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Recommendations and metaanalyses

2023 French recommendations for diagnosing and managing prepatellar and olecranon septic bursitis

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ABSTRACT

Septic bursitis (SB) is a common condition accounting for one third of all cases of inflammatory bursitis. It is often related to professional activities. Management is heterogeneous and either ambulatory or hospital-based, with no recommendations available. This article presents recommendations for managing patients with septic bursitis gathered by 18 rheumatologists from the French Society for Rheumatology work group on bone and joint infections, 1 infectious diseases specialist, 2 orthopedic surgeons, 1 general practitioner and 1 emergency physician. This group used a literature review and expert opinions to establish 3 general principles and 11 recommendations for managing olecranon and prepatellar SB. The French Health authority (Haute Autorité de santé [HAS]) methodology was used for these recommendations. Designed for rheumatologists, general practitioners, emergency physicians and orthopedic surgeons, they focus on the use of biological tests and imaging in both outpatient and inpatient management.

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Antibiotic treatment options (drugs and duration) are proposed for both treatment modalities. Finally, surgical indications, non-drug treatments and prevention are covered by specific recommendations.

1. Introduction

The olecranon and prepatellar bursae are superficial bursae that allow the skin and musculoskeletal structures to slide during joint motion. Due to their superficial location, they are exposed to work-related microtrauma. Olecranon and prepatellar bursae are therefore the site of acute or chronic inflammatory episodes. The annual incidence of bursitis is estimated at 1/10,000 inhabitants [1,2]. Septic bursitis (SB) is defined as inflammation of the bursae caused by contamination by a cultivable microorganism, most often bacterial. However a bursal puncture is not always performed, so the definition can be extended to bursitis with fever and/or cutaneous and subcutaneous inflammation, or bursitis initially not considered septic but with unfavorable evolution under symptomatic treatment.

Septic bursitis accounts for a third of all cases of bursitis and 0.01% to 0.1% of hospital admissions. However, it is mainly treated on an outpatient basis by general practitioners (GPs) [3]. A literature review showed that management of SB or presumed SB is heterogeneous and that guidelines are needed [3]. These recommendations for general practitioners, emergency physicians, rheumatologists, and orthopedic surgeons cover the diagnosis and therapeutic management of olecranon and prepatellar SB.

2. Method

The methodology was based on Haute Autorité de santé (HAS) recommendations [4]. The work group comprised 18 rheumatologists (15 hospital practitioners and 3 in private-practice), 1 infectious diseases specialist, 2 orthopedic surgeons, 1 GP, and 1 emergency doctor.

The group relied on a literature review carried out by C.D.L., G.C. and C.S. to summarize the data available on diagnosis and therapeutic management of SB. The objective of this literature review was to answer 10 practical questions previously validated by the group (Table S1). Three general principles and 11 specific recommendations were established, and the level of evidence was presented with each proposal.

According to the Delphi method, a review group including 50 physicians (20 rheumatologists (10 were hospital practitioners and 10 private practitioners), 6 infectious diseases specialists, 11 general practitioners, 8 emergency physicians, and 5 orthopedic surgeons) rated their level of agreement for each proposal on a Lickert scale from 0 to 10. Some group members previously reviewed recommendations for diagnosing and managing septic arthritis proposed in 2019 by the French Society for Rheumatology (SFR), the French Language Infectious Diseases Society (SPILF), and the French Orthopedic and Trauma Surgery Society (SOFCOT) [5].

A level of agreement of 0 meant total disagreement with the recommendation, while 10 meant total agreement, and 5 meant neither agree nor disagree. Reviewers were asked to specify why they disagreed with the recommendation when the level of agreement was ≤ 7. Consensus was predefined as follows: a mean level of agreement > 8 and/or $\geq 80\%$ of reviewers with a level of agreement ≥ 8 and/or $\geq 90\%$ of reviewers with a level of agreement > 6.

The response rate for the first round of Delphi was 86% (43/50). Later consensus was reached for all recommendations. Nevertheless, 3 recommendations were reworded (numbers 4, 7 and 10) according to the reviewers' comments and then submitted again for validation [response rate 74% (37/50)].

3. General principles and recommendations

Table 1 summarizes all recommendations, with their levels of evidence and consensus (see table S2 for the grading method).

3.1. General principles

3.1.1. Principle A – Bursitis is an extra-articular inflammation that should not be confused with arthritis. Most of the time, clinical examination can distinguish between them

Olecranon or prepatellar bursitis is a painful inflammation of the bursa and surrounding tissues. It causes swelling that should not be confused with arthritis [6–12]. Clinical examination is usually critical for distinguishing between the two diagnoses: no or moderate limitation in joint range of motion due to pain and no patella tap test. In addition, in bursitis, tissue swelling is located in the anterior or infra-patellar region of the knee, while joint effusion is located in the sub-quadricipital (supra-patellar) region in arthritis. At the elbow, swelling is located in the olecranon area, while joint swelling (effusion and/or synovitis) is usually located in the lateral sub-epicondylar region of the elbow in arthritis (Table 2).

3.1.2. Principle B – Septic etiology of olecranon or prepatellar bursitis should be considered in cases of bursitis associated with signs of infection and/or in the context of immunosuppression and/or in case of failure to treat bursitis initially considered nonseptic

Infection occurs mainly through direct contamination following skin lesions such as folliculitis, wounds, scratching lesions, animal scratches/bites, plant stings or injections. Pre-existing conditions can contribute to the occurrence of SB: chronic hygroma (micro-traumatic bursopathy), chronic rheumatic diseases (rheumatoid arthritis, gout), or post-traumatic acute bursopathy. But, it can also occur in the absence of risk factors. In immunocompromised patients, particularly in patients with diabetes, septic etiology should be considered in all cases of acute bursitis [3,6,13].

