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Review

Management of septic bursitis

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ABSTRACT

Superficial septic bursitis is common, although accurate incidence data are lacking. The olecranon and prepatellar bursae are the sites most often affected. Whereas the clinical diagnosis of superficial bursitis is readily made, differentiating aseptic from septic bursitis usually requires examination of aspirated bursal fluid. Ultrasonography is useful both for assisting in the diagnosis and for guiding the aspiration. *Staphylococcus aureus* is responsible for 80% of cases of superficial septic bursitis. Deep septic bursitis is uncommon and often diagnosed late. The management of septic bursitis varies considerably across centers, notably regarding the use of surgery. Controlled trials are needed to establish standardized recommendations regarding antibiotic treatment protocols and the indications of surgery.

1. Introduction

Bursitis is a common reason for seeking medical help, notably via emergency room visits. The olecranon and prepatellar bursae are the most often involved sites, as their superficial location exposes them to injury [1]. About one-third of cases of olecranon and prepatellar bursitis are septic [2]. The annual incidence of olecranon and prepatellar bursitis has been estimated at 0.1/1000 population [2]. Septic olecranon and prepatellar bursitis has been reported to account for 0.01% to 0.1% of all hospital admissions [1]. These numbers may underestimate the true incidence of septic bursitis, as only the most severe forms require admission [1,3].

The management of septic bursitis varies widely due to the absence of recommendations about antibiotic and surgical treatments. This article reviews important anatomical, clinical, and bacteriological features then discusses published data on the pharmacological and surgical treatment of superficial septic bursitis.

2. Risk factors

Among patients with bursitis, 80% are males aged 40 to 80 years [3], who constitute the population most exposed to trauma and micro trauma during manual labor or recreational activities [4]. In one study, the incidence of septic olecranon bursitis was found to increase during the summer due to greater engagement in outdoor activities [4]. Contamination of superficial bursae by microorganisms usually occurs through the skin, either by direct inoculation during an injury, aspiration, or injection or via spread from a skin infection [5]. Hematogenous contamination is exceedingly rare, as the vascular supply to superficial bursae is meagre [3,6]. Deep septic bursitis is far less common and produces misleading manifestations that result in diagnostic delays [5,6]. Septic ischial bursitis has been reported in patients with spinal cord injury and in those who engage in activities responsible for an increase in local pressure, such as weaving [5,7]. Contamination can occur during glucocorticoid injections into the deep sub acromial/sub deltoid and trochanteric bursae [8,9]. When no cause is obvious, deep septic bursitis should be ascribed to hematogenous contamination or to spread from a neighboring infection such as arthritis, osteomyelitis, or a soft tissue infection [6]. General health factors that increase the risk of septic bursitis include diabetes, chronic alcohol abuse, glucocorticoid therapy, and rheumatoid arthritis [3,10]. Reported proportions of patients with at least one comorbidity range from 33% to 74% [2,3]. An association between septic bursitis and protease inhibitor therapy to treat hepatitis C virus infection has been suggested [11]. Finally, although several cases of septic bursitis have been described in patients with HIV infection, no proof exists

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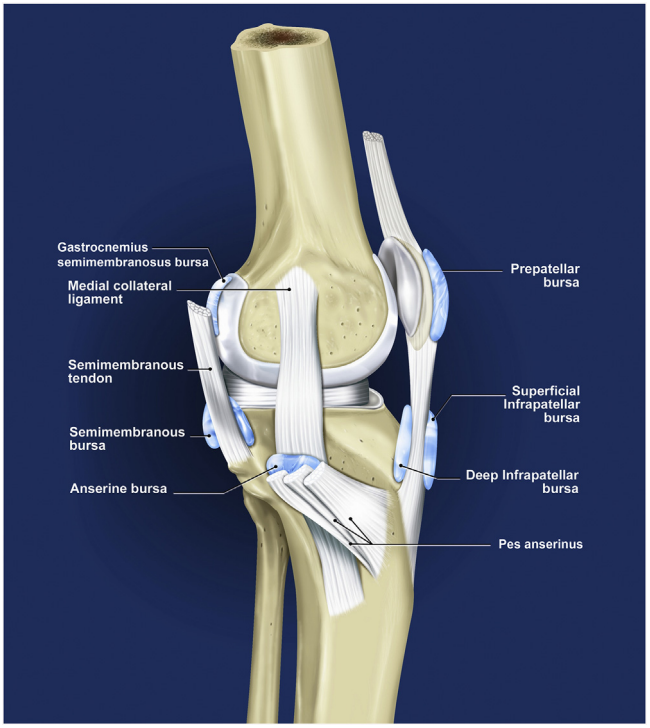

