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Olecranon bursitis

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Background: Bursitis is a common medical condition, and of all the bursae in the body, the olecranon bursa is one of the most frequently affected. Bursitis at this location can be acute or chronic in timing and septic or aseptic. Distinguishing between septic and aseptic bursitis can be difficult, and the current literature is not clear on the optimum length or route of antibiotic treatment for septic cases. The current literature was reviewed to clarify these points.

Methods: The reported data for olecranon bursitis were compiled from the current literature.

Results: The most common physical examination findings were tenderness (88% septic, 36% aseptic), erythema/cellulitis (83% septic, 27% aseptic), warmth (84% septic, 56% aseptic), report of trauma or evidence of a skin lesion (50% septic, 25% aseptic), and fever (38% septic, 0% aseptic). General laboratory data ranges were also summarized.

Conclusions: Distinguishing between septic and aseptic olecranon bursitis can be difficult because the physical and laboratory data overlap. Evidence for the optimum length and route of antibiotic treatment for septic cases also differs. In this review we have presented the current data of offending bacteria, frequency of key physical examination findings, ranges of reported laboratory data, and treatment practices so that clinicians might have a better guide for treatment.

Level of evidence: Narrative Review.

Keywords: Bursitis; olecranon bursitis; septic bursitis; aseptic bursitis; nonseptic bursitis; elbow bursitis

There are 3 types of inflammatory pathologies of the bursa (bursitides): acute, chronic, and septic. Acute bursitis usually results from direct trauma or prolonged pressure on the bursa.[26] If there are multiple acute episodes or if the patient is involved in occupational or recreational activities requiring prolonged pressure on the bursa, a chronic bursitis can develop.[26] Chronic bursitis is also frequently secondary to systemic disorders, for example, crystal deposition from gout or pseudogout and diseases such as rheumatoid arthritis.[49] Finally, septic bursitis is secondary to direct inoculation of the bursa through a skin wound or local spread from nearby cellulitis.[26] Hematogenous seeding of the bursa is extremely rare because the bursa has a poor blood supply.[4,15,30]

Anatomy

Bursae are closed fluid filled sacs with a synovial lining that facilitates gliding of musculoskeletal structures over one another during motion.[39,51] More than 150 bursae have been identified throughout the body.[30] These can be categorized into 3 groups: deep, superficial, and adventitious. Deep

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bursae are located between muscles or between muscle and bone[33,49] and develop in utero concurrently with synovial joints.[51] Superficial bursae appear to form in response to pressure and friction, over months or years after birth, and are found between bone and the overlying skin.[33,49,51] Adventitial bursae are acquired even later in life secondary to pressure over bony prominences or deformities.[51]

The olecranon bursa is a superficial bursa, and a cadaveric study showed that the olecranon bursa forms between the ages of 7 and 10 years.[9] The floor of the olecranon bursa lies on the triceps tendon and olecranon, and the roof is loosely connected to the overlying skin of the elbow (Fig. 1).

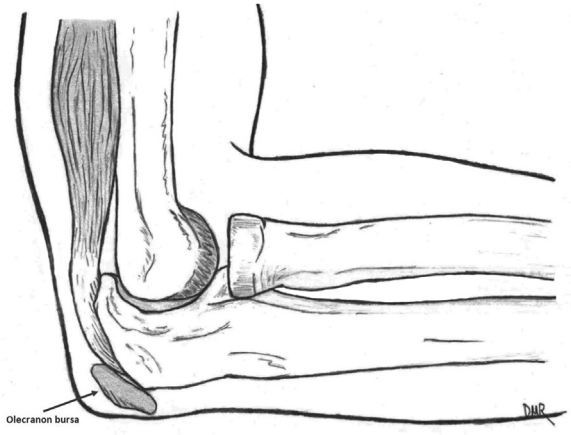

Figure 1 Illustration shows the location of the olecranon bursa and its proximity to the skin.

Epidemiology

The olecranon and prepatellar bursa are the most clinically relevant of the superficial bursae because they are predisposed to inflammation and infection given their locations.[30] The actual incidence of olecranon bursitis is unknown and difficult to quantify. It has been estimated to be between 0.01% and 0.1% of hospital admissions.[30] One reason olecranon bursitis is difficult to measure is that most studies are done in hospital-based systems, and many patients with milder cases are treated successfully in the community setting or as outpatients by primary care offices.[15,27] Another reason is that chronic idiopathic olecranon bursitis can be mild enough that patients do not seek medical attention, and these cases are often diagnosed when the patient has elbow surgery for other reasons.[16]

A prospective study by Smith et al[43] found that olecranon bursitis was diagnosed in 3 of every 1000 emergency or outpatient visits at their institution and that 1 of every 3 to 4 cases was septic. Ho and Tice[19] studied men presenting to a Veterans Administration medical center and found olecranon bursitis was more common than prepatellar bursitis, with an approximate ratio of 4:1 in this population.

