Review Article

Four Common Types of Bursitis: Diagnosis and Management

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Abstract

Bursitis is a common cause of musculoskeletal pain and often prompts orthopaedic consultation. Bursitis must be distinguished from arthritis, fracture, tendinitis, and nerve pathology. Common types of bursitis include prepatellar, olecranon, trochanteric, and retrocalcaneal. Most patients respond to nonsurgical management, including ice, activity modification, and nonsteroidal anti-inflammatory drugs. In cases of septic bursitis, oral antibiotics may be administered. Local corticosteroid injection may be used in the management of prepatellar and olecranon bursitis; however, steroid injection into the retrocalcaneal bursa may adversely affect the biomechanical properties of the Achilles tendon. Surgical intervention may be required for recalcitrant bursitis, such as refractory trochanteric bursitis.

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Pain of the knee, elbow, hip, and heel is among the most common musculoskeletal complaints. Septic and aseptic bursitis are common causes of pain, and they must be differentiated from arthritis, tendinitis, fracture, tendon or ligament injury, infection, and neoplasm. Bursitis arises from infectious and noninfectious etiologies, and distinguishing between the two can be challenging. A thorough history and physical examination is required for diagnosis. Adjunct tests are helpful in determining the diagnosis. Most patients with bursitis can be successfully treated nonsurgically. For patients who do not respond to nonsurgical treatment, surgical options include open bursectomy, arthroscopic bursal excision, and partial excision of involved bony processes.

Prepatellar Bursitis

Bursitis arises from many inflammatory phenomena, but infection is the primary concern. Approximately 80% of cases of septic prepatellar bursitis are caused by *Staphylococcus aureus*.[1] Other organisms, including other *Staphylococcus* species, and *Streptococcus*, *Mycobacterium*, *Brucella*, and fungal species, have been implicated in the pathogenesis of prepatellar bursitis.[1,2] The mechanism of infection is believed to be direct inoculation, not hematogenous seeding, likely because of the poor blood supply to the bursa. Noninfectious etiologies of bursitis include trauma; gout; sarcoid; idiopathic calcification; and calcinosis, Raynaud phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia (ie, CREST) syndrome.

Anatomy

The subcutaneous prepatellar bursa and the superficial infrapatellar bursa are the two main bursae about the knee joint. They are typically referred to collectively as the prepatellar bursa, but they are usually ana-

Figure 1

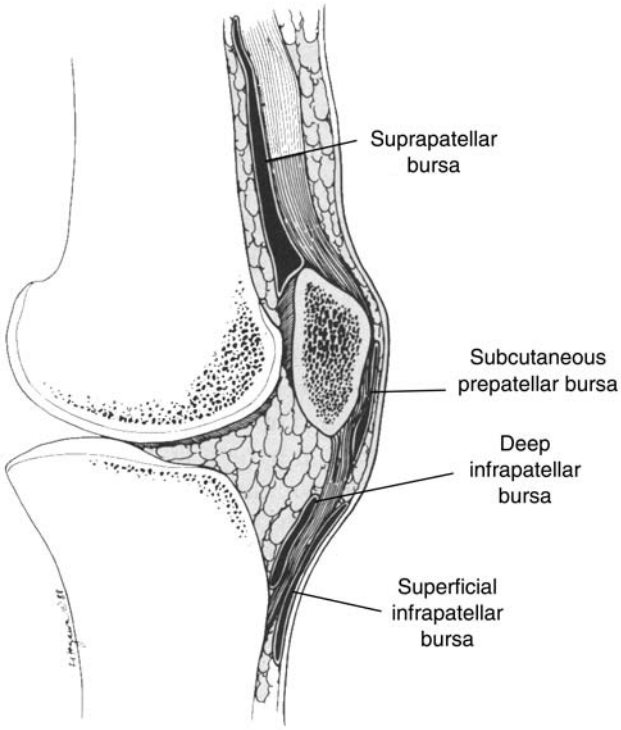


Illustration demonstrating the anatomy of the prepatellar bursa, which consists of the subcutaneous prepatellar bursa and the superficial infrapatellar bursa. (Adapted with permission from McAfee JH, Smith DL: Olecranon and prepatellar bursitis: Diagnosis and treatment. *West J Med* 1988;149:607-610.)

tomically distinct. The subcutaneous prepatellar bursa lies between the skin and the patella, and the superficial infrapatellar bursa lies between the skin and the tibial tubercle. These structures typically do not communicate with the knee joint (Figure 1).[3]

Presentation and Physical Examination

The clinical indications of septic prepatellar bursitis are swelling, pain, erythema, and warmth. Local tenderness to palpation is a hallmark of this condition. Pain with joint range of motion is atypical except for discomfort at extreme flexion, which compresses the inflamed bursa.[3] Predisposing factors include a history of trauma to the area, such as repetitive microtrauma (eg, prolonged kneeling), as well as immunocompromised status, alcoholism, chronic obstructive pulmonary disease, chronic renal failure, history of local corticosteroid therapy, and previous bursal inflammation. There is a strong correlation between these risk factors and *S aureus* bursitis.[1] Septic bursitis caused by *S aureus* is most common in the summer months.