Fig. 1. Anterior and medial bursae about the knee.

that HIV-positive patients are at increased risk for septic bursitis [5,12].

3. Anatomy

Bursae are closed extra articular sacs that decrease friction between soft tissues and bony protuberances [13]. They are composed of connective tissue with a synovial lining [13]. Bursae may be superficial (e.g., the olecranon prepatellar, and infrapatellar bursae) or deep (e.g., the ischial, sub acromial, and retrocalcaneal bursae). The superficial bursae develop after birth (e.g., between 7 and 10 years of age for the olecranon bursa) and are inconsistently present [14,15]. Adventitial bursae, which lack a synovial lining, develop in response to excessive soft-tissue friction over bone [15], due for instance to hallux valgus. The number of bursae in the body has been estimated at over 150, although differences occur due to variations in the development of adventitial bursae and to the inconsistent existence of some of the superficial bursae [1,16].

The extensor apparatus of the knee includes three bursae, the prepatellar bursa and the superficial and deep infrapatellar bursae (Fig. 1). The superficial infrapatellar bursa is inconsistent, as shown by its identification in only 55% of participants in one study [17]. It lies over the distal third of the patellar tendon and may communicate with the prepatellar bursa [17,18]. Bursae may communicate with the adjacent joint cavity; examples include the iliopsoas bursa, as well as the gastrocnemius-semimembranosus bursa [15], whose distension results in a popliteal (Baker's) cyst. Whether the deep infrapatellar bursa can communicate with the knee joint is debated [15,18].

4. Clinical diagnosis of septic bursitis

The symptoms produced by superficial septic bursitis consist of pain and functional impairment of variable intensity [16]. Physical findings may include local swelling, warmth, and erythema, which are not specific of infection [2,3]. Deep septic bursitis produces even less specific physical findings that vary with the location of the bursa [16]. A fever suggests infection but is inconsistent, being reported in 20% to 77% of patients [3].

The clinical features may be insufficient to distinguish septic bursitis from bursitis due to crystal deposition disease, inflammatory rheumatic disease, or trauma [2]. Bursal effusion, cutaneous erythema, and pain are not sufficiently specific [1]. Similarly, neither a fever nor the presence of skin abrasions is sufficiently sensitive or specific to establish a diagnosis of septic bursitis [3]. A review of 55 publications concluded that the cause of bursitis cannot be determined based on clinical findings alone [2]. The risk of confusion is particularly great in patients with fungal septic bursitis, which can mimic a mechanical condition and run a chronic course. A review of the literature on candidal bursitis identified 10 cases, of which 6 were diagnosed only 4 to 18 months after symptom onset and 4 occurred in immunocompetent individuals [19].

Given the lack of specificity of the clinical findings, aspiration to allow examination of the bursal contents is usually advocated [1,2,5]. In doubtful forms, antibiotic treatment started immediately after the aspiration and continued until the microbiological results are available has been suggested [5]. Nevertheless, the appropriateness of routine aspiration in patients with mild manifestations has been challenged [20]. Indeed, the effectiveness of empirical antibiotic therapy in the vast majority of cases means that the availability of microbiological data rarely affects the choice of antibiotics or the clinical course [21]. Consequently, bursal fluid aspiration and analysis is not universally advocated. In a retrospective study of 118 patients with olecranon bursitis treated on an outpatient basis, only 38% of patients underwent aspiration [4]. However, this approach has been criticized as a possible source of diagnostic mistakes and unwarranted antibiotic treatments [3]. Bursal aspiration must be performed by an experienced operator, as inserting the needle too deeply can cause septic arthritis [16]. Repeating the aspirations until bursal fluid cultures revert to negative or the bursal effusion resolves has been suggested [2,6].