3.1.3. Principle C – The diagnosis of septic bursitis is clinical. Septic bursitis is characterized by inflammation of the bursa and surrounding subcutaneous tissues, sometimes with cellulitis, fever, or a skin lesion next to the bursa

In case of acute bursitis, no clinical signs are specific to the septic origin. Fever (17–77%), "cellulitis" (dermo-hypodermal inflammatory reaction with extensive erythematous edema) (28–93%) and elevated skin temperature (29–94%) are inconstant (Table S3) [7,13–25]. Table S3 summarizes the clinical features of SB in the 3 largest published retrospective series [14,15,26]. Fever and cellulitis around the bursa was reported in only 1/3 of patients [7,14,15]. A skin lesion was identified in 50 to 60% and an inguinal lymphadenopathy was found in 26% of patients [2].

Table 1
General principles and recommendations for diagnosing and managing septic bursitis.

	Level of evidence	Level of agreement of the review group			
		Delphi tour[a]	Mean ± SD	≥ 8/10 (%)	[6–10] (%)
General principles					
A – Bursitis is an extra-articular inflammation that should not be confused with arthritis. Most of the time, clinical examination can distinguish between them	2a-B	1	9.4 ± 1.0	93	100
B – Septic etiology of olecranon or prepatellar bursitis should be considered in cases of bursitis associated with signs of infection and/or in the context of immunosuppression and/or in case of failure to treat bursitis initially considered nonseptic	2a-B	1	9.3 ± 1.2	93	95.3
C – The diagnosis of septic bursitis is clinical. Septic bursitis is characterized by inflammation of the bursa and surrounding subcutaneous tissues, sometimes with cellulitis, fever, or a skin lesion next to the bursa	2b-B	1	9.2 ± 1.1	95.3	97.7
Specific recommendations					
1 – The diagnosis of septic bursitis is clinical and requires no further investigation in most cases. If necessary, ultrasound can be used to distinguish bursitis from arthritis	4-C	1	9.1 ± 1.5	90.7	97.7
2 – A bursal puncture is not mandatory. It may be performed to help manage bacteriological analysis, search for crystal-induced bursitis, or drainage of effusion	5-D	1	8.5 ± 1.7	81.4	90.7
3 – Medical management is ambulatory in the absence of severity criteria (extensive cellulitis, sepsis, presence of comorbidities, immunodepression)	5-D	1	9.5 ± 0.7	97.7	100
4 – Outpatient management is based on a 10-day course of oral antibiotics, effective against the bacteria responsible for septic bursitis (Staphylococcus aureus and β-hemolytic streptococci): amoxicillin-clavulanic acid or pristinamycin	4-C	1[a]	9.3 ± 1.0	97.2	97.2
5 – In case of outpatient medical treatment, a clinical reassessment is suggested 3 to 5 days after the start of antibiotic therapy	5-D	1	8.9 ± 1.5	95.3	95.3
6 – ospitalization should be considered in case of initial signs of severity (extensive cellulitis, sepsis, presence of comorbidities, immunocompromised condition) or unfavorable evolution within 3 to 5 days of outpatient management	4-C	1	9.2 ± 1.1	90.7	97.7
7 – In case of hospitalization, initial intravenous (IV) administration of antibiotics should be reserved for the most severe cases (sepsis and immunosuppression). Cefazolin or cloxacillin are suggested as first-line antibiotics. Oral relay should be based on clinical improvement. The total duration of antibiotics is 14 to 21 days, depending on clinical progression	4-C	1[a]	9.2 ± 1.0	97.2	97.2
8 – Iterative punctures of the infected bursa are not indicated	4-C	1	9.5 ± 0.7	100	100
9 – Initial surgical advice is indicated in case of local complications (abscess, necrosis, or fistulization) or regional complications (osteitis), or at a later stage, in case of unfavorable evolution despite well-managed hospital-based medical treatment	4-C	1	9.5 ± 0.9	95.3	97.7
10 – After surgery, the duration of antibiotic therapy is 7 to 10 days	4-C	1[a]	9.0 ± 1.5	91.7	94.4
11 – Other measures include verifying tetanus vaccination status in case of skin lesions, and prevention advice to avoid recurrence	5-D	1	9.6 ± 0.9	97.7	97.7

SD: standard deviation.
[a] Final formulation obtained in the second round of proofreading using the Delphi method.

Table 2
Clinical differences between septic bursitis and arthritis of the knee or elbow.

	Septic arthritis	Septic bursitis
Knee		
Swelling location	Supra-patellar	Infra-patellar
Patellar tap test	Present	Absent
Cutaneous inflammatory sign	Usually absent	Often present
Flessum	Often present	Absent
Skin injury	Rarely	Often
Elbow		
Swelling location	Lateral	Posterior
Cutaneous inflammatory sign	Generally absent	Often present
Flessum	Present	Absent
Skin injury	Rarely	Often

3.2. Specific recommendations

3.2.1. Recommendation 1 – The diagnosis of septic bursitis is clinical and requires no further investigation in most cases. If necessary, ultrasound can be used to distinguish bursitis from arthritis

Increased levels of serum inflammatory markers such as C-reactive protein (CRP) are frequent but also found in crystal-induced bursitis or rheumatoid bursitis. Blood cultures are rarely positive (0–19% of cases) [21,23,24].

X-rays of the affected joint are not necessary for diagnosis. However, in cases of unfavorable outcomes, they can identify cortical erosions (osteitis), reveal a differential diagnosis (hydroxyapatite calcification in the resorptive phase).