Diagnosis

Diagnosis of septic prepatellar bursitis is based on clinical presentation and risk factors. Distinguishing infectious from noninfectious etiologies can be challenging. Aspiration of the bursa is often necessary. McAfee and Smith[3] recommend a lateral approach for aspiration of the prepatellar bursa because entering the bursa anteriorly increases the risk of iatrogenic sinus tract formation. Diagnostic thresholds have been proposed for septic prepatellar bursitis, including a bursal aspirate nucleated cell count that is far lower than the cell count for septic arthritis (>1,000 per µL and 50,000 per µL, respectively).[1] Gram stain and culture of the aspirate should be obtained. Gram stain may be negative in some cases, and regular cultures may be negative for mycobacterial, fungal, and bacterial (ie, *Brucella*) infections.[2-5] Inoculation of the bursal aspirate into liquid media is a more sensitive method of culture than plating on solid media.[6]

Management

Management of septic prepatellar bursitis is controversial. Recommendations range from oral antibiotics alone to surgical excision of the bursal sac. The primary decision in developing a treatment algorithm is whether to initiate nonsurgical or surgical management. Most patients respond to nonsurgical treatment. Surgery is a definitive option that is associated with complications. Management of aseptic prepatellar bursitis typically consists of rest, compression, and nonsteroidal anti-inflammatory drugs (NSAIDs). It may include local corticosteroid injection.[7]

Stell[8] performed aspiration followed by prescription of a 10-day course of oral antibiotics in seven patients with septic prepatellar bursitis. Mean time to recovery was 3 weeks (range, 1 to 4 weeks). Two of seven patients required admission for intravenous antibiotics.

Knight et al[9] managed two cases of septic prepatellar bursitis with intravenous antibiotics and placement of a percutaneous tube to facilitate suction-drainage and irrigation. Symptoms resolved with 12 days of

irrigation and an average of 19 days of antibiotic coverage. No recurrences or sinus tract formation were reported.

In a retrospective analysis, Cea-Pereiro et al[1] compared septic bursitis caused by *S aureus* with septic bursitis caused by other organisms. Surgery was required in only 4 of 47 patients with bursitis caused by *S aureus* and in 2 of 11 patients with bursitis caused by other organisms.

Bursectomy is one surgical option for patients whose symptoms do not resolve with nonsurgical management. Wang et al[5] performed bursectomy in a patient with septic prepatellar bursitis caused by *Sporothrix schenckii*. In their review of the literature, the authors found that five of seven patients with bursal sporotrichosis required bursectomy after failure of antimicrobial therapy. Complications of bursectomy include wound healing problems, atrophic skin changes, accumulation of subcutaneous hematoma, and severe tenderness.

Quayle and Robinson[10] described a modified technique in which only the posterior wall is resected, leaving the anterior wall adherent to the subcutaneous tissue. They speculated that this technique would protect the skin and reduce the risk of complications. No major complications were associated with the procedure.

Open surgical management of aseptic prepatellar bursitis is subject to the same risk of complications as that of the infectious form. More recently, an endoscopic approach to bursectomy has been reported.[11] Wound complications are considerably lower compared with open approaches. Ogilvie-Harris and Gilbart[11] reported no significant complications following endoscopic resection in 19 cases of aseptic prepatellar bursitis. Two thirds of patients were asymptomatic postoperatively.

Although compressive wrapping, elevation, and NSAIDs are accepted first-line management for aseptic prepatellar bursitis, few data exist on their success rate. Intrabursal corticosteroid injections also are commonly used to manage aseptic prepatellar bursitis. Although symptomatic relief may be faster with injection than with more conservative approaches, injection has associated risks, including infection, skin atrophy, and chronic pain. Historically, prepatellar bursitis has been managed with intrabursal injection of autologous blood (ie, blood patch) or a caustic chemical, such as sodium morrhuate, and placement of a short-term indwelling drainage catheter. These techniques have not proved to be successful, however.[12]

Olecranon Bursitis

Olecranon bursitis is the most common superficial bursitis.[13] Fluid collection within and inflammation around the bursa are caused by traumatic, inflammatory, and infectious processes. Olecranon bursitis is typically noninfectious in origin; septic bursitis accounts for approximately 20% of all acute cases.[14] Although olecranon bursitis is readily recognized on physical examination, the etiology may be difficult to determine.