Confusion between septic bursitis and septic arthritis is not extraordinarily uncommon. A careful physical examination is usually successful in differentiating the two conditions. Passive motion of the underlying joint is preserved in the event of septic bursitis, whereas septic arthritis results in severe functional impairment and in an intra-articular effusion [22]. Erysipelas is the main differential diagnosis of septic bursitis. The absence of bursal effusion in erysipelas is an important distinguishing feature [20]. However, the two conditions may coexist, as erysipelas may complicate septic bursitis and vice versa [22–24].

5. Microbiology

Staphylococcus aureus (S. aureus) is responsible for 80% of cases of septic bursitis [4,5,25–27]. The *S. aureus* strains recovered in Europe are usually susceptible to methicillin. In a study from Switzerland, *S. aureus* was identified in 217 of 343 patients with septic olecranon or prepatellar bursitis, and was resistant to methicillin in only 3 patients [28]. Methicillin-resistant *S. aureus* (MRSA) is more often responsible for septic bursitis in the US [3]. Thus, in a study conducted in Boston, *S. aureus* was the causative organism in 39 of 44 patients with septic bursitis and 7 of the strains were resistant to methicillin [27]. Two cases of subacromial bursitis due to MRSA reported in the UK in 2015 developed after local glucocorticoid injections [8]. Streptococci are the second most common causes of septic bursitis [3,5,28]. Coagulase-negative staphylococci, enterococci, *Escherichia coli*, *Pseudomonas aeruginosa*, and anaerobic bacteria have been reported less often [5,20,25]. More than one microorganism was identified in 10% of patients in one study [5].

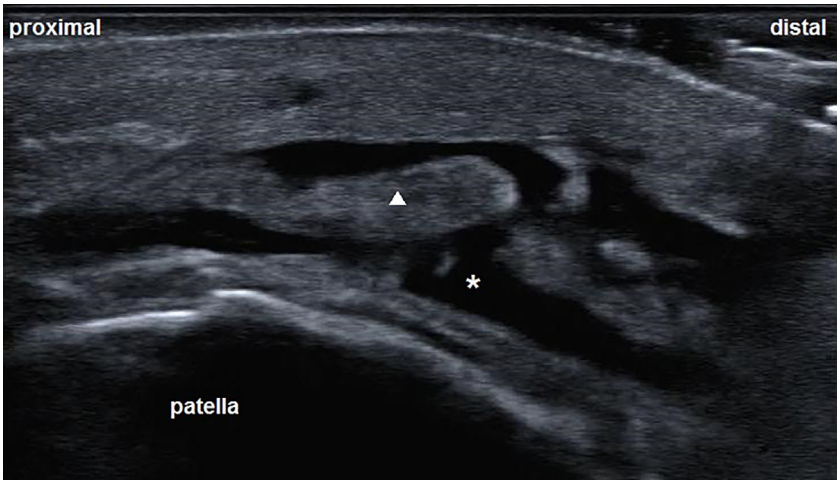

Fig. 2. Ultrasonography in a patient with prepatellar septic bursitis, longitudinal view: note the anechoic bursal effusion (asterisk) and synovial lining hypertrophy (arrowhead).

Atypical mycobacteria is a rare cause of septic bursitis seen chiefly in association with immunodepression, although cases in immunocompetent patients have been reported [29]. Tuberculous infection of the trochanteric bursa, although classical, is a rare event that accounts for less than 2% of tuberculous osteoarticular infections [30]. The frequently misleading clinical presentation and insidious course may delay the diagnosis. Despite the current slow decline in the incidence of tuberculosis throughout the world, cases of tuberculous trochanteric bursitis are still reported regularly [30]. Anecdotal case-reports of septic arthritis due to *Brucella*, *Propionibacterium acnes*, *Prototheca*, *Roussoella percutanea*, and syphilis have been published [31–35].