Magnetic resonance imaging (MRI) should be performed only in cases of suspected complications (myositis, muscle abscess or olecranon osteitis), which remain uncommon (< 2% of cases) [27,28].

For the diagnosis, ultrasound of the bursae can:

- confirm the diagnosis of olecranon or prepatellar bursitis and rule out joint effusion when there is diagnostic doubt;
- differentiate effusion and synovial thickening;
- help differentiate from other diagnoses (resorbing hydroxyapatite calcifications or inflammatory gouty tophus);
- look for active cortical erosion(s) on Doppler that might suggest a complication (osteitis/periostitis) [29–31].

Ultrasound can also be helpful to guide bursal puncture if needed.

3.2.2. Recommendation 2 – A bursal puncture is not mandatory. It may be performed to help manage bacteriological analysis, search for crystal-induced bursitis, or drainage of effusion

Fig. 1 summarizes the diagnostic approach for all-causes bursitis. If bursal puncture seems useful in the etiological diagnosis of nonseptic bursitis, its contribution in case of septic origin looks more limited.

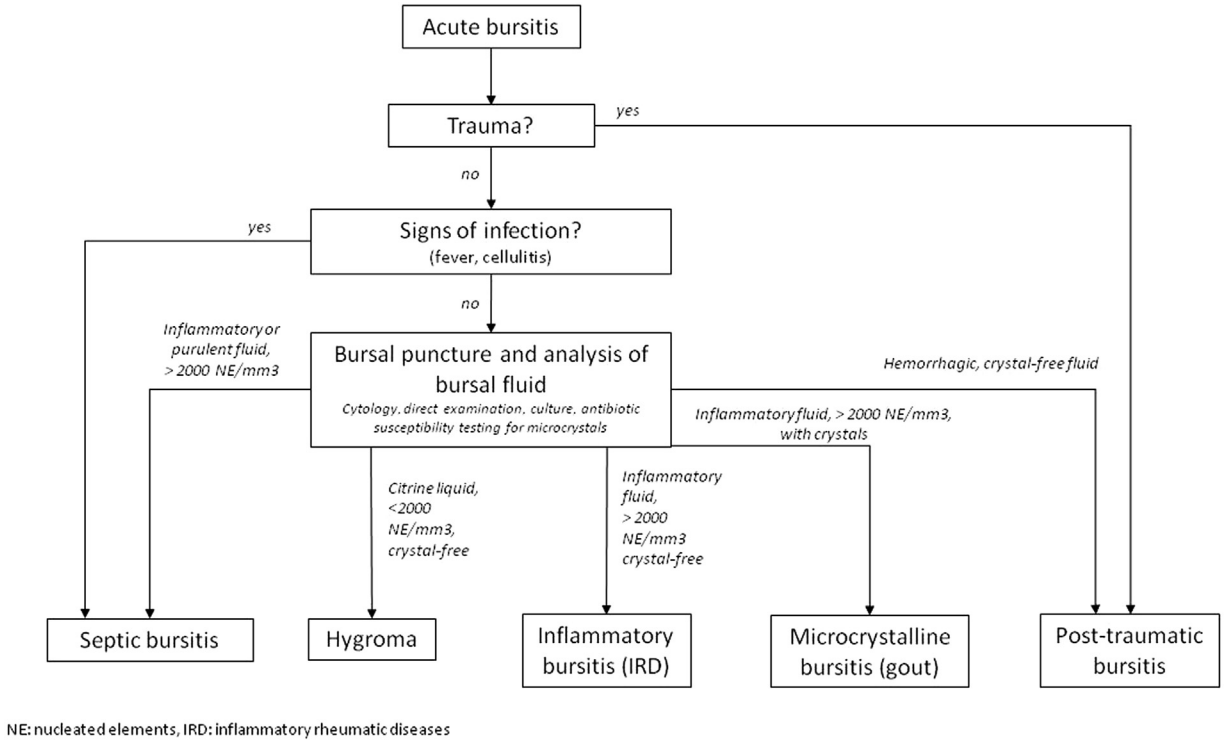

Fig. 1. Diagnostic approach to bursitis.

An inflammatory fluid (> 2000/mm^3 nucleated elements) has high sensitivity for the diagnosis of SB (94%) but is not specific enough, also being encountered during microcrystalline or rheumatic bursitis [19]. Moreover, a recent study reported poorer performance, with 31.5% of fluids with cellularity < 2000/mm^3 despite septic origin. Another retrospective study suggested a cut-off of 4700/mm^3: 66% sensitivity and 93% specificity for diagnosing septic bursitis [32].

Bursal fluid culture was positive in 75–80% of SB in 2 recent studies [14,15]. However, its contribution is questionable, since gram-positive cocci are involved in more than 95% of cases. *S. aureus* is the pathogen most frequently involved (62–88%), followed by β-hemolytic Streptococci (10–20%). Polymicrobial infections are possible, especially in case of skin wounds or animal bites.

Bacterial identification rarely led to a change in antibiotic molecule (< 1 in 10 cases) [33]. Furthermore not having bacterial identification was not associated with a higher rate of failure [14].

However, in certain situations, such as inflammatory tophus in the tricipital tendon or patellar ligament with contiguous reactive bursitis, the puncture can be useful for searching for crystals.

Thus, bursal puncture does not seem to be mandatory but the practitioner may want help with diagnostic orientation and/or management, at bursitis onset or during follow-up (sterilization of bursal fluid with antibiotic treatment is obtained after 4 days of treatment [16]). In published studies, bursal puncture was performed in 36–80% of patients [14,15,33,34].

3.2.3. *Recommendation 3 – Medical management is ambulatory in the absence of severity criteria (extensive cellulitis, sepsis, presence of comorbidities, immunodepression)*

While most cases of SB are treated on an outpatient basis, little data is available on factors influencing the choice between inpatient and outpatient management because most studies are hospital-based.