Anatomy

The olecranon bursa forms after age 7 years.[15] Pressure from the bony olecranon and shearing forces applied to the overlying skin during activity may contribute to bursa formation.[15] This superficial bursa covers the dorsal olecranon and extends from the most distal triceps insertion to several centimeters along the proximal subcutaneous border of the ulna. The acutely distended bursa may be 6 to 7 cm long and 2.5 cm wide.[16]

Presentation and Physical Examination

Olecranon bursitis typically presents with unilateral swelling over the proximal olecranon. History of minor or repetitive local trauma is common. Aseptic traumatic bursitis is characterized by a nontender fluctuant mass over the olecranon. However, depending on the degree of associated inflammation, 20% to 45% of these patients report tenderness.[17,18] Septic olecranon bursitis is often associated with greater tenderness than the aseptic form, and septic olecranon bursitis may have a visible cellulitic component. Aseptic and septic olecranon bursitis may be indistinguishable on initial examination, however.

Sterile bursitis is associated with varying degrees of hyperemia of the skin overlying the bursa as well as edema extending into the forearm.[19] In persons undergoing hemodialysis, the arm used for vascular access has a noted predilection for olecranon bursitis.[20] Severe olecranon bursitis may result in a sympathetic effusion of the underlying elbow joint.[18] Effusion typically resolves with management of the bursitis.

Diagnosis

A fluid-filled olecranon bursa is generally recognized. However, olecranon bursitis occurs in conjunction with several systemic conditions, such as rheumatoid arthritis, gout, pseudogout, chondrocalcinosis, and pigmented villonodular synovitis.[21] These underlying processes must be recognized and managed to provide adequate treatment and prevent recurrence.

In the patient with an acutely inflamed olecranon bursa, the clinician must distinguish between septic and aseptic bursitis. In some cases, physical examination alone is insufficient to establish a diagnosis.[17] In addition

Figure 2

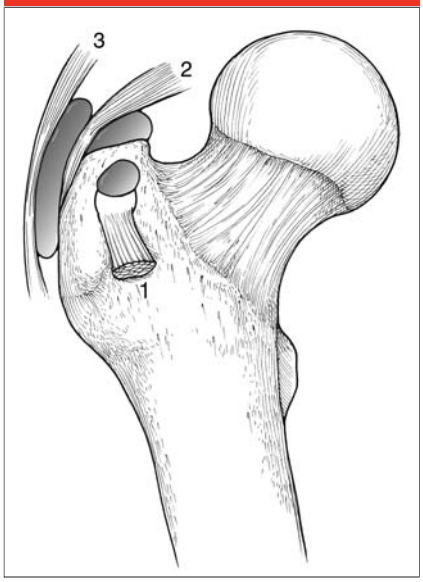


Illustration demonstrating the location of the trochanteric bursa between the gluteus medius (2) and the iliotibial band (3) as well as the bursa located between tendon and bone at the gluteus minimus, which is reflected downward (1). (Redrawn with permission from Lequesne M: From "periarthritis" to hip "rotator cuff" tears: Trochanteric tendinobursitis. *Joint Bone Spine* 2006;73[4]:344-348. http://www.sciencedirect.com/science/journal/1297319X.)

to physical examination, bursal fluid analysis and skin temperature measurements may be used to establish the diagnosis. Aspirated bursal fluid is quantitatively analyzed on Gram stain, culture, white blood cell (WBC) count, and glucose level.

Positive Gram stain and culture definitively demonstrate a septic process. However, Gram stains are positive in only 50% to 60% of cases, and it may take several days to obtain the results of culture.[17,19] A WBC count <1,000/mm^3 is consistent with aseptic bursitis, and a WBC count >10,000/mm^3 is generally consistent with septic bursitis.[18,22] With counts between these levels, the predominant cell type may be used to distinguish septic from aseptic bursitis. A preponderance of polynuclear cells is indicative of septic bursitis, whereas predominance of mononuclear leukocytes is indicative of aseptic bursitis.[18,22] Bursal fluid glucose levels indicate infection when values are <50% of serum levels.[18]

Smith et al[17] measured skin temperature in 46 patients to distinguish septic from aseptic olecranon bursitis. Skin temperature of the affected bursa ≥2.2°C (36°F) warmer than the contralateral olecranon bursa predicted a septic process with 100% sensitivity and 94% specificity.[17] Two of 35 aseptic cases demonstrated this temperature differential. Mean surface temperature difference was 0.7°C (33.3°F) in aseptic cases and 3.7°C (39°F) in septic cases.