6. Diagnostic workup

Anteroposterior and lateral radiographs of the adjacent joint are often recommended in patients with superficial septic bursitis [2,3,36,37]. However, it has been suggested that routine radiographs may be unnecessary when the diagnosis of superficial bursitis is obvious, except in the event of trauma or when a foreign body is suspected [38]. Soft tissue swelling and subcutaneous fat stranding are the main radiographic features [36]. A foreign body may be visible. The underlying bone cortex may be abnormal, for instance by the presence of enthesophytes, which are common in olecranon bursitis [3]. More rarely, gas bubbles may be visible, indicating an infection by a gas-producing microorganism [37].

Ultrasonography has several advantages for the diagnosis and management of septic bursitis [2,36,37,39,40]. Swelling of the bursa with an effusion or peripheral edema may be found, as well as thickening of the bursal wall or inflammation of the surrounding soft tissues (Fig. 2) [37]. Ultrasonography may detect a foreign body or features that assist in the differential diagnosis such as a tophus, a rheumatoid nodule, or calcifications [40]. The effusion due to septic bursitis may be thick and echogenic, mimicking hypertrophy of the bursal synovial lining [37]. In this situation, the compressible and mobilizable nature of the image and the absence of increased blood flow by power Doppler support a diagnosis of bursal effusion [15]. Ultrasonography cannot distinguish septic bursitis from bursitis due to crystal deposition disease, inflammatory rheumatic disease, or chronic posttraumatic alterations [37,41]. The rare grain-of-rice appearance produced by the presence of small echogenic structures disseminated throughout the bursal cavity suggests tuberculosis but is not specific and has also been reported in patients with rheumatoid arthritis [37,39,41]. Gas bubbles may produce echogenic foci [39]. Aspiration of the bursa can be facilitated by using ultrasonography to identify the bursa or to guide the needle insertion (Fig. 3) [36,42].

Magnetic resonance imaging (MRI) is rarely indicated for the diagnosis of superficial septic bursitis but may be helpful to look for an abscess or for evidence of osteitis in patients with severe manifestations [43]. However, MRI does not perform better than ultrasonography for differentiating septic from aseptic bursitis [37,39,43]. The presence of gas bubbles seen as signal-free areas on all MRI sequences supports a diagnosis of septic bursitis [39].

In patients with deep bursitis, in contrast, computed tomography and/or MRI is in order to confirm the presence of a bursal effusion and to look for evidence of an abscess or of concomitant involvement of the bone and/or joint (Fig. 4) [44].

The gross appearance of the bursal fluid provides diagnostic orientation. Clear yellow or blood-tinged fluid suggests aseptic bursitis and cloudy or purulent fluid septic bursitis [2]. Bursal fluid cell counts are usually higher in septic than in aseptic bursitis, although no cutoff for definitively distinguishing the two conditions is universally agreed on. A literature review on olecranon bursitis indicated that the white blood cell count in bursal fluid ranged from 690 to 418,000/mm^3 in septic bursitis and from 50 to 10,000/mm^3 in aseptic bursitis [3]. In a study of 36 patients with olecranon or prepatellar bursitis, a white-blood-cell cutoff of 2000/mm^3 was 94% sensitive and 79% specific for diagnosing septic bursitis; however, the wide confidence intervals for both parameters deserve noting [45].

Bursal fluid must be cultured even when found to contain monosodium urate crystals, as an episode of gout may coincide with a bacterial infection [5,40,46]. Consequently, the antibiotic treatment is best continued until the culture results are available [20]. Calcium pyrophosphate crystal bursitis is rare and, to our knowledge, no cases coinciding with bacterial infection have been reported [47].