Only 2 retrospective studies provide information on these factors [13,15]. Factors associated with hospital care included extensive cutaneous inflammation (erysipelas, peribursal cellulitis), use of non-steroidal anti-inflammatory drugs, general signs (fever, biological inflammatory syndrome, hyperleukocytosis), age, comorbidities (diabetes, immunosuppressive therapy, active cancer, HIV infection, transplantation, cirrhosis), prepatellar involvement, and skin lesion [13,15]. Identifying the pathogen did not appear to influence the type of management [13]. Table 3 summarizes severity criteria worth considering when choosing the appropriate type of care [1–3,9–11,16,19,26,35,36]. In cases of severe presentation (extensive cellulitis, sepsis, presence of comorbidities, immunodepression) hospitalization should be considered.

3.2.4. *Recommendation 4 – Outpatient management is based on a 10-day course of oral antibiotics, effective against the bacteria responsible for septic bursitis (*S. aureus *and β-*hemolytic streptococci*): amoxicillin-clavulanic acid or pristinamycin*

As indicated above, mild SB will be treated in outpatient care, with oral probabilistic antibiotics as bursal puncture for microbial identification is not required.

No randomized studies comparing treatments are available. Data on outpatient management of SB come from retrospective studies with limited numbers of patients:

- the first study included 118 patients with olecranon SB [34]. Data were collected in a facility administering parenteral home antibiotics with all patients being treated intravenously. Thirty-eight percent of patients underwent bursal puncture. The others were treated empirically. The most commonly prescribed antibiotic was cefazolin (85%), followed by clindamycin (13%) and cloxacillin (2%). After 4 days of IV treatment (range: 1–14 days), clindamycin was prescribed in 55% and cephalexin in 25% of patients for a median duration of 7 days (range: 5–30 days). The median total duration of antibiotics was 12 days (range: 8–35 days);
- the second study included 30 patients with olecranon SB [33]. Nineteen patients were treated empirically. In this group of 19 patients, patients received 10 days of clindamycin or

Table 3
Severity criteria for septic bursitis [1–3,9–11,16,19,26,35,36].

Local clinical signs	General clinical signs	Comorbidities
Extensive peribursal cellulitis	Systemic infectious signs (fever, chills, blood hyperleukocytosis, elevated acute phase reactants) or Sign of sepsis (quickSOFA ≥ 2 of the following criteria: respiratory rate > 22/min, disturbed alertness and/or systolic blood pressure < 100 mmHg)	Diabetes Chronic alcoholism Hypogammaglobulinemia Neutropenia ≤ 500/mL Long-term corticosteroid therapy > 20 mg/day for at least 4 weeks or autoimmune disease requiring long-term corticosteroid therapy Active cancer HIV infection Organ transplantation Cirrhosis Child–Pugh B or C

trimethoprim/sulfamethoxazole or cefalexin. Sixteen patients recovered, and 3 required further antibiotic treatment. No surgical treatment was needed in this group treated empirically. In the puncture group ($n = 11$), culture results led to a change in antibiotic therapy in only 1 case (methicillin- and clindamycin-resistant *S. aureus*), 5 received a second course of antibiotics, and 8 underwent bursectomy. As the study was not randomized, it is possible that the cases were more severe in the puncture group, which may have explained the different outcomes;
- the third study included 22 patients [8], all of whom had undergone bursal puncture. The primary antibiotic prescribed was oral flucloxacillin ($n = 19/22$). During follow-up, 1 patient was hospitalized with no further details given, and another underwent surgery because of an abscess.

The first-line treatment must be effective against *S. aureus* and *β-hemolytic streptococci*, as these microorganisms are involved in almost all cases (respectively around 80–90% of cases [7,17,21,23,34] and 10–20% of cases [14,21,37]).

The combination of amoxicillin and clavulanic acid is an interesting option given its broad spectrum. And, the bactericidal power of pristinamycin on *S. aureus* argues in favor of this compound [38–40].

Data on the resistance profile of the microorganisms involved in SB are limited. However, in a recently published study from western France [14], most staphylococci were sensitive to methicillin (≤ 5% resistance), and the most frequently encountered resistance was to clindamycin (15%). According to the French REUSSIR hospital network (data from 2018), less than 7% of *Staphylococcus sp* were resistant to clindamycin [41]. However, the prevalence of resistant strains remains unknown among staphylococci involved in outpatient cases of SB. It could be lower than in a hospital. Finally, the bactericidal power of pristinamycin on *S. aureus* argues in favor of this compound [36–38].

The following options are proposed for first-line oral probabilistic antibiotics:

- amoxicillin–clavulanic acid: 50–100 mg/kg/24 h in 3 doses per day (no more than 6 g/day of amoxicillin and no more than 600 mg/day of clavulanic acid);
- pristinamycin: 3 g/24 h in 3 doses per day.

Clindamycin (600 mg × 3/day) can be an interesting alternative in case of allergy to penicillin. It is less expensive than pristinamycin.

Cefalexin seems to be a valid alternative in the case of methicillin-sensitive *S. aureus* (MSSA) infections, but it does not have authorization for use in soft tissue infections in France.

As nausea and diarrhea are common side effects of amoxicillin–clavulanic acid and pristinamycin, it is important to assure that the treatment is well tolerated.

For outpatient cases, a 10-day course of antibiotics is proposed. This short duration is justified by the type of infection (periarticular soft tissue) and the absence of severity [8,32,34]. As a reminder, the duration of antibiotic therapy for erysipelas is 7 days [42]. The treatment could be extended to 14 days in case of unfavorable evolution.