Septic bursitis is traditionally thought of as a condition of young to middle-aged men involved in manual labor, related specifically to direct traumatic inoculation.[20] All published studies have had a male predominance, with a reported female occurrence of 0% to 13%.[7,20,27,38] However, a study from rural Spain reported an increased female occurrence of 17.3%, which was most likely due to the increased number of women involved in manual labor in the rural setting.[15] One study also found a seasonal distribution of septic olecranon bursitis that peaked in the summer months, perhaps secondary to an increase in outdoor activities or labor, or both.[27] This illustrates that involvement in manual labor or other predisposing activities is likely more important than gender as a risk factor for the development of septic olecranon bursitis.

Etiology

Trauma

There are two main causes of olecranon bursitis, the most common being traumatic.[8,15] Trauma to the olecranon can lead to septic or noninfective bursitis. Patients with septic or nonseptic bursitis report antecedent trauma to the affected elbow between 33% and 77% of the time.[35,45,51] However, septic bursitis is almost always preceded by some kind of trauma. Minor trauma and sometimes repetitive microtrauma are enough to allow bacterial invasion of the bursa.[45] Septic olecranon bursitis is therefore an occupational hazard of those involved in manual labor such as plumbers, miners, gardeners, mechanics, and athletes.[30,37] Larson and Osternig[26] noted an increase in the number of cases of olecranon bursitis after the installation of artificial turf in football arenas, which may be accounted for by the relative abrasive nature of the artificial surface compared with grass.

Associated medical conditions

The second major reason patients develop olecranon bursitis is secondary to a pre-existing systemic medical condition. Development of bursitis is directly due to the comorbidity or secondary to immunosuppression caused by the treatment of it.[4] As with traumatic injuries, these conditions can lead to septic, noninfective, or chronic bursitis. The reported rate of at least 1 comorbidity varies in the literature from 33% to 74%.[27,38] A small study by Canoso and Sheckman[7] evaluated 16 patients with bursitis, 12 of which involved the olecranon, and all but 1 patient had a comorbid condition.

Common comorbid conditions that have a direct association with olecranon bursitis are diabetes, alcoholism, immunosuppression from chronic steroid therapy, psoriasis, HIV infection, crystalline diseases, such as gout and pseudogout, and rheumatoid arthritis (Fig. 2).[4,5,7,20,36,45,51]

Alcoholism causes some degree of systemic immunosuppression and a higher incidence of trauma to the elbows, leading to the increased risk for septic olecranon bursitis.[20] The small lacerations over extensor surfaces associated with psoriasis are thought to act as entry points for bacteria, thus leading to an increased risk of septic bursitis.[45] In addition, some patients with psoriasis receive immunosuppressive therapy that could contribute to their increased risk. Conditions such as chronic obstructive pulmonary disease and the requirement for chronic hemodialysis, in addition to steroid-based immunosuppression, often require patients to place pressure on the elbows for prolonged periods of time for support and mobilization, which increases their risk of developing olecranon bursitis.[21,51]

Crystalline diseases, such as gout, cause many cases of bursitis in general, but the olecranon bursa is one of the most affected bursae.[11,17,32] The olecranon bursa is very commonly involved in tophaceous gout because of the tendency of monosodium urate crystals to deposit in superficial structures with low temperatures. The nodular appearance of the skin and the gross appearance of the excised bursa with tophi are shown in Fig. 3.[12] Figs. 4-7 show the histologic appearance of various crystal deposits in the olecranon bursa. Long slender needles can be identified at high magnification, and the surrounding inflammatory reaction caused by this crystal deposition is evident. Other kinds of crystals have also been identified in the olecranon bursa, notably calcium pyrophosphate dihydrate, apatite, and cholesterol crystals.[25,28,31]

Microbiology

By far, the most likely causative organism of septic olecranon bursitis is *Staphylococcus aureus*.[2,18,20,24,27,35,38,45,51] Most of the isolated strains are also resistant to penicillin.[18,20,45] The second most isolated organism are streptococcal species with β-hemolytic *Streptococcus* the most common.[18,24,35,51] The reported incidence of major causative organism can be found in Table I. Polymicrobial infections have been reported in approximately 10% of infections.[51] More rare organisms have also been reported in immunocompromised patients.[42] Local epidemiology and microbe profiles can also influence the infection source and should be taken into consideration when cultures are being ordered.

Diagnosis

Clinical presentation and physical examination findings

As evident by the above discussion, considerable overlap exists between the causes of septic and noninfective olecranon bursitis. This probably contributes to the inherent difficulty of distinguishing between the 2 pathologies from the history and physical examination alone because many of the findings overlap.[4,19–21,30] Bursal edema, erythema, and tenderness are almost universally present.[4] Fig. 8 is representative of the bursal edema seen at presentation.

