Septic Olecranon Bursitis: Recognition And Treatment

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Background: **The superficial location of the olecranon bursa places it at high risk for injury, possibly leading to the entry of bacteria into the bursal sac. Early differentiation between septic and nonseptic olecranon bursitis is paramount to direct therapy, to hasten recovery, and to prevent chronic inflammation.**

Methods: **A literature review was performed using MEDLINE files from 1967 to the present. Additional references from the bibliographies of these were also utilized.**

Results and Conclusions: **Olecranon bursitis is a common condition that requires the treating physician to be aware of the predisposing factors, clinical signs and symptoms, and laboratory findings of both septic and nonseptic olecranon bursitis. With early recognition, prompt therapy, and preventive measures, the morbidity of septic olecranon bursitis can be considerably reduced, but surgical incision and drainage or excision could be required if conservative therapy fails. (J Am Board Fam Pract 1995; 8:217-20.)**

Bursae are closed, saclike structures that produce small amounts of fluid allowing for smooth and almost frictionless motion between contiguous muscles, tendons, bones, ligaments, and skin.[1,2] The superficial location of the olecranon bursa puts it at high risk for injury, possibly leading to the entry of bacteria into the bursal sac.[3]

Olecranon bursitis is common in athletes who play basketball, football, soccer, and hockey as a result of the athlete falling and striking an elbow on hard playing surfaces,[2] which can lead to inflammation (bursitis) within the olecranon bursal sac. Occasionally an inflamed bursa can also become infected, requiring differentiation between septic and nonseptic bursitis.

Most cases of nonseptic olecranon bursitis are due to overuse injury and are seen in athletes who play sports that involve repetitive overhead throwing and elbow flexion and extension, such as baseball, swimming, skiing, gymnastics, and weight lifting.[4] Direct trauma to the elbow is also a common cause of olecranon bursitis as a result of repeated scrapes and falls on artificial turf, wrestling mats, hardwood floors, ice, and exercise mats.[2] Both overuse injury and trauma to the bursa lead to inflammation, effusion, and thickening of the bursal wall.[5]

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Case Report

A National Collegiate Athletic Association Division I college basketball player struck his left elbow on the hardwood court while falling during a game. Because of pain and swelling of his left elbow, the next morning he sought treatment at a local emergency department. There was localized swelling of his left olecranon bursa with induration and erythema restricted to the bursal area. No abrasions or skin lesions were visible. His elbow had full pronation, supination, and flexion, with terminal extension loss secondary to pain. Radiographs of the left elbow showed soft tissue swelling of the bursa but no bony injury. The bursa was aspirated under sterile conditions in the emergency department and 3 mL of cloudy fluid were drained and sent for Gram stain and culture. No organisms were seen on Gram stain. He was discharged from the emergency department with a compression dressing around the left elbow. Twenty-four hours later, the culture grew *Staphylococcus aureus* and 750 mg of oral ciprofloxacin was prescribed to be taken every 12 hours.

After 4 days of oral ciprofloxacin, compression wraps, ice, elevation, and rest, he continued to experience pain, swelling, spreading erythema, decreased range of motion, and warmth of the left elbow area. He was admitted to the hospital for intravenous antibiotics.

At the time of admission he was afebrile, but the erythema had extended proximally to the left upper arm and distally to the extensor surface of

the forearm. There was extreme tenderness to palpation at the olecranon bursa and warmth over the entire affected area, but no epitrochlear or axillary lymphadenopathy. Complete blood count with a differential leukocyte count was normal with a white cell count of 8800/μL. We concluded that the septic olecranon bursitis had worsened to include cellulitis of the left arm and forearm.

We prescribed intravenous nafcillin, 2 g every 6 hours, and during a 3-day hospitalization, his erythema, tenderness, warmth, and swelling diminished. At the time of discharge the erythema, warmth, and induration had almost completely resolved; there was mild tenderness over the bursa area, and full active range of motion was observed. Near terminal extension of the elbow yielded mild pain. He remained afebrile throughout the hospitalization. At the time of discharge oral ciprofloxacin, 750 mg twice a day, was prescribed based on laboratory sensitivities. He was instructed in mild range-of-motion exercises of the left elbow and advised not to play basketball until pain was completely gone and to avoid further trauma to the elbow.