Management

Management of olecranon bursitis is dictated by its etiology. Acute traumatic or idiopathic olecranon bursitis typically resolves with nonsurgical management. Ice, compressive dressings, and avoidance of aggravating activity are sufficient in most patients.[23] When a patient does not improve as expected, aspiration should be performed to rule out infection. Alternatively, in the patient in whom fluid collection is bothersome at presentation, aspiration with or without concurrent corticosteroid injection may be done.[21] In a study of 47 patients with traumatic bursitis who underwent aspiration, 90% recovered in 6 months.[24] Intrabursal corticosteroid injection is associated with complications, including infection, skin atrophy, and chronic pain.[24]

Septic bursitis is managed with drainage of collected fluid, mechanical rest, and systemic antibiotics. Serial aspiration or open incision and drainage may be performed. Antibiotics may be administered orally or intravenously, but parenteral preparations require fewer days to sterilize the bursal fluid.[18] Although positive cultures guide the selection of antibiotics, most cases of septic bursitis are attributable to *Staphylococcus* and other gram-positive organisms.[21]

Several surgical procedures have been described for the management of olecranon bursitis, including traditional open bursectomy, arthroscopic bursal excision, and partial excision of the olecranon.[16,23,25] The skin incision should not be placed over the bony olecranon process during open procedures because even well-planned surgery may result in sensitive scars, adherent skin, and hypoesthesia.[16] Stewart et al[23] reported satisfactory outcomes at an average follow-up of 5.2 years in 15 of 16 nonrheumatoid patients who underwent surgical treatment for aseptic bursitis. Postoperatively, patients are often splinted in 90° of flexion for 2 weeks to rest the soft tissues and minimize hematoma formation.

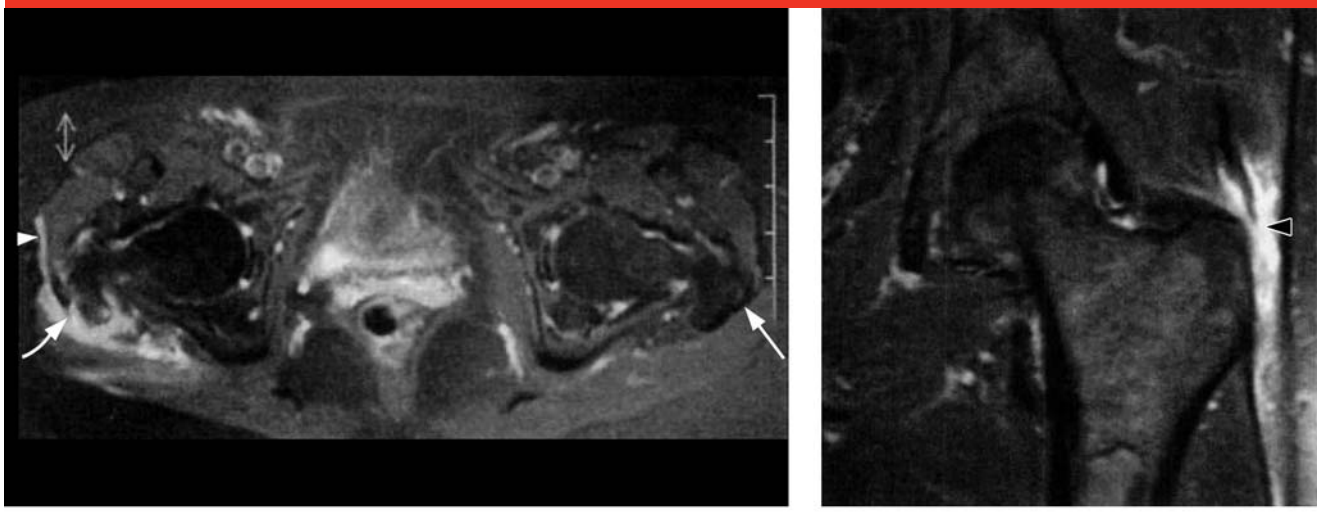
Trochanteric Bursitis

Anatomy and Pathophysiology

The trochanteric bursa lies deep to the iliotibial band, just superficial to the gluteus medius, at the lateral aspect of the proximal thigh[26] (Figure 2). The glutei medius and minimus have additional bursae deep to their respective tendons in the peritrochanteric area. The glutei medius and minimus attach to the greater trochanter superiorly and anteriorly, and they act to abduct and internally rotate the hip.