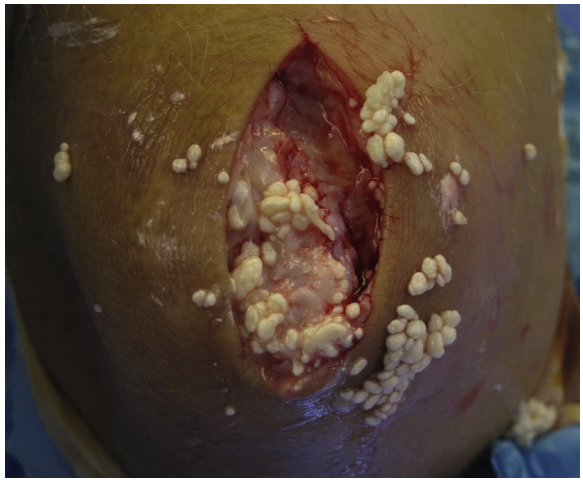

Figure 2 Intraoperative photograph of a patient with rheumatoid arthritis and chronic olecranon bursitis. Abundant rice bodies were found when the bursa was excised.

An important part of the physical examination is careful determination of skin/contour changes, region of pain, and elbow range of motion. Full range of motion is generally preserved in septic and noninfective bursitis, but pain is noted in flexion and extension with septic arthritis.[4,6,20,30,39] Pain may be an occasional feature of noninfective bursitis, particularly during end arc flexion, presumably due to increased peribursal pressures. A study of intrabursal pressures and pain found that pain did not occur even at the highest intrabursal pressures and that the pain elicited in full flexion was probably from receptors in the surrounding osseotendinous structures.[6] The presence of pyrexia and noticeable lacerations/abrasions seems to be variable but is not sensitive or specific for identifying septic cases. Fever has been reported in 20% to 77% of septic cases, depending on which study is referenced.[15,27,45] Table II compares the overlap of the clinical and laboratory findings between septic and nonseptic olecranon bursitis.

Given the difficulty of distinguishing septic from nonseptic olecranon bursitis, a thorough physical examination and history should be conducted. This should focus on the patient's associated medical conditions, current or recent medications, including steroid injections, family history, personal history of recurrent bursitis or trauma to the affected elbow, and occupation and hobbies.[4] Specific questioning relating to direct posterior elbow trauma (overt or repetitive) should be considered.

In addition, there are some special considerations to bear in mind. Firstly, septic bursitis and septic arthritis can occur simultaneously, and any clinical concern for septic arthritis

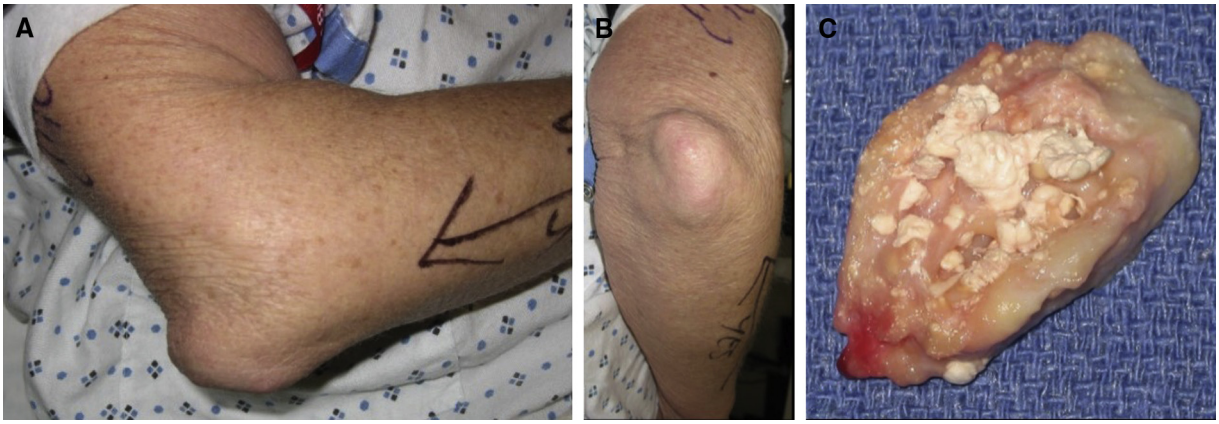

Figure 3 (**A**) Lateral preoperative photograph of patient with tophaceous gout. The patient's hand is to the right of the photo. The subcutaneous nodules characteristic of tophaceous gout are visible. (**B**). Anteroposterior view of the same patient. The hand is toward the bottom of the photograph. (**C**). Intraoperative photograph shows the bursa after excision with clearly visible tophi.