3.2.5. *Recommendation 5 – In case of outpatient medical treatment, a clinical reassessment is suggested 3 to 5 days after the start of antibiotic therapy*

There is no published data on the follow-up of outpatient cases of SB. Stell et al. recommended clinical reassessment 3 to 5 days after the start of a probabilistic oral therapy to assess efficacy and tolerance [8]. This time before reassessment was based on the duration of antibiotic treatment needed to sterilize the bursal fluid: 4 days on average [16].

3.2.6. *Recommendation 6 – Hospitalization should be considered in case of initial signs of severity (extensive cellulitis, sepsis, presence of comorbidities, immunocompromised condition) or unfavorable evolution within 3 to 5 days of outpatient management*

Seven single-center and 2 multicenter studies addressed the prognosis and severity criteria of SB [8,13–15,24–26,33,43,44]. However, their level of evidence is limited because of the retrospective design. Moreover, the results were influenced by local practices and involved only hospitalized patients.

The severity criteria for SB are the same as those observed in other infections. They are local and/or general, and/or related to the patients' comorbidities. Table S4 summarizes literature data on factors associated with the severity of SB (need for surgery or intravenous administration of antibiotics, duration of antibiotic therapy > 14 days, prolonged hospitalization, or relapse).

Immunosuppression (caused by diagnoses such as diabetes mellitus, alcoholism, hypogammaglobulinemia, neutropenia, corticosteroid treatment) may impact the course of SB and complicate its management [43]. Bursal fluid sterilization was obtained after courses of antibiotics 3 times longer than those in immunocompetent subjects [43].

Only one study found that *S. aureus* identification was associated with more surgical interventions, more intravenous IV administration, and a longer duration of antibiotic therapy [15].

Table S4 summarizes published data on factors associated with the severity of SB.

3.2.7. *Recommendation 7 – In case of hospitalization, initial intravenous (IV) administration of antibiotics should be reserved for the most severe cases (sepsis and immunosuppression). Cefazolin or cloxacillin are suggested as first-line antibiotics. Oral relay should be based on clinical improvement. The total duration of antibiotics is 14 to 21 days, depending on clinical progression*

No randomized studies comparing treatment options in terms of antibiotic molecules or duration of treatment are available. Treatment proposals are therefore based on data from retrospective studies and experts' opinions.

Table 4
Antibiotic therapy regimens for septic bursitis (SB) with or without severity criteria.

SB with severity criteria	SB without severity criteria or as a relay to the intravenous route
Initial intravenous probabilistic antibiotic therapy	Oral probabilistic antibiotics
Cefazolin: loading dose of 2 g over 1 h then 60–80 mg/kg/24 h (i.e. approximately 5 g/24 h if < 70 kgs, 6–7 g/24 h if > 70 kgs) distributed every 6 hours, i.e. 4 infusions of 1 to 2 h/24 h	Amoxicillin–clavulanic acid: 50–100 mg/kg/24 h in 3 doses (no more than 6 g/day of amoxicillin and no more than 600 mg/day of clavulanic acid) Pristinamycin: 3 g/24 h in 3 doses
Cloxacillin: loading dose of 2 g over 1 h then 100 mg/kg/24 h distributed every 6 hours, i.e. 4 infusions of 1 to 2 h/24 h	Alternatives: – Clindamycin: 600 mg taken 3 times a day – Cephalexin: 3–4.5 g/24 h in 3 doses per day (not with meals, do not take with dairy products)

In published studies, the proportion of IV-treated patients varied from 32% to 64% [14,15].

Fever, extensive cellulitis, skin wound, and microbial identification were associated with IV treatment but the presence of comorbidities was not associated [14,15].

The IV route but is suggested only in case of systemic signs or immunosuppression [16,26]. The first-line treatment should be either an anti-staphylococcal penicillin or a first-generation cephalosporin [1,2,8,14,24,25,34]. Cefazolin or cloxacillin should be administered instead of amoxicillin–clavulanic acid (Table 4). In case of SAMS bacteremia, there is a higher mortality rate with amoxicillin–clavulanic acid than with cloxacillin or cefazolin (OR = 2.68 [1.23–5.85]) [43]. Furthermore the amoxicillin–clavulanic acid combination was not retained by HAS 2019 for managing common bacterial skin infections such as cutaneous abscesses, furunculosis and severe impetigo. It was retained only in post-bite superinfection [42]. Subsequently, antibiotic therapy will have to be adapted to the antibiogram if available.

It was previously suggested that IV administration should last 7 to 10 days [2,21,24]. However, this duration has been shortened to 3 to 4 days without affecting clinical results [26,34]. This time frame makes it possible to verify the negativity of the blood cultures (if available). In the absence of bacteremia, IV treatment duration should be based on clinical improvement.

As SB is a soft tissue infection, antibiotics with good tissue diffusion can be started orally. In the absence of any signs of severity, oral antibiotics can be used for hospitalized patients (amoxicillin–clavulanic acid or pristinamycin) (Table 4).

In hospitalized cases of SB, the total duration of treatment varied from 10 to 21 days when treatment was exclusively medical [1–3,7,14,16,24,43,45,46]. Bursal fluid was sterilized after 4 days of treatment but increased to 9 days when symptoms had begun more than 7 days prior to hospital admission [16,47], and to 11 days when patients were immunocompromised [43]. In hospitalized cases of SB, antibiotic therapy of less than 14 days may be associated with more relapses (15%) [14].

We suggest antibiotics for 14 days for patients with moderate forms of SB and 21 days for patients with severe presentation and/or immunodepression. Patients should be reassessed at the end of treatment to verify recovery. Fig. 2 summarizes therapeutic management of SB according to initial severity and clinical progression.