Upon attempting to shoot baskets on the 2nd and 3rd day after discharge, he developed worsening pain and was readmitted to the hospital with a markedly tender, swollen, and fluctuant left olecranon bursa. There were palpable enlarged epitrochlear but no axillary lymph nodes. In view of the failure of conservative therapy of compression wraps, ice, rest, closed-needle aspiration, and both oral and intravenous antibiotic therapy, the decision was made for surgical excision of the left olecranon bursa. At surgery the bursa was thickened and edematous, but there was no evidence of infection in the surrounding tissues. Microscopic examination of the 3.5 × 3.0 × 0.8-cm bursa revealed florid granulation tissue formation and fibrin deposits consistent with a microscopic diagnosis of acute and chronic bursitis. Stains for acid-fast organisms, culture and stains for fungi, and a tissue Gram stain were negative. The patient was discharged on the second postoperative day with no signs of active infection, minimal pain, and a compressive dressing in place. We prescribed ciprofloxacin, 750 mg twice a day, and on postoperative day 7 he was allowed to begin light activity; 2 weeks postoperatively he returned to competition.

Discussion

The development of a septic bursitis depends on several predisposing factors. The most important is a history of trauma to the bursa, with transcutaneous bacterial contamination.[1] With its superficial location and high injury rate, the olecranon bursa is the most common site for septic bursitis to occur.[1] Local or distal skin disruption is commonly seen near the area of elbow trauma. Even in elbow trauma with no visible skin abrasions, the avenue for bacterial infection is still most likely through the skin.[1] Other predisposing factors for the development of a septic bursitis include frequent pressure on the bursa, an immunocompromised state (human immunodeficiency virus infection, oral corticosteroid therapy), rheumatoid arthritis, systemic lupus erythematosus, and gout.[6-10]

Organisms Associated with Septic Bursitis

The organism most frequently identified in septic olecranon bursitis is *Staphylococcus aureus*, which is present in 72 to 92 percent of the infections,[1,6-8,11,12] reflecting that bacterial seeding of most cases of septic olecranon bursitis occurs transcutaneously. In cases of systemic illnesses or an immunocompromised state, however, case reports of rare types of organisms have been documented.

Cryptococcus neoformans was identified as the causative organism for olecranon bursitis in a patient with cirrhosis.[13] A patient with diabetes mellitus type II was reported to have an olecranon bursitis caused by *Aspergillus terreus*. Both of these cases emphasize the importance of organism identification in immunocompromised patients with a septic olecranon bursitis.[10]

Differentiation between Septic and Nonseptic Olecranon Bursitis

The most common clinical findings with septic olecranon bursitis are bursal swelling, pain, erythema, tenderness, and tissue warmth.[1,6,14] In a study by Ho and colleagues,[1] bursal swelling and pain were present in 100 percent and 92 percent of septic olecranon bursitis cases, respectively. A surrounding cellulitis often accompanies the septic olecranon bursitis[1,7,8] as much as 89 percent of the time.[7] The presence of fever varies, ranging from 15 percent in a study by Pien, et al.[8] to 86 percent in a Scandinavian hospital series.[15]

Joint motion, including rotation, is usually painless except for full flexion and extension.[1]

In comparison with nonseptic bursitis, patients with septic bursitis seek medical attention sooner, are more likely to be febrile, have a bursa that is more often tender to touch, develop a cellulitis of the surrounding skin, and more frequently have overlying skin lesions. These findings, however, are not diagnostic of an infection.[11] The diagnosis is best made by aspiration of the bursal sac under sterile conditions with a Gram stain and culture of the aspirated fluid obtained.[7]

Because the signs and symptoms of nonseptic bursitis are similar to infectious bursitis, aspiration and culture of the bursal fluid are essential in all cases of bursitis.[1,6,17,11] Despite reported rates of only 21.4 percent[8] and 65 percent[1] in identifying the responsible organism based on Gram staining of aspirated fluid, this test is still recommended to aid in appropriate antimicrobial selection.