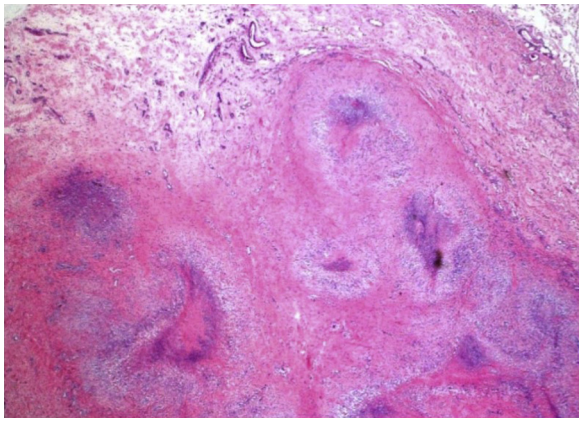

Figure 4 Hematoxylin and eosin–stained section at low magnification shows multiple palisaded necrobiotic nodules/granulomas and fibrosis. Original magnification 40x.

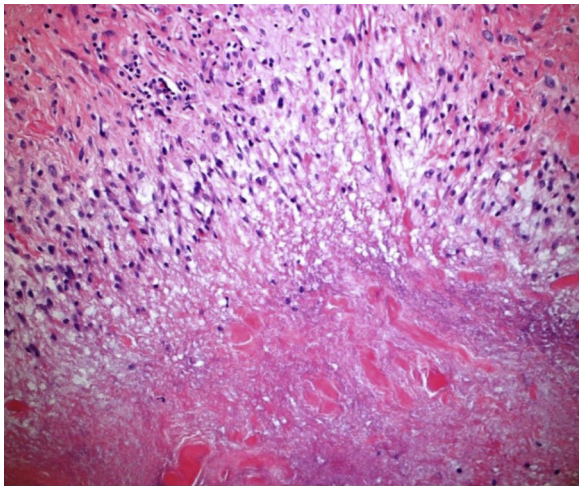

Figure 5 Hematoxylin and eosin–stained section at high magnification shows necrobiotic nodules with necrotic collagen, scant lymphohistiocytic inflammation, and rare plasma cells. Original magnification 100x.

warrants an arthrocentesis.[51] However, septic bursitis can also cause a sympathetic sterile joint effusion.[19,30] Secondly, nonseptic olecranon bursitis can be concurrent with synovitis caused by rheumatoid arthritis or gout.[19] Thirdly, it is possible for the olecranon bursa to rupture, which can result in fluid extravasation throughout the soft tissues of the forearm and lead to significant dorsal edema.[29] Finally, findings suggestive of neoplasia, such as rapid expansion, overt invasive skin changes, and weight loss, should always be observed with suspicion, because recurrent sarcomas have been initially misdiagnosed as olecranon bursitis, leading to loss of valuable time.[4]

Laboratory investigations

The gold standard for diagnosing septic olecranon bursitis is a positive culture of bursal fluid, but cultures take several days and do not help in deciding the initial treatment.[49] Unfortunately, no single laboratory test has been identified that is highly sensitive and specific for distinguishing between septic and sterile bursitis. However, combining several laboratory tests with a thorough history and physical examination is usually sufficient to make the distinction. There are very few circumstances where an inflamed bursa should not aspirated under sterile conditions, and this should ideally be done before antibiotic therapy is initiated.[4] In fact, some authors suggest that if antibiotics have been administered before the aspiration, the aspiration should not be performed because the likelihood of isolating the offending organism is very low.[2] The resulting bursal fluid should be sent for microbiologic culture, Gram staining, leukocyte count with differential, crystal analysis, and glucose.[3,4,30,39,42] Peripheral blood should be sent for complete blood count with differential, C-reactive protein, erythrocyte sedimentation rate, and glucose.[3,4,30,39,42]

Gram stains are reported as being positive in 50% to 100% of culture-proven septic bursitis and therefore cannot

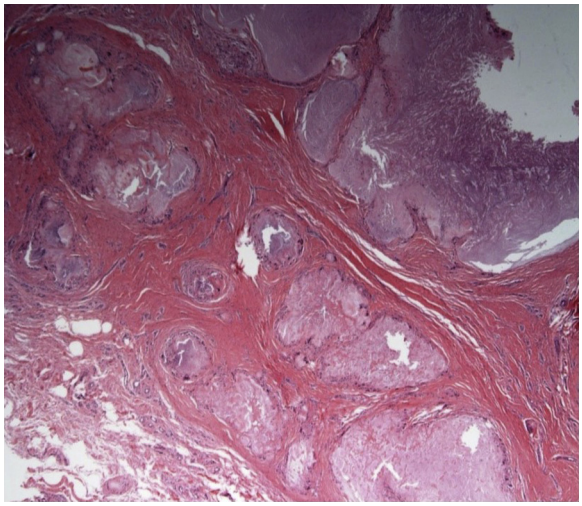

Figure 6 Hematoxylin and eosin–stained section shows multiple nodular deposition of crystalline material with surrounding inflammation and foreign body–type giant cells in a patient with gout who underwent bursectomy. Original magnification 40x.