Tendinosis of the gluteus medius and/or minimus tendons is increasingly accepted as the primary pathology of trochanteric bursitis.[27,28] In fact, some authors have proposed the term "greater trochanter pain syn-

Figure 3



A, Axial T2-weighted magnetic resonance image demonstrating an inflamed trochanteric bursa (arrowhead) and tear of the gluteus medius tendon (curved arrow). Note the normal gluteus medius tendon (arrow) on the contralateral side. **B,** Coronal T2-weighted magnetic resonance image demonstrating tendinosis of the gluteus medius tendon (arrowhead). (Reproduced with permission from Kingzett-Taylor A, Tirman PF, Feller J, et al: Tendinosis and tears of gluteus medius and minimus muscles as a cause of hip pain: MR imaging findings. *AJR Am J Roentgenol* 1999;173[4]: 1123-1126.)

drome" (GTPS) as a more accurate description of the condition.[27] Gluteal tendon tearing is not well understood; thus, well-defined clinical indications, physical examination tools, and management options are needed.

In a review of 250 magnetic resonance images of the hip to evaluate pain of the buttock, lateral hip, and groin, Kingzett-Taylor et al[27] identified 35 patients with evidence of tendinopathy of the gluteus medius and minimus tendons (Figure 3). Twenty-two patients had gluteus medius tears (8 complete, 14 partial). Thirteen patients had tendinosis of the gluteus medius. The gluteus minimus was involved in 10 patients (5 each in the tear and tendinosis groups). Fourteen had evidence of fluid collection in the trochanteric bursa. Six patients had osteoarthritis of the hip. The authors concluded that tendinopathy of the gluteus medius and minimus tendons is a substantial and underrecognized cause of GTPS.

Another retrospective review of MRI studies reported that patients with trochanteric bursitis had abnormalities of the gluteus medius tendon without swelling of the trochanteric bursa.[28] Bird et al[29] reported that 20 of 24 patients with definitive diagnosis of trochanteric bursitis had either abnormal signal at the insertional gluteus medius or a frank tear at the musculotendinous junction on MRI.

Presentation and Physical Examination

Patients with trochanteric bursitis typically present with lateral hip pain, which may radiate to the buttock, groin, or low back. Symptoms may be exacerbated by ambulation, walking uphill, stair climbing, and rising from a seated position.

Physical examination may reveal normal range of motion at the hip joint. Positive findings include tenderness over the lateral aspect of the greater trochanter, Trendelenburg sign, pain with resisted abduction and internal rotation, and pain elicited with the Ober and flexion, abduction, and external rotation (FABER) tests. The Ober test is used to detect contracture of the iliotibial band. With the patient in the lateral position, the affected leg is abducted, and the hip is extended with the knee in extension. The leg is then allowed to adduct past neutral. A positive test is represented by inability to adduct past the midline. A positive FABER test is represented by pain in the sacroiliac region. According to the modified Krout and Anderson diagnostic criteria developed by Ege Rasmussen and Fanø,[30] the diagnosis of trochanteric bursitis requires lateral hip pain and tenderness over the greater trochanter as well as one of the following criteria: pain at the extremes of rotation, abduction, or adduction; pain on forceful contraction of the hip abductors; and pseudoradiculopathy, with pain primarily radiating down the lateral aspect of the

thigh. Although these criteria are widely used in practice, their sensitivity, specificity, and predictive value have not been established.[31]

Diagnosis

The diagnosis of trochanteric bursitis is typically clinical, made after excluding lumbar pathology such as spinal stenosis, spondylosis, and radiculopathy; intrinsic hip pathology such as osteoarthritis, osteonecrosis, and stress fracture; and local diseases, such as soft-tissue infection and bone and soft-tissue tumors.[31] MRI is a reliable modality in the diagnosis of trochanteric bursitis. Standard hip radiographs are obtained to evaluate for concomitant arthritic disease of the hip joint and prior trauma to the trochanter.

Bird et al[29] used MRI to assess 24 patients with lateral hip pain and tenderness over the greater trochanter. Three physical examination techniques were performed as well, including assessment of the Trendelenburg sign, pain on resisted abduction, and pain on resisted internal rotation. Only two patients had evidence of bursal distension on magnetic resonance images, and no patient exhibited distention without concomitant pathology of the gluteus medius. Trendelenburg assessment exhibited the greatest sensitivity, specificity, and intraobserver reliability.

Management

Initial management consists of physical therapy and oral NSAIDs. If symptoms persist, local glucocorticoid injection is performed.[32,33] Most patients respond to nonsurgical management.

Increased understanding of the underlying pathology of GTPS has led to the development of enhanced surgical options for refractory trochanteric bursitis. Reattachment of the abductor tendons into the bone has been described to manage tendinosis or partial or complete tear of the gluteus medius—the so-called rotator cuff tear of the hip.[34,35] Degenerative and necrotic tissue is débrided, and the tendon stump is secured to the trochanter with suture anchors. In a small series by Lequesne,[26] six of seven patients who underwent this procedure were symptom-free after surgery. The remaining patient had partial improvement.

