intracellular microorganism such as mycobacteria), the picture is less clear. We then talk of suppurative tenosynovitis. In all cases, any iota of suspicion of a diagnosis of phlegmon of the flexor sheaths must lead to surgical exploration. Importantly, the clinical (fever) and biologic (hyperleucocytosis and CRP greater than 5) markers of inflammation are not always present. Absence of these markers, therefore, does not rule out the diagnosis of phlegmon of the flexor sheaths. Imaging, such as ultrasonography or MRI, must not in any case delay the diagnosis [13] but are useful in cases of an atypical picture, or to localize a foreign body. Finally, we believe that samples should be taken for blood culture in all cases of clinical diagnosis and despite the absence of fever, because they may contribute to the bacteriologic diagnosis, particularly in cases of haematogenous spread.

The microbiology of infectious tenosynovitis varies with the mode of contamination. With contamination that occurred by direct inoculation (for example a needle-prick or wound), the microbial flora corresponded to the flora present on the skin. Thus, Staphylococci (*Staphylococcus aureus* and *Staphylococcus epidermidis*) and β -haemolytic and non β -haemolytic Streptococci were isolated.

However, a polymicrobial flora was typically found after a bite. In cases involving a human bite, Staphylococci, Streptococci, *Eikenella corrodens* [14], Haemophili, Neisseriae as well as anaerobic bacteria like Peptostreptococci, Veillonellae, fusobacteria and bacteroids were identified.

Animal bites had a similar microbiologic profile, with risk of infection by *Pasteurella multocida* (a gram-negative coccobacillus, responsible for rapid evolution and significant lymphangitis), *Capnocytophaga*, Bacilli, Protei, Pseudomonades, Actinomyces and *Leptotrichia buccalis*.

Tenosynovitis resulting from haematogenous spread essentially involved Gonococci (*Neisseria gonorrhoeae*) [15], and certain Mycobacteria.

It is worth noting that other bacteria can infect the flexor sheath, and cause development of a low-grade chronic infection.

These microorganisms are some atypical mycobacteria such as *M. marinum*, *M. kansasii*, *M. asiaticum*, *M. avium*, *M. fortuitum*, *M. chelonae* and *M. abscessus*. Thus, we use the term "infectious tenosynovitis". *Mycobacterium tuberculosis* is sometimes responsible for tenosynovitis [16] and it frequently, but not always, has its origins in an active pulmonary tuberculosis. Tenosynovitis that have a fungal origin, however, remain exceptional and are discussed in few publications. These occur in the context of immune deficiency.

Although hand infections are not rare in children, there are very few publications about infectious tenosynovitis in children. The essential factor in such bacterial infections in children is often paronychia. Among the bacterial population, the most frequent causal microbe in children is *Staphylococcus aureus* (40%), as is the case in adults (50–80%).

Group A β -haemolytic Streptococci [18] are found more frequently in children than in adults (20%). Co-infections with aerobic and anaerobic bacteria are of the same proportions in children as in adults (30%), despite adults more often having factors for mixed infection (e.g. bite wound, intravenous drug use etc.). There is currently no explanation for the data on mixed infections in children, apart from local (sucking of the little finger, biting of nails), or general (immaturity of the immune system) causes. However, there is a rare cause with severe consequences which must be noted, because it is particular to children. This is rupture of the flexor sheath after complete epiphyseal separation of the distal phalanx (door-crush finger injury) [17].

Recent epidemiologic and microbiologic research found that in France, two thirds of *Staphylococcus aureus* are sensitive to Augmentin (amoxicillin and clavulanic acid). On the contrary, the trend is the inverse in the United States [19,20], where two thirds of *Staphylococcus aureus* are resistant to Augmentin.

These microbiological data allow for the administration of empirical

antibiotic therapy, while definitive microbiologic results are awaited. At the national level, there is really no consensus on specific antibiotic therapy for phlegmons of the flexor sheaths.

In practice, the first choice of empirical antibiotic therapy is always Augmentin. In the case of allergy, we suggest the use of macrolides (Clindamycin), or Pristinamycin. In case there are signs of severe disease, antibiotic therapy can be dual therapy (Augmentin and Clindamycin or a Fluoroquinolone), or monotherapy with a second generation cephalosporin (Cefamandol). In case polymicrobial infection is suspected, it is acceptable to administer dual antibiotic therapy (Augmentin and a Fluoroquinolone or Augmentin and Gentamycin). Antibiotic therapy can then be subsequently adjusted according to the bacteriologic results [21].

With respect to the duration of treatment, studies on treatment duration are rare. We think that the duration of effective antibiotic therapy should be between 10 days and three weeks, depending on the initial stage and the response to treatment.