3.2.8. *Recommendation 8 – Iterative punctures of the infected bursa are not indicated*

Iterative punctures were proposed every 24 to 72 hours, in case of purulent fluid, until the effusion had resolved [1,16,19,47,48]. It was suggested that healing could not be achieved without these aspirations [19]. However this has never been confirmed with any difference in evolution, whether it was done or not [34]. Nor has association between punctures and fistulization been established [8]. In practice, in cases of abundant effusion requiring iterative punctures, referral to surgery is suggested for more complete drainage and/or bursectomy.

3.2.9. *Recommendation 9 – Initial surgical advice is indicated in case of local complications (abscess, necrosis, or fistulization) or regional complications (osteitis), or at a later stage, in case of unfavorable evolution despite well-managed hospital-based medical treatment*

The studies published show heterogeneous practices for surgery, with almost systematic surgical interventions in some centers and few indications in others [2,26].

In general, surgery is indicated in the following situations [2,24,48–50]:

- unfavorable evolution after 3 to 5 days of antibiotic treatment as long as the molecules and dosage were appropriate;
- local complications such as abscess, necrosis or fistulization;
- presence of a foreign body;
- contiguous bone or joint infection;
- persistent symptomatic bursopathy (after infection resolution).

Several types of intervention have been described for the surgical management of SB: drainage and/or bursectomy (under endoscopy or open surgery) [51]. The latter can be performed in 1 or 2 stages (bursectomy followed by skin closure or flap coverage in a second step, in necrosis, for example). The bursectomy may be combined with excision of foreign bodies or resection of enthesophytes.

Some complications have been reported: skin complications (delayed healing, hematoma, chronic fistulization), nerve damage, scar pain, extension of the infection to surrounding tissues or recurrence [2,25,46]. The only randomized study [35] showed no difference in results between 1- and 2-stage bursectomy. However, the 1-stage procedure generated less skin dehiscence and was associated with shorter hospitalization and lower costs [35].

Endoscopic bursectomy, which can be performed on an outpatient basis, appears to be an attractive option. It allows external access to the inflammatory zone, reducing the risk of skin complications [52]. A review of 8 publications did not find any difference in relapse rate between endoscopic procedures and open bursectomy in prepatellar SB [46]. In general, identifying the anatomical limits of the bursa is easier to perform during surgery once the inflammation has "cooled down" with antibiotics.

3.2.10. *Recommendation 10 – After surgery, the duration of antibiotic therapy is 7 to 10 days*

After surgery, 7 days postoperative antibiotic therapy appeared to give good results in most cases in immunocompetent patients [26,35,46]. A literature review by Brown et al. reported that antibiotic therapy of less than 8 days was not significantly associated with more relapses than treatment of 8 days or more in prepatellar SB [48]. However, antibiotic therapy of less than 14 days was associated with more recurrences in 1 study (15% of patients) [14].

Antibiotic treatment for 7 to 10 days postop could therefore be suggested.

3.2.11. *Recommendation 11 – Other measures include verifying tetanus vaccination status in case of skin lesions, and prevention advice to avoid recurrence*

Non-drug measures can be combined with antibiotic therapy. The most frequently reported measures are analgesic rest, postop

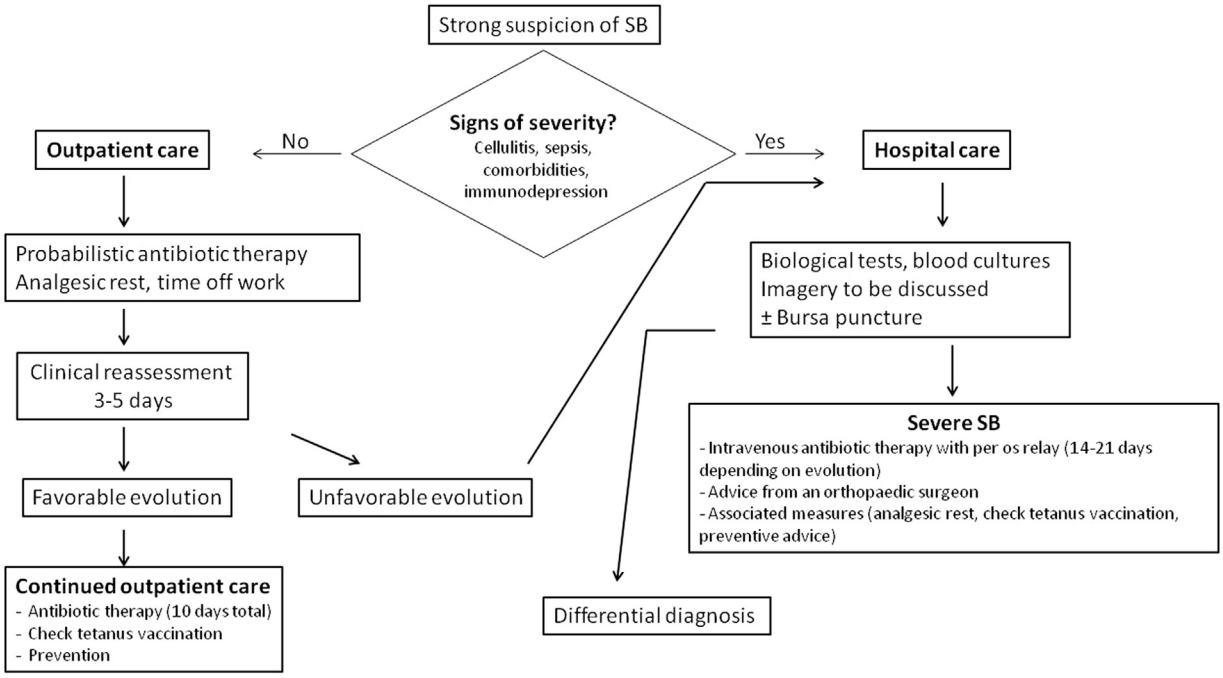

Fig. 2. Management algorithm for septic bursitis (SB).

immobilization with removable orthoses, and compression dressings [2,35,48]. In case of a skin lesion, anti-tetanus vaccination status should be checked. Alcohol-based dressings are not indicated in the management of SB.