The diagnostic yield of blood cultures is low, as bacteremia is relatively uncommon during superficial septic bursitis. The rate of blood culture positivity varied across studies from 4% to 30% [6,28,48]. The results of bursal fluid Gram staining vary even more widely, with positivity rates ranging from 15% to 100% [10,44]. In studies confined to septic olecranon bursitis, smear microscopy was positive in one-half to two-thirds of patients [3,5]. Cultures of fluid from septic bursitis identified the causative microorganism in 67% to 100% of patients [6,25,28,45,49,50]. Inoculation of the bursal fluid into blood culture media may increase the diagnostic yield. This

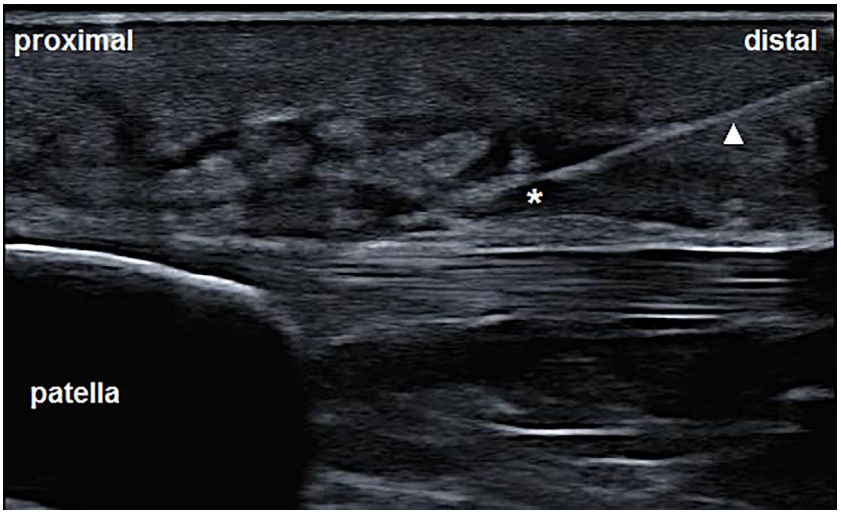

Fig. 3. Aspiration in a patient with prepatellar septic bursitis, with the needle inserted longitudinally along the axis of the probe; the needle (arrowhead) is visible within the bursal effusion (asterisk).

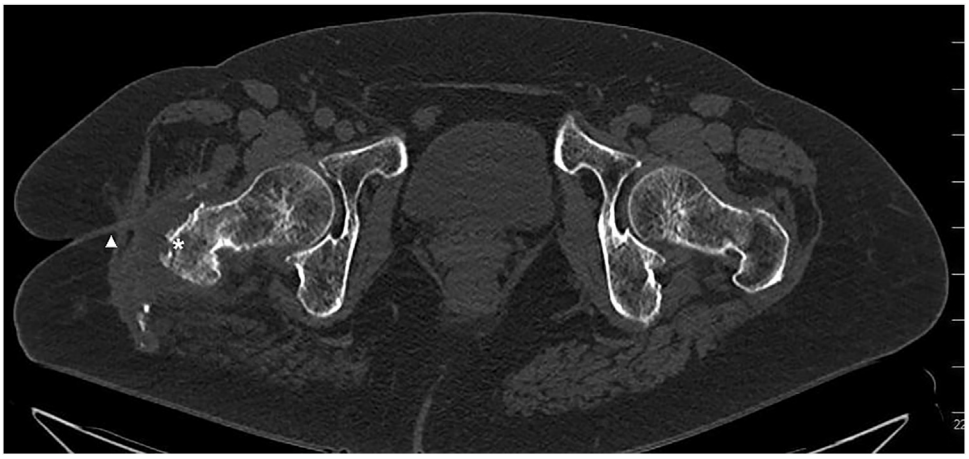

Fig. 4. Computed tomography in a patient with tuberculous trochanteric bursitis, osteitis (asterisk), and a sinus tract (arrowhead).

Table 1
Severity classification of septic bursitis (from Ho and Su [52]).