Govaert et al[36] described a trochanteric reduction osteotomy for the management of recalcitrant trochanteric bursitis. The authors initially used the technique as a salvage procedure for patients with failed arthroscopic bursectomy and iliotibial band release. The osteotomy is performed medial to the gluteus medius insertion proximally and extends beyond the vastus ridge distally. Depending on the prominence of the trochanter, a wafer of bone measuring 5 to 10 mm thick is removed. The trochanter is transferred medially and distally and secured with two 4.5-mm cortical lag screws. Good results were reported at a mean follow-up of 23.5 months.

Complications of trochanteric osteotomy have been well described in the setting of total hip arthroplasty. Nonunion is the primary complication, occurring in 5% to 32% of cases.[37] Impaired abductor function is another potential complication. Transfer of the greater trochanter distally, as is done in trochanteric advancement, maintains the length and strength of the hip abductors.[38]

Retrocalcaneal Bursitis

Inflammation of the retrocalcaneal bursa can limit function and cause pain. The Achilles tendon and its bony insertion may be involved in severe cases. This spectrum of disease has been given many names, including Haglund syndrome, Albert disease, calcaneus altus, pump bump, winter heel, and achillodynia.[39]

Anatomy

The posterior calcaneal tuberosity serves as the attachment point for the Achilles tendon. This tuberosity, which is located just proximal to the insertion of the Achilles tendon, is covered with fibrocartilage[40] (Figure 4). This area typically defines the anterior wall of the retrocalcaneal bursa. The bony projection of Haglund deformity typically lies superior to this point. The retrocalcaneal bursa lies between the calcaneus anteriorly and the Achilles tendon. A synovial lining on the superior aspect separates the bursa from the Achilles fat pad (ie, Kager fat pad).[41] This fat pad is bordered by the flexor hallucis longus anteriorly, the calcaneus inferiorly, and the Achilles tendon posteriorly. The Achilles fat pad appears as a sharply marginated, radiolucent area on radiographs.

The anterior wall of the retrocalcaneal bursa is cartilaginous, and the posterior wall is tendinous. The bursa is filled with highly viscous fluid rich in hyaluronate.[42]

Pathology and Physical Examination

Because of the close anatomic relationship between the elements of the posterior aspect of the calcaneus, pathology affecting one structure often affects the surrounding structures as well. Pain anterior to the Achilles tendon and just superior to the calcaneus is the hallmark of retrocalcaneal bursitis. Patients often have a positive two-finger squeeze test, that is, pain when pressure is applied with two fingers placed medially and laterally anterior to the Achilles insertion.[43] Pain may be elicited with dorsiflexion of the foot and on active

Figure 4

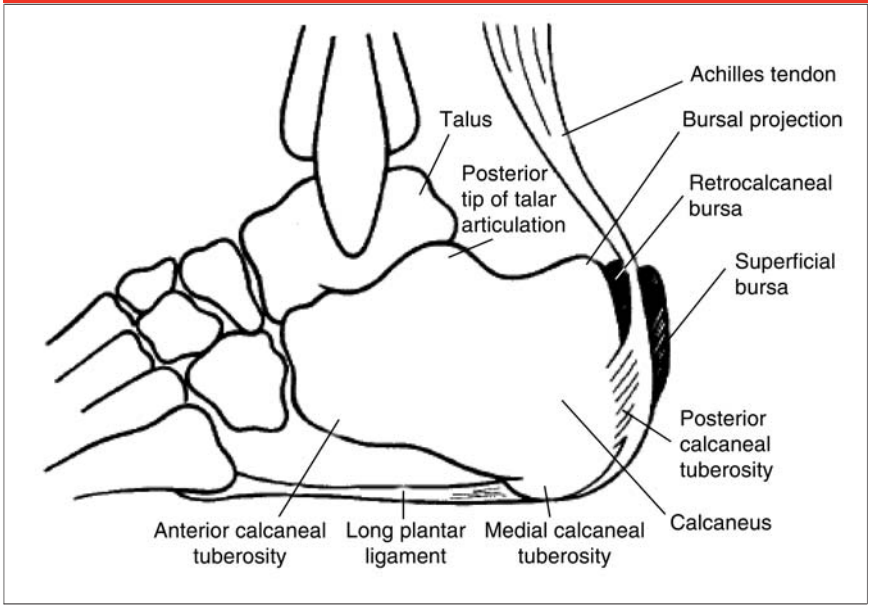


Illustration demonstrating the anatomy of the hindfoot. The posterior calcaneal tuberosity is covered with fibrocartilage just proximal to the insertion of the Achilles tendon. This tuberosity apposes the anterior wall of the retrocalcaneal bursa. (Reproduced with permission from Stephens MM: Haglund's deformity and retrocalcaneal bursitis. *Orthop Clin North Am* 1994;25[1]:41-46.)