NSAIDs are contraindicated in the treatment of septic bursitis as they can exacerbate soft tissue infections.

In terms of prevention, kneepads should be worn by workers who kneel for prolonged periods and by priests and nuns [53–55]. Skin and wound care should be encouraged in patients at risk of SB. Acute hygroma and chronic bursopathy can be considered occupational diseases (Table 57 in https://www.inrs.fr/publications/bdd/mp/tableau.html?refINRS=RG%2057).

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Appendix A. Supplementary data

Supplementary data (Tables S1–S4) associated with this article can be found, in the online version, at https://doi.org/10.1016/j.jbspin.2023.105664.

References

[1] McAfee JH, Smith DL. Olecranon and prepatellar bursitis. Diagnosis and treatment. West J Med 1988;149:607–10.
[2] Baumbach SF, Lobo CM, Badyine I, et al. Prepatellar and olecranon bursitis: literature review and development of a treatment algorithm. Arch Orthop Trauma Surg 2014;134:359–70.
[3] Lormeau C, Cormier G, Sigaux J, et al. Management of septic bursitis. Joint Bone Spine 2019;86:583–8.
[4] https://www.has-sante.fr/jcms/c_272505/recommandations-par-consensus-formalise-rcf.
[5] Couderc M, Bart G, Coiffier G, et al. 2020 French recommendations on the management of septic arthritis in an adult native joint. Joint Bone Spine 2020;87:538–47.
[6] Schermann H, Karakis I, Dolkart O, et al. Olecranon Bursitis in a military population: epidemiology and evidence for prolonged morbidity in combat recruits. Mil Med 2017;182:e1976–80.
[7] Mathieu S, Prati C, Bossert M, et al. Acute prepatellar and olecranon bursitis. Retrospective observational study in 46 patients. Joint Bone Spine 2011;78:423–4.
[8] Stell IM. Management of acute bursitis: outcome study of a structured approach. J R Soc Med 1999;92:516–21.
[9] Blackwell JR, Hay BA, Bolt AM, et al. Olecranon bursitis: a systematic overview. Shoulder Elbow 2014;6:182–90.
[10] Khodaee M. Common superficial bursitis. Am Fam Physician 2017;95:224–31.
[11] Aaron DL, Patel A, Kayiaros S, et al. Four common types of bursitis: diagnosis and management. J Am Acad Orthop Surg 2011;19:359–67.
[12] Darrieutort-Laffite C. Infectious bursitis. Rev Rhum Monogr 2022;89:51–6 [Available on: https://www.sciencedirect.com/science/article/pii/S1878622721000916].
[13] Cea-Pereiro JC, Garcia-Meijide J, Mera-Varela A, et al. A comparison between septic bursitis caused by Staphylococcus aureus and those caused by other organisms. Clin Rheumatol 2001;20:10–4.
[14] Charret L, Bart G, Hoppe E, et al. Clinical characteristics and management of olecranon and prepatellar septic bursitis in a multicentre study. J Antimicrob Chemother 2021;76:3029–32.
[15] Nguyen K, Coquerelle P, Houvenagel E, et al. Characteristics and management of olecranon or prepatellar septic bursitis. Infect Dis Now 2023;53:104652.
[16] Ho Jr G, Su EY. Antibiotic therapy of septic bursitis. Its implication in the treatment of septic arthritis. Arthritis Rheum 1981;24:905–11.
[17] Raddatz DA, Hoffman GS, Franck WA. Septic bursitis: presentation, treatment and prognosis. J Rheumatol 1987;14:1160–3 [PMID: 3437425].
[18] Smith DL, McAfee JH, Lucas LM, et al. Septic and nonseptic olecranon bursitis. Utility of the surface temperature probe in the early differentiation of septic and nonseptic cases. Arch Intern Med 1989;149:1581–5.
[19] Stell IM. Septic and non-septic olecranon bursitis in the accident and emergency department – an approach to management. J Accid Emerg Med 1996;13:351–3.
[20] Pien FD, Ching D, Kim E. Septic bursitis: experience in a community practice. Orthopedics 1991;14:981–4.
[21] Gómez-Rodríguez N, Méndez-García MJ, Ferreiro-Seoane JL, et al. Infectious bursitis: study of 40 cases in the pre-patellar and olecranon regions. Enferm Infecc Microbiol Clin 1997;15:237–42.
[22] Stell IM, Gransden WR. Simple tests for septic bursitis: comparative study. BMJ 1998;316:1877.
[23] García-Porrúa C, González-Gay MA, Ibañez D, et al. The clinical spectrum of severe septic bursitis in northwestern Spain: a 10 years study. J Rheumatol 1999;26:663–7.
[24] Martinez-Taboada VM, Cabeza R, Cacho PM, et al. Cloxacillin-based therapy in severe septic bursitis: retrospective study of 82 cases. Joint Bone Spine 2009;76:665–9.
[25] Lieber SB, Fowler ML, Zhu C, et al. Clinical characteristics and outcomes of septic bursitis. Infection 2017;45:781–6.
[26] Perez C, Huttner A, Assal M, et al. Infectious olecranon and patellar bursitis: short-course adjuvant antibiotic therapy is not a risk factor for recurrence in adult hospitalized patients. J Antimicrob Chemother 2010;65:1008–14.