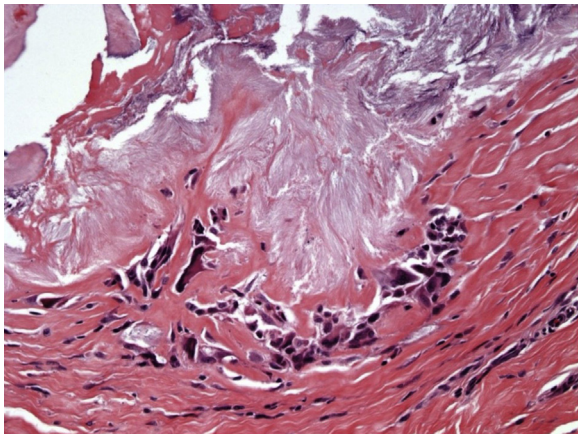

Figure 7 A hematoxylin and eosin–stained section at higher magnification shows long, slender, and needle-shaped crystals with associated inflammation and foreign body–type giant cells. Original magnification 100x.

be relied on to rule out sepsis.[4,19,30,33,51] Aspirate white blood cell counts are also nondiagnostic on their own and have been reported between 690 cells/mm^3 and 418,000 cells/mm^3 in septic cases and between 50 cells/mm^3 and 10,000 cells/mm^3 in sterile cases.[15,19,20,50] The aspirate differential can provide more helpful information when distinguishing septic from noninfective. A polymorph predominance of >50% has been associated with infection, and a mononuclear predominance >50% is seen with sterile cases.[19,42]

Ho and Tice[19] found that a bursal fluid glucose concentration <50% of the serum glucose was diagnostic of infection. However, further studies have found this measure is unreliable, and Garcia-Porrua et al[15] found this comparison had a false-negative rate of 9%.[51] The reported laboratory data for septic and nonseptic olecranon bursitis are summarized in Table II. In addition, it is important that bursal aspirate be sent for both crystal analysis and culture because bacterial infection may occur concomitant with crystal disease.[51]

Table I Organisms cultured from bursitis aspirates

Organism	Reference
Gram-positive cocci	
Staphylococcus aureus	Most common. Found in every published study
Staphylococcus epidermidis	8,16,19,25,37,39,44,48
Group A *Streptococcus*	9,16,19,25,28,39,41,44,46,50
Group B *Streptococcus*	8,9
Group C *Streptococcus*	44,46
Group G *Streptococcus*	28,46,48
Streptococcus agalactiae	16
Streptococcus pyogenes	36,46
Enterococcus faecalis	36
Gram-positive bacilli	
Bacillus subtilis	39
Gram-negative bacilli	
Klebsiella oxytoca	36
Escherichia coli	16
Enterobacter cloacae	39
Gram-negative coccobacilli	
Pseudomonas fluorescens	39
Anaerobes	
Brucella abortus	16
Propionibacterium acnes	37
Mycobacterium	
Mycobacterium tuberculosis	16
Mycobacterium marinum	37

Smith et al[44] have examined the utility of surface temperature measurements as a way to distinguish septic from aseptic. They found that the classification of patients at presentation based on standard laboratory results and physical examination was impossible in 36% who were eventually culture positive and in 37% who were culture negative.[44] When surface temperature readings were taken into consideration, they found that a difference in surface temperature of 2.2°C or more compared with the contralateral limb was able to distinguish septic from noninfective with a sensitivity of 100% and specificity of 94%.[44] These data have not yet been validated by prospective trials.

Obtaining peripheral blood cultures is a clinical decision. Bacteremia has been reported to occur at rates of 4% to 30%.[15,35,40] The highest rates have been reported in immunocompromised individuals, and these are the patients in whom obtaining blood cultures will have clinical relevance.

Imaging

In most patients with suspected olecranon bursitis, anteroposterior and lateral radiographs are the only imaging necessary. They serve to rule out fracture and retained

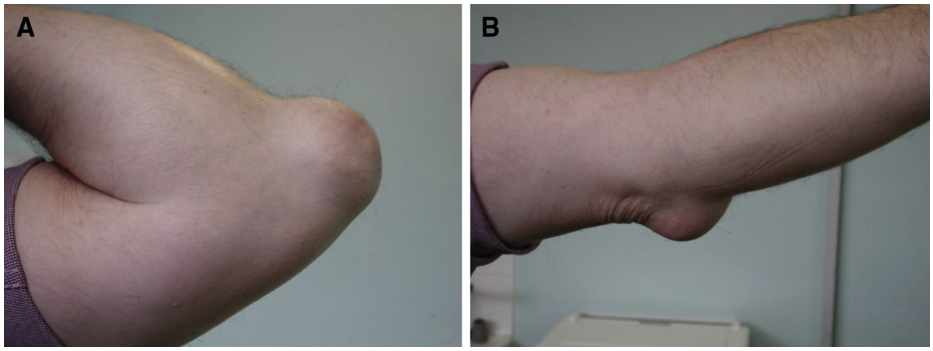

Figure 8 Preoperative photographs show a patient with large chronic olecranon bursitis causing functional and cosmetic issues. Full range of motion is preserved and demonstrated in these photos.