Our study has led to the observation that the evolution of an infection could depend on the microorganism present. With Group A β -haemolytic streptococcal infections, there is rapid evolution of infection. This is linked to the pathogenicity of the bacterium itself - its capsule and membrane proteins protect it from phagocytosis by polymorphonuclear neutrophils. It adheres to epithelial cells of the host and, by producing exo-enzymes (exotoxins), contributes to the extension of the lesions by tissue destruction and toxic shock. Similarly, infections with *Pasteurella multocida* are rapidly evolving (less than 24 h) and manifest by severe pain and extension of regional inflammation (lymphangitis and lymphadenopathy).

Inversely, with chronic disease the bacterial distribution tends to be disposed toward microorganisms that develop intracellularly (e.g. Gonococci and Mycobacteria). In such cases, the delay between the lesion and the onset of symptoms may be several weeks or months.

Once again, the appearance of an antibiotic-resistant bacterial strain arises from the problem of antibiotic prescription. Nowadays, too many antibiotics are prescribed blindly - particularly in cases where there is suspicion of a phlegmon of the flexor sheaths. Therefore, blind prescription has two consequences: 1) a modification of the clinical picture and 2) delaying diagnosis and surgical management. This increase in the delay before management leads to a poorer prognosis, the selection of a resistant strain of bacteria and compromised effectiveness of antibiotics, which is a real public health problem. Essentially, sensitization about antibiotic prescription must involve all of the medical community, particularly those in the first line of contact (e.g. general practitioners and emergency physicians).

Secondly, self-medication without medical advice should be discussed, particularly regarding the ingestion of NSAIDs. With respect to the use of NSAIDs, the results are variable from study to study. However, these studies recommend prudence in using NSAIDs due to their biologic effect of inhibiting immunity [22].

Finally, our study confirms the need to continue our efforts towards the prevention of risk factors such as cigarette smoking and sexually transmitted infections (STIs) (particularly gonococcal infections). However, minimizing the spread of the bacteria remains the best method of prevention.

The functional prognosis of the patient depends on several factors: the initial stage of the phlegmon during peri-operative assessment, risk factors (including smoking, diabetes and immunosuppression) [23–25] and the delay before surgical management (especially for rapidly evolving infections such as those caused by Group A β -haemolytic Streptococcus).

The principal sequelae were stiffness of the finger or amputations, and this essentially involved Stage II and III cases. Thus, it is vital that any suspicion of a phlegmon of the flexor sheaths must lead to emergency surgical exploration, in order to arrest the aggravation of the infection. There is also a need to be able to modify the operation plan for this emergency. It is important to note that there is also an

occupational impact, with increasing inability to work depending on the severity of the phlegmon. Here, cigarette smoking was identified as a new risk factor, with phlegmons of the flexor sheaths more severe in smokers, with more significant functional consequences.

It is therefore important that the patient be given complete information regarding the severity of phlegmons of the flexor sheaths on a functional level. This information should address: his/her surgical and medical management (long duration of antibiotic therapy), the risk of amputation, the risk of early or medium-term repeat surgical intervention, long-term rehabilitation, the risk of functional sequelae, the need to stop cigarette smoking and the risk for infection in the case of elective hand surgery.

5. Conclusion

A phlegmon of flexor sheaths is a severe infection of flexor tendon sheaths of the fingers, which may impact on the survival and functional prognosis of the patient. It is more often due to direct inoculation of cutaneous microorganisms (Staphylococcus and Streptococcus) or diffusion from a local sepsis (paronychia), but may result in haematogenous spread of bacteria (Gonococci and certain Mycobacteria, including tuberculosis).

Cigarette smoking was identified as a new risk factor on the same level as diabetes or immunodeficiency and was found to increase the risk of a more severe and protracted infection. Our study elaborates a little bit more on the major role of the vascularisation of flexor tendons and their sheaths in the pathophysiology of phlegmons of the flexor sheaths.

The clinical results show that long-term prognosis depends on several factors such as the initial stage of the infection, the responsible microorganisms and time interval to surgical intervention. From a medicolegal viewpoint, we suggest that the patient should always be counselled about the severity of this infection and the uncertainty about the functional prognosis in spite of appropriate treatment.

Furthermore, this work demonstrates the gaps in knowledge about this severe infection in the medical community. Thus, any suspicion of a phlegmon of the flexor sheaths warrants an emergency surgical exploration, and not a blind prescription of antibiotics. The outcome of surgical exploration is an improvement of the functional prognosis through rapid and specialized management, and targeted use of antibiotics to limit the emergence of resistant bacteria - a real public health problem which needs to be addressed.

Conflicts of interest

none

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