Severity grade		Treatment
Severe	Extensive infection with marked erysipelas or an infected wound, systemic signs including a fever and rigors, and peripheral blood white blood cell count > 10,000/mm^3	Admission and intravenous antibiotic therapy
Moderate	Moderately severe local inflammation, with or without minor skin breaching and systemic signs	Oral antibiotic therapy and outpatient follow-up
Mild	Mild-to-moderate local inflammation, usually without skin breaching or systemic signs	Oral antibiotic therapy and outpatient follow-up

technique consistently identified the causative microorganism in a study of 17 patients [45].

7. Antibiotic therapy

No recommendations on the antibiotic treatment of septic bursitis have been issued by scientific societies. A questionnaire survey of infectious disease specialists and surgeons in Switzerland, reported in 2013, identified considerable variability in treatment practices [51].

In patients with suspected septic bursitis, empirical antibiotic therapy effective against staphylococci and streptococci is advisable and should ideally be started only after aspiration of the bursa [2,3,49]. Prompt antibiotic therapy initiation decreases the time needed to sterilize the bursal fluid [6,52]. Reported data on the route of administration, duration, and selection of antibiotics are conflicting. A scheme for classifying olecranon and prepatellar bursitis as mild, moderate, or severe has been suggested to guide the management, including the antibiotic treatment (Table 1) [52]. However, this classification does not consider comorbidities that may affect healing or the immune response, such as diabetes and rheumatoid arthritis. A modification consisting in increasing severity by one level in patients with such comorbidities has been suggested [53]. This is an important point, as oral antibiotic therapy has a higher failure rate in immunocompromised patients [10,48]. Furthermore, a study has demonstrated that the duration of antibiotic therapy needed to eradicate the infection is longer in immunocompromised patients [6].

The antibiotic regimen is therefore usually tailored to the severity of the clinical presentation and to the comorbidity profile. Oral antibiotic therapy can be used in patients with mild-to-moderate septic bursitis and normal immune function [2,5,10,49]. In patients with immunodepression or systemic evidence of infection, admission and initial antibiotic administration via the intravenous route are recommended [1,2,5,6,10,49]. However, available data on the compared effectiveness of oral and intravenous antibiotic therapy come only from studies that were done many years ago and did not consider the bioavailability of each antibiotic [54]. Clindamycin, for instance, which is fairly rarely used in France to treat septic bursitis, is highly bioavailable by the oral route [55].

The antibiotics used vary across countries and centers. In Germany and Austria, a penicillinase-resistant penicillin or first-generation cephalosporin has been advocated for the empirical antibiotic treatment of septic bursitis [2]. In a recent prospective study that included 164 patients admitted for septic bursitis in Switzerland, the most often used antibiotic was amoxicillin/clavulanic acid, followed by cefuroxime then by clindamycin [56]. Of 82 patients with severe septic bursitis included in a retrospective study from Spain, 25 were given cloxacillin alone in a dosage of 2 g every 4 hours intravenously until an improvement was obtained followed by 1 g/6 h orally until complete resolution, a regimen that proved highly effective [49]. The American Sanford Guide of infectious disease recommendations indicates that a penicillin M or cefazolin should be used if the causative microorganism is a methicillin-susceptible staphylococcus [57].

The duration of antibiotic therapy also varies widely across publications. A 12-day course was recommended [46] by a group that subsequently reported a mean time to bursal fluid sterilization of 4 days and a 100% infection eradication rate when the antibiotic treatment was continued for 5 days after culture reversion to negative [52]. However, the duration of antibiotic therapy is usually adjusted based on the clinical course and is generally 2–3 weeks. Oral antibiotic therapy for 2 weeks has been suggested in patients with mild or moderate septic bursitis [2,5,10,49]. In contrast, in those with severe septic bursitis or compromised immune responses, admission is recommended, as well as intravenous antibiotic therapy for the first 4–10 days followed by oral antibiotic therapy for a mean of 2 weeks [1,2,4–6,10,49].