Table II Average reported physical examination and laboratory findings of septic and nonseptic bursitis

Findings	Septic	Nonseptic	Reference
Physical exam (%)			
Tenderness Avg (Range)	88 (59-100)	36 (20-61)	18,19,26,37,42,46
Erythema/Cellulitis Avg (Range)	83 (41-100)	27 (23-33)	5,15,18,19,26,35,37,39,46
Warmth Avg (Range)	84 (36-100)	56 (51-61)	26,37,42,46
Trauma/skin lesion* Avg (Range)	50 (0-82)	25 (0-53)	5,7,18-20,23,34,35,39,42-44,47
Fever Avg (Range)	38 (0-86)	0	7,15,18-20,26,35,37,39,44
Labs			
Bursal WBC count (mm^3) Avg (Range)	62,923 (900-392,5000)	2,215 (50-11,700)	5,6,8,18,19,35,37,39,42,43,46,48,50
Peripheral WBC count (mm^3) Avg (Range)	11,197 (3,700-22,600)	(10,200-21,000)	15,19,26,44
% PMN Avg (Range)	82 (45-100)	20 (0-94)	8,18,35,37,42,43,48,50
% Mononuclear cells Avg (Range)	-	78 (10-100)	5,18
Positive Gram stain (%) Avg (Range)	56 (21-78)	0	7,35,37,39,42

PMN, polymorphonuclear; *WBC*, white blood cell.
* Represents any patient-reported prior trauma or physical examination evidence of trauma.

foreign body in cases of trauma, as well as evaluating for soft tissue abnormalities and bony pathology, such as olecranon spurs, which are often associated with olecranon bursitis.[4,29,41] Radiographically, a swollen bursa appears as concentric circles on the anteroposterior view.[41] Representative radiographs are shown in Fig. 9. Other imaging modalities, such as contrast studies, are occasionally necessary when bursal rupture is suspected.[29] More recently, the ability of magnetic resonance imaging to differentiate septic and nonseptic bursitis was studied. Soft tissue enhancement was present in up to 76% of cases of bursitis regardless of the cause; however, absence of enhancement was indicative of nonseptic bursitis.[14]

Treatment

Noninfective

Conservative

As mentioned, most cases of olecranon bursitis are purely inflammatory. The mainstays of treatment for these patients are nonsteroidal anti-inflammatory medications, rest or activity modification, compression, ice, well-padded splinting, possibly physical therapy, and needle aspiration, which is therapeutic and diagnostic.[3,10,16,26,30,39]

Other treatments have been investigated because these noninfective cases are the most likely to recur or become

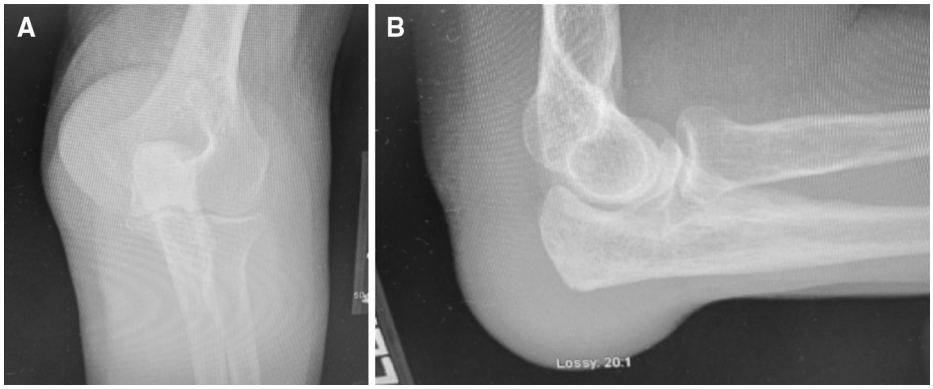

Figure 9 (**A**) Anteroposterior and (**B**) lateral radiographs of a patient with bursitis.

Table III Proposed classification system of clinical severity and recommended intervention

Classification	Ho and Su classification[19]	Treatment
Severe	Extensive local infection with intense peribursal cellulitis or infected skin wound. Systemic symptoms and signs of fever >37.7°C, chills, or peripheral WBC count >10,000/mm^3.	Hospitalized for intravenous antibiotics
Moderate	Moderately severe local inflammation, with or without minor skin wound and systemic signs.	Oral antibiotics and outpatient follow-up
Mild	Mild to moderate local inflammation, usually without skin lesion or systemic symptoms or signs.	Oral antibiotics and outpatient follow-up

WBC, white blood cell.

chronic. Fisher[13] found that a single aspiration was inadequate and that there were fewer recurrences after insertion of a 16-gauge angiocatheter surrounded by a bulky dressing. This allowed the bursa to decompress for at least 3 days and the walls to approximate, leading to fewer recurrences.[13] However, there are no reports of complications, such as conversion to septic bursitis, of this method, and it has not been studied or verified in any prospective trials.