Measurement of total leukocyte count and glucose levels of aspirated bursal fluid helps differentiate septic from nonseptic bursitis. Bursal leukocyte counts are usually greater than 10,000/μL in septic olecranon bursitis, with the percentage of polymorphonucleocytes ranging from 52 to 98 percent.[7] Patients with nonseptic olecranon bursitis usually have bursal leukocyte counts less than 1000/μL, with a predominance of mononuclear cells (67 percent).[11] Bursal fluid glucose levels are low in many of the cases of septic bursitis; in nonseptic cases the level is usually normal.[11]

An elevated peripheral white cell count can be helpful in further diagnosing a septic olecranon bursitis. The absence of a peripheral leucocytosis does not rule it out, however. In a study by Forouzesh and colleagues,[6] the peripheral white cell count ranged from 7,900 to 17,700/μL in athletes with a septic olecranon bursitis.

Elbow radiographs are frequently required to rule out an olecranon or elbow fracture in athletes who report symptoms consistent with septic or nonseptic olecranon bursitis. Elbow radiographs done on athletes with a septic olecranon bursitis reveal a characteristic soft tissue density consisting of a distended bursa without evidence of joint effusion.[1]

Treatment of Septic Olecranon Bursitis

As *Staphylococcus aureus* is the most common organism associated with septic olecranon bursitis, the initial antibiotic of choice is a penicillinase-resistant semisynthetic penicillin.[6] A 2-week course is necessary for successful resolution of the bacterial infection.[6,11] Immunocompromised patients must undergo treatment longer to guarantee a successful outcome.[3] Outpatient treatment with oral antibiotics is effective in patients with mild to moderate infections, e.g., who do not have fever, extensive cellulitis, or systemic leukocytosis.[8] More severe infections will require hospitalization and intravenous antibiotics.[1,8]

Closed-needle aspiration of the excess fluid from the bursal sac is frequently required in addition to antibiotic therapy to treat septic bursitis successfully. Closed-needle aspiration is the preferred initial drainage procedure for most patients with septic bursitis.[7] Incision and drainage of the bursal sac are recommended in cases that do not respond to at least one aspiration procedure.[8] If closed-needle aspiration, antibiotics, or incision and drainage fail, excision of the bursal sac could be required.[16]

Treatment of Nonseptic Olecranon Bursitis

Rest, ice, and reduced activity are the hallmarks of conservative treatment for nonseptic olecranon bursitis.[4] The bursa should be aspirated and the fluid sent for culture to rule out an infectious process.[16] If the patient is compliant with the above regimen, acute bursitis should resolve quickly.[4] Patients who have a 2-month or longer history of bursitis and who are considered to have a more advanced chronic condition, however, often do not improve with conservative treatment. Repeat aspiration, along with corticosteroids injected into the bursa, can hasten resolution of inflammation.[16] Nonsteroidal anti-inflammatory drugs will often provide analgesic and anti-inflammatory relief, and a compression wrap maintained around the olecranon bursa for several weeks will prevent recurrence of bursal fluid accumulation.[16] Protecting the affected elbow with padding to prevent repeated trauma and avoiding excessive movement of the extremity are paramount to hasten resolution.

Occasionally closed-needle aspiration of the olecranon bursae leads to bacterial seeding of the surrounding elbow tissue secondary to fistula formation. The occurrence of both infection and fistula formation through the thin skin of the elbow can be decreased by proper surgical

preparation of the area and lidocaine infiltration through the skin and subcutaneous tissue into the olecranon bursa.[17]

Conclusion

Olecranon bursitis is a common condition that requires the treating physician to be aware of the predisposing factors, clinical signs and symptoms, and laboratory findings of both septic and nonseptic olecranon bursitis. Septic must be distinguished from nonseptic olecranon bursitis early to hasten resolution and prevent chronic inflammation and cellulitis. With early recognition, prompt therapy, and preventive measures, the morbidity of septic olecranon bursitis can be considerably reduced, but surgical incision and drainage or excision could be required if outpatient therapy fails.

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