[27] Chin TY, Peh WC. Imaging update on musculoskeletal infections. J Clin Orthop Trauma 2021;22:101600.
[28] Turecki MB, Taljanovic MS, Stubbs AY, et al. Imaging of musculoskeletal soft tissue infections. Skeletal Radiol 2010;39:957–71.
[29] Costantino TG, Roemer B, Leber EH. Septic arthritis and bursitis: emergency ultrasound can facilitate diagnosis. J Emerg Med 2007;32:295–7.
[30] Ruangchaijatuporn T, Gaetke-Udager K, Jacobson JA, et al. Ultrasound evaluation of bursae: anatomy and pathological appearances. Skeletal Radiol 2017;46:445–62.
[31] Naredo E, Uson J, Jiménez-Palop M, et al. Ultrasound-detected musculoskeletal urate crystal deposition: which joints and what findings should be assessed for diagnosing gout? Ann Rheum Dis 2014;73:1522–8.
[32] Faudemer M, Cabon M, Banal F. Olecranon and prepatellar bursitis. A retrospective monocentric study of 88 cases. Revue du rhumatisme 2023;90:596–602.
[33] Deal Jr JB, Vaslow AS, Bickley RJ, et al. Empirical treatment of uncomplicated septic olecranon bursitis without aspiration. J Hand Surg Am 2020;45:20–5.
[34] Laupland KB, Davies HD, Calgary Home Parenteral Therapy Program Study Group. Olecranon septic bursitis managed in an ambulatory setting. The Calgary Home Parenteral Therapy Program Study Group. Clin Invest Med 2001;24:171–8.
[35] Seymour CW, Liu VX, Iwashyna TJ, et al. Assessment of clinical criteria for sepsis: for the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA 2016;315:762–74.
[36] Buskila D, Tenenbaum J. Septic bursitis in human immunodeficiency virus infection. J Rheumatol 1989;16:1374–6.
[37] Uçkay I, von Dach E, Perez C, et al. One- vs. 2-stage bursectomy for septic olecranon and prepatellar bursitis: a prospective randomized trial. Mayo Clin Proc 2017;92:1061–9.
[38] Lamy B, Laurent F, Gallon O, et al. Antibacterial resistance, genes encoding toxins and genetic background among Staphylococcus aureus isolated from community-acquired skin and soft tissue infections in France: a national prospective survey. Eur J Clin Microbiol Infect Dis 2012;31:1279–84.
[39] van Bijnen EM, Paget J, de Lange-de Klerk ES, et al. Antibiotic exposure and other risk factors for antimicrobial resistance in nasal commensal Staphylococcus aureus: an ecological study in 8 European countries. PLoS One 2015;10:e0135094, http://dx.doi.org/10.1371/journal.pone.0135094.
[40] den Heijer CD, van Bijnen EM, Paget WJ, et al. Prevalence and resistance of commensal Staphylococcus aureus, including meticillin-resistant S. aureus, in nine European countries: a cross-sectional study. Lancet Infect Dis 2013;13:409–15, http://dx.doi.org/10.1016/S1473-3099(13)70036-7 [Epub 2013 Mar 6. Erratum in: Lancet Infect Dis. 2013 Dec;13(12):1011. Flemming, Douglas [corrected to Fleming, Douglas]].
[41] http://onerba.org/publications/rapports-onerba/.
[42] https://www.has-sante.fr/jcms/c_2911550/fr/prise-en-charge-des-infections-cutanees-bacteriennes-courantes.
[43] Roschmann RA, Bell CL. Septic bursitis in immunocompromised patients. Am J Med 1987;83:661–5.
[44] Germawi L, Westenberg RF, Wang F, et al. Factors associated with revision surgery for olecranon bursitis after bursectomy. J Shoulder Elbow Surg 2021;30:1135–41.
[45] Paul M, Zemer-Wassercug N, Talker O, et al. Are all beta-lactams similarly effective in the treatment of methicillin-sensitive Staphylococcus aureus bacteraemia? Clin Microbiol Infect 2011;17:1581–6.
[46] Brown OS, Smith TO, Parsons T, et al. Management of septic and aseptic prepatellar bursitis: a systematic review. Arch Orthop Trauma Surg 2021.
[47] Ho Jr G, Tice AD. Comparison of nonseptic and septic bursitis. Further observations on the treatment of septic bursitis. Arch Intern Med 1979;139:1269–73.
[48] Wilson-MacDonald J. Management and outcome of infective prepatellar bursitis. Postgrad Med J 1987;63:851–3.
[49] Zimmermann 3rd B, Mikolich DJ, Ho Jr G. Septic bursitis. Semin Arthritis Rheum 1995;24:391–410.
[50] Hoffmeyer P, Chalmers A, Price GE. Septic olecranon bursitis in a general hospital population. Can Med Assoc J 1980;122:874–6.
[51] Kerr DR, Carpenter CW. Arthroscopic resection of olecranon and prepatellar bursae. Arthroscopy 1990;6:86–8.
[52] Ogilvie-Harris DJ, Gilbart M. Endoscopic bursal resection: the olecranon bursa and prepatellar bursa. Arthroscopy 2000;16:249–53.
[53] Atkins JB, Marks J. The role of staphylococcal infection in beat disorders of miners. Br J Ind Med 1952;9:296–302.
[54] Sharrard WJ. Pressure effects on the knee in kneeling miners. Ann R Coll Surg Engl 1965;36:309–24.
[55] Thun M, Tanaka S, Smith AB, et al. Morbidity from repetitive knee trauma in carpet and floor layers. Br J Ind Med 1987;44:611–20.