No consensus exists regarding the optimal duration of antibiotic therapy after surgery for septic bursitis. A shorter antibiotic therapy duration than usually prescribed in this situation has been advocated [28,49,51,56]. A retrospective study of 343 immunocompetent patients with severe olecranon or prepatellar septic bursitis requiring admission, including 91% who were treated surgically, suggested similar recovery rates with 7 days compared to 14 or 21 days of antibiotic therapy [28]. A randomized trial comparing one- vs. two-stage bursectomy for olecranon or prepatellar bursitis in 164 patients also showed that a 7-day antibiotic course was effective [56]. Thus, 7 days of antibiotic therapy may be sufficient in immunocompetent patients [2]. Shortening the duration of antibiotic treatments is among the measures recommended to combat the development of resistant strains [58].

8. Surgical treatment

The role for surgery in the treatment of septic bursitis is not well standardized. Surgery is often considered as a second-line treatment for refractory or recurrent septic bursitis [2,4,5,22]. However, a survey of infectious disease specialists and surgeons in Switzerland showed that 85% of respondents were in favor of first-line surgery for the treatment of olecranon or prepatellar bursitis [51]. First-line surgery is sometimes advocated in patients with evidence of severe infection, skin complications, foreign bodies, depressed immunity, or unfeasible needle aspiration [2,6,49,53].

Outcomes of surgery vary across studies. In the study from Switzerland of 164 patients with moderate-to-severe olecranon ($n = 130$) or prepatellar ($n = 34$) septic bursitis treated by first-line bursectomy, the failure rate was 13% [56]. A literature review of olecranon and prepatellar bursitis showed failure rates of 20% after bursectomy compared to 0% to 14% after pharmacological treatment only, as well as a higher complication rate in surgically treated patients [2]. Similarly, a review of 29 studies of olecranon bursitis (including 15 of septic bursitis), also suggested that surgery was less effective and produced more complications [26]. Although both these literature reviews were based on low-powered studies, they support first-line non-operative treatment.

The most classical surgical technique is incision and drainage, although bursectomy has been advocated [3,5]. The main complications are delayed healing, painful scars, local hypoesthesia, and recurrence [2]. In the prospective study from Switzerland, 20% of the 164 patients experienced postoperative symptoms consisting of pain, motion range limitation at the knee, and loss of strength at the upper limb [56]. Bursectomy is performed by open surgery or, more rarely, endoscopy [59]. Advantages of endoscopic bursectomy may include a lower morbidity rate, better cosmetic outcomes, and expedited functional recovery [60,61].

9. Management strategy in practice

Although the available data do not allow the development of recommendations based on a high level of evidence, the management strategy below can be suggested for patients with septic bursitis:

- microbiological sampling followed by empirical antibiotic therapy effective against staphylococci and streptococci, i.e., penicillinase-resistant penicillin or first-generation cephalosporin [2,3,62] or, in patients with known hypersensitivity, clindamycin [62];
- for mild-to-moderate septic bursitis, oral antibiotic therapy for 2 weeks on average [2];
- for severe septic bursitis and in immunocompromised patients, admission and intravenous antibiotic therapy for the first 4–10 days on average [1,2,4–6,10,49] followed by a switch to the oral route after the fever resolves and the local abnormalities and inflammatory syndrome abate; cefazolin 2 g every 8 hours can be used as the first-line antibiotic [57]; total antibiotic duration is 2–3 weeks;
- surgery should be considered in patients with refractory or recurrent septic bursitis [2,4,5,22];
- after bursectomy, a 7-day antibiotic course is sufficient in immunocompetent patients [28,57].

10. Conclusion

Treatment practices vary widely in patients with septic bursitis. The relevant literature consists chiefly of anecdotal case-reports and small single-center retrospective observational studies. No controlled trials have compared antibiotic treatment strategies that differ regarding the nature of the antibiotic, route of administration, and/or duration. Neither are any studies available that compare surgery to antibiotic therapy alone. Well-designed studies are needed to answer the unresolved issues and to identify the most cost-effective strategy. The role for ultrasonography in the diagnosis and follow-up of patients with septic bursitis also deserves to be investigated.

Disclosure of interest

The authors declare that they have no conflict of interest.

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