resisted plantar flexion, as well.[39] Pain is primarily caused by overuse; however, pain resulting from septic retrocalcaneal bursitis has been reported.[44] Retrocalcaneal bursitis is particularly common in runners, especially those who regularly train on inclines, because ankle dorsiflexion augments stress on the bursa. Persons with hindfoot varus as well as those with a rigid plantarflexed first ray are also susceptible to retrocalcaneal bursitis. A diagnosis of bilateral retrocalcaneal bursitis is suggestive of inflammatory arthritis.[43]

The morphologic relationship between the shape of the posterior tuberosity and retrocalcaneal bursitis is unclear. Although prominence of the posterosuperior lateral aspect of the tuberosity (ie, Haglund deformity) seems to be related to the occurrence of bursitis, it is by no means causal. These deformities are seldom symptomatic. Symptoms typically manifest as pain lateral to the Achilles insertion.

Insertional Achilles tendinosis is another common diagnosis that must be differentiated from retrocalcaneal bursitis. This tendinitis occurs with or without such bursal involvement. Insertional Achilles tendinosis causes pain directly at the insertion of the Achilles tendon.[45]

Management

Management of these causes of posterior heel pain begins with ice, activity modification, NSAIDs, and orthoses. Shoe wear modification to prevent irritation of the posterior heel by the shoe counter should be considered, as well. Maneuvers that stretch the local Achilles tendon may aid in attenuating the symptoms. Recent evidence in a rabbit model indicates that steroid injection into the retrocalcaneal bursa may adversely affect the biomechanical properties of the Achilles tendon.[46] Careful consideration is warranted prior to administration of corticosteroid injection.

Surgical intervention is warranted for retrocalcaneal bursitis that does not resolve with nonsurgical management. Accurate clinical diagnosis guides surgical management. For refractory cases associated with Haglund deformity, open procedures include resection of the calcaneal prominence proximal to the Achilles insertion, débridement of Achilles tendinopathy, and complete excision of the retrocalcaneal bursa.[47-49] Alternatively, dorsal closing wedge osteotomy may be considered to rotate the posterior calcaneus to a lesser prominence.[50] Complications of open procedures include skin breakdown, Achilles tendon avulsion, altered sensation, and painful scar formation.[39,51]

Although the goal of these procedures is the removal of inflamed tissue, recovery time is based in part on the etiology of the symptoms. Watson et al[45] reported significantly longer recovery times in patients whose primary etiology of posterior heel pain was preexisting calcific tendinitis of the Achilles tendon ($P < 0.05$).

Endoscopic techniques were developed to reduce recovery time and decrease morbidity compared with open procedures. Ortmann and McBryde[51] reported excellent results in their series of 30 patients who underwent endoscopic bony and soft-tissue decompression for the management of retrocalcaneal bursitis with Haglund deformity. Of the 28 patients available for follow-up, average American Orthopaedic Foot and Ankle Society score increased from 62 preoperatively to 97 postoperatively.

Summary

Prepatellar, olecranon, trochanteric, and retrocalcaneal bursitis should be considered in the differential diagnosis in the patient with musculoskeletal pain. Bursitis must be distinguished from other causes of pain, including arthritis, tendinitis, fracture, tendon or ligament injury, and nerve pathology. Infectious etiology must be promptly ruled out or managed. Although nonsurgical management is a therapeutic mainstay, refractory cases may require surgical intervention. The orthopaedic surgeon should be familiar with the pathophysiology and clinical presentation of the most common forms of bursitis as well as useful diagnostic modalities and management options.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 6, 8, 33, 34, and 46 are level I studies. References 17, 29, 31, and 37-39 are level II studies. References 1, 27, 28, 43, 45, and 50 are level III studies. References 2, 4, 5, 9-13, 16, 19-26, 30, 35, 36, 41, 42, 44, 47-49, and 51 are level IV studies.

References printed in **bold type** are those published within the past 5 years.