Another treatment that has been studied more extensively is corticosteroid injection into proven culture-negative olecranon bursitis. Corticosteroid injection has been shown to decrease the duration of symptoms, and some authors advocate its use.[1,10,44,50] Others, however, have reported significant complications after steroid injection. Soderquist and Hedstrom[45] retrospectively reviewed 52 cases of suspected septic bursitis (both prepatellar and olecranon) and found that 11% of the septic cases were preceded by corticosteroid injection of the bursa.

Weinstein et al[50] evaluated 47 patients with traumatic olecranon bursitis. Twenty-five patients were treated with aspiration, followed by injection of 20 mg of triamcinolone hexacetonide after cultures were negative, and the remaining patients were treated with aspiration alone. Patients who received steroid injections resolved sooner, but septic bursitis developed in 3 and skin atrophy developed in 5; these complications were not seen in the aspiration group.[50] In addition, chronic pain while leaning on the affected elbow developed in 7 patients who received steroids, whereas only 2 patients in the aspiration group had this complication.[50]

Finally, Smith et al[44] reported a controlled blinded prospective trial in which 42 patients were randomized into 4 different treatment groups. They found that injection of corticosteroids significantly decreased symptom duration and that the addition of naproxen had no effect on disease course.[44] Interestingly, they reported no secondary septic bursitis or skin atrophy, which they credit to using a thin needle and a lateral olecranon approach, thereby avoiding infiltration or backflow of the steroid into the thin subcutaneous tissue of the posterior olecranon.[44] Collectively, these data suggests that nonsteroidal anti-inflammatory drugs are more effective for symptomatic relief and that steroid injection can be effective but must be weighed against the potential complications.

Operative

If conservative management fails, operative treatment may be necessary. Olecranon bursectomy is notorious for being associated with wound healing problems and should be reserved for only truly chronic cases of olecranon bursitis that interfere with function.[10,26,39] Open bursectomy has been the traditional surgical method, but many wound healing problems have been reported postoperatively.[3,10] These complications are likely due to the watershed midline blood supply, and the attachment of the bursa to the thin overlying skin inevitably leads to blood vessel and

nerve damage of the dermis.[4,37] Degreef and de Smet[10] retrospectively reviewed 37 cases of open olecranon bursectomy and found that 27% of the patients had wound healing problems, which were defined as hematoma, prolonged exudation, and skin necrosis. Surgical resection was not necessarily curative, because 22% of their patients experienced a recurrence requiring aspiration or further resection, and 1 patient needed a lateral arm flap.[10]

Arthroscopic bursectomy has been proposed as a way to move incisions away from the midline and decrease soft tissue trauma, thereby mitigating the common wound healing problems seen after open olecranon bursectomy.[4,22,34] Several studies have shown that the success rate of arthroscopic bursectomy is equal to that of open bursectomy while having less wound healing problems and a faster recovery.[3,22,23,34] Arthroscopic bursectomy also allows for olecranon spur removal, and removal of this bony pathology recommended as well when it is found associated with bursitis.[3,48] Some authors have even advocated for the removal of the spur only and leaving the bursa intact.[37]

Special consideration should be given to patients with rheumatoid arthritis before undertaking olecranon bursectomy. Stewart et al[48] found that open bursectomy resulted in long term symptomatic relief in 94% of their patients without rheumatoid arthritis but only in 40% of their patients with rheumatoid arthritis. The success of arthroscopic treatment of patients with rheumatoid arthritis has not been reported to date; however, Kerr et al[23] reported their only treatment failures of arthroscopic excision occurred in patients with CREST (calcinosis, Raynaud phenomenon, esophageal hypomobility, sclerodactyly, telangiectasia) syndrome or gout.

Septic

Conservative

The cornerstones of treatment of olecranon septic bursitis have traditionally been drainage and initiation of effective antibiotics. Some authors question the need for drainage. Laupland et al[27] retrospectively reviewed 118 cases of septic olecranon bursitis in an ambulatory patient population. Aspiration was only performed in 38% of patients, and only 51% underwent at least 1 drainage procedure (incision and drainage or aspiration).[27] They found no difference in outcome between patients who did and did not receive drainage procedures.[27] These results do not apply to most patient populations, however, because all of these patients initially received parenteral antibiotic therapy that was monitored by home parenteral therapy clinics, and access to these types of programs is limited in most communities. Given the difficulty in clinically distinguishing septic and nonseptic olecranon bursitis and the low percentage of culture-proven cases in this study, one also has to question if sterile cases were being treated.

Other authors, however, have agreed that needle aspiration may not be necessary for successful treatment of mild septic olecranon bursitis.[18,20,27,36] Most published studies, though, still advocate for the use of needle decompression.[7,26,30,47,51] Percutaneous suction and continuous irrigation has also been advocated for episodes of severe sepsis that fail to respond adequately to needle decompression and intravenous antibiotics.[24,30]

Because most of the septic bursitis is caused by penicillinase-producing *Staphylococcus aureus*, empiric antibiotic therapy should be penicillinase resistant or a first-generation cephalosporin. If patient allergies prohibit these agents, intravenous vancomycin can be used.[20,27,51]

The length of antibiotic treatment and route of administration are debated. The current literature has no consensus for treatment, and the published results can vary widely. Several studies have proven that oral and parenteral antibiotics both accumulate in the bursa at a high enough concentration to be effective, so intrabursal instillation of antibiotics is no longer recommended.[18,19]

Several studies have sought to identify the optimum length of treatment by monitoring the duration of antibiotic treatment needed to achieve sterility of bursal fluid. Using this method, Ho and Su[18] developed a classification system for bursitis (olecranon, prepatellar, and infrapatellar) severity meant to guide route of antibiotic administration as well as treatment as an outpatient vs admission. A summary of this classification system can be found in Table III. It is important to note that this study excluded patients with diabetes mellitus, renal or hepatic disease, rheumatic disease, or malignancy.[18] Blackwell et al[4] proposed that the presence of a comorbidity likely to affect healing or immune response should increase the classification by one level.

Ho and Su[18] were able to draw some important conclusions from their results. First, prompt administration of definitive antibiotic therapy decreased the time to bursal fluid sterilization. Other authors have corroborated this finding.[15,19,30] Second, continuing antibiotics for 5 days after bursal fluid sterility was confirmed led to curative treatment.[18] This treatment strategy led to an average length of antibiotic treatment of 9.7 days.[18] Several other studies recommend a total 10-day to 14-day antibiotic regimen consisting of intravenous, followed by oral treatment, or oral treatment alone.[27,30] Use of oral antibiotic therapy only should be considered cautiously because the treatment failure rate has been reported between 39% and 50%.[20,36,38]

Whether patients should be hospitalized is a clinical decision based on overall presentation of the patient, associated medical conditions, access to a parenteral antibiotic clinic if necessary, and the patient's ability be monitored closely in a clinic. Admission is generally reserved for those patients with extensive disease requiring multiple days of parenteral antibiotics, multiple drainage procedures or acute surgical intervention, and significant comorbidities.[20,40,46] Significantly immunocompromised patients, such as those with uncontrolled diabetes mellitus, HIV, and current steroid use, deserve special mention. These patients have clinical presentations and causative organism profiles that are similar to their

immunocompetent counterparts; however, they have been found to require 3 times as long to achieve bursal fluid sterility, and their bursal fluid aspirates had higher white blood cell counts.[15,40] Oral antibiotic treatment has also been found to fail more frequently in immunocompromised patients; thus, these patients should be initially treated with parenteral antibiotics.[20,40]

If not treated adequately, septic bursitis can lead to chronic bursal infection, osteomyelitis, and cutaneous fistula formation.[15,24]

Operative

Surgical intervention for septic olecranon bursitis does not seem to provide long-term advantages in outcomes. The most common procedure is incision and drainage.[47,51] Indications for surgical intervention include inadequate needle aspiration due to thick pus or loculations, presence of a pointing abscess or foreign body, refractory disease, or a need to investigate the extent of the infection.[51] Refractory disease is generally considered infection that does not respond to adequate drainage and appropriate antibiotics after 1 week.[51] Some cases are so severe at presentation that they require an acute bursectomy. Perez et al[35] advocated a single-stage procedure with immediate closure because it reduced the in-hospital stay by 4 days and produced satisfactory outcomes.

Prevention

Prevention of olecranon bursitis is the best treatment because once an episode of bursitis has occurred, recurrence can become more frequent and requires less trauma to trigger.[26,35] If the triggering activity cannot be avoided, a padded orthosis with a hard exterior can be worn over the affected elbow.[35] It is important that this protective pad does not interfere with range of motion significantly because this can lead to contractures. If the bursitis is secondary to occupation, it may be helpful to find ways to simplify the required tasks as well as using good posture and body mechanics to minimize the stress placed on the elbow.

Perez et al[35] found that immunosuppression was a risk factor for recurrence that was independent of length of antibiotic treatment and surgical intervention. This is a difficult risk factor to mitigate, but these results stress the importance of adequate treatment and control of systemic medical conditions as a way to prevent recurrence of olecranon bursitis.

Conclusion

Olecranon bursitis is a very common disease, and yet, distinguishing between septic and nonseptic bursitis based on physical examination alone remains difficult. Laboratory data can aid in diagnosis; however, there are no clear-cut thresholds for many laboratory parameters such as white blood cell or bursal leukocyte count. In addition, many different opinions exist on what is the minimum and optimum length of antibiotic treatment for septic cases and the best route of antibiotic delivery. Given this lack of clear guidelines, we have reviewed the current literature and have presented the data of offending bacterial species, frequency of key physical examination findings, ranges of reported laboratory data, and treatment practices so that clinicians might have a better guide for treatment.

Disclaimer

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