1. Cea-Pereiro JC, Garcia-Meijide J, Mera-Varela A, Gomez-Reino JJ: A comparison between septic bursitis caused by Staphylococcus aureus and those caused by other organisms. *Clin Rheumatol* 2001;20(1):10-14.
2. Schickendantz MS, Watson JT: Mycobacterial prepatellar bursitis. *Clin Orthop Relat Res* 1990;258:209-212.
3. McAfee JH, Smith DL: Olecranon and prepatellar bursitis: Diagnosis and treatment. *West J Med* 1988;149(5):607-610.
4. **Traboulsi R, Uthman I, Kanj SS: Prepatellar Brucella melitensis bursitis: Case report and literature review. *Clin Rheumatol* 2007;26(11):1941-1942.**
5. Wang JP, Granlund KF, Bozzette SA, Botte MJ, Fierer J: Bursal sporotrichosis: Case report and review. *Clin Infect Dis* 2000;31(2):615-616.
6. Stell IM, Gransden WR: Simple tests for septic bursitis: Comparative study. *BMJ* 1998;316(7148):1877.
7. Langford CA, Gilliland BC: Periarticular disorders of the extremities, in Fauci AS, Braunwald E, Kasper DL, et al, eds: *Harrison's Principles of Internal Medicine*, 17e. http://www.accessmedicine.com/content.aspx?aID=2863288.
8. Stell IM: Management of acute bursitis: Outcome study of a structured approach. *J R Soc Med* 1999;92(10):516-521.
9. Knight JM, Thomas JC, Maurer RC: Treatment of septic olecranon and prepatellar bursitis with percutaneous placement of a suction-irrigation system: A report of 12 cases. *Clin Orthop Relat Res* 1986;206:90-93.
10. Quayle JB, Robinson MP: An operation for chronic prepatellar bursitis. *J Bone Joint Surg Br* 1976;58(4):504-506.
11. Ogilvie-Harris DJ, Gilbart M: Endoscopic bursal resection: The olecranon bursa and prepatellar bursa. *Arthroscopy* 2000;16(3):249-253.
12. **Ike RW: Chemical ablation as an alternative to surgery for treatment of persistent prepatellar bursitis. *J Rheumatol* 2009;36(7):1560.**
13. Pien FD, Ching D, Kim E: Septic bursitis: Experience in a community practice. *Orthopedics* 1991;14(9):981-984.
14. Jaffe L, Fetto JF: Olecranon bursitis. *Contemp Orthop* 1984;8:51-56.
15. Chen J, Alk D, Eventov I, Wientroub S: Development of the olecranon bursa: An anatomic cadaver study. *Acta Orthop Scand* 1987;58(4):408-409.
16. Quayle JB, Robinson MP: A useful procedure in the treatment of chronic olecranon bursitis. *Injury* 1978;9(4):299-302.
17. Smith DL, McAfee JH, Lucas LM, Kumar KL, Romney DM: 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(7):1581-1585.
18. Ho G Jr, Tice AD: Comparison of nonseptic and septic bursitis: Further observations on the treatment of septic bursitis. *Arch Intern Med* 1979;139(11):1269-1273.
19. Canoso JJ, Yood RA: Reaction of superficial bursae in response to specific disease stimuli. *Arthritis Rheum* 1979;22(12):1361-1364.
20. Cruz C, Shah SV: Dialysis elbow: Olecranon bursitis from long-term hemodialysis. *JAMA* 1977;238(3):243.
21. Morrey BF: Bursitis, in Morrey BF, Sanchez-Sotelo J, eds: *The Elbow and its Disorders*, ed 4. Philadelphia, PA, Saunders, 2009, pp 1164-1173.
22. Canoso JJ: Idiopathic or traumatic olecranon bursitis: Clinical features and bursal fluid analysis. *Arthritis Rheum* 1977;20(6):1213-1216.
23. Stewart NJ, Manzanares JB, Morrey BF: Surgical treatment of aseptic olecranon bursitis. *J Shoulder Elbow Surg* 1997;6(1):49-54.
24. Weinstein PS, Canoso JJ, Wohlgethan JR: Long-term follow-up of corticosteroid injection for traumatic olecranon bursitis. *Ann Rheum Dis* 1984;43(1):44-46.
25. Kerr DR, Carpenter CW: Arthroscopic resection of olecranon and prepatellar bursae. *Arthroscopy* 1990;6(2):86-88.
26. **Lequesne M: From "periarthritis" to hip "rotator cuff" tears: Trochanteric tendinobursitis. *Joint Bone Spine* 2006;73(4):344-348.**
27. Kingzett-Taylor A, Tirman PF, Feller J, et al: Tendinosis and tears of gluteus medius and minimus muscles as a cause of hip pain: MR imaging findings. *AJR Am J Roentgenol* 1999;173(4):1123-1126.
28. Oakley SP, Bird PA, Kirkham BW: Gluteus medius tears presenting as the clinical syndrome of trochanteric bursitis. *Arthritis Rheum* 1999;42(suppl 9):S340.
29. Bird PA, Oakley SP, Shnier R, Kirkham BW: Prospective evaluation of magnetic resonance imaging and physical examination findings in patients with greater trochanteric pain syndrome. *Arthritis Rheum* 2001;44(9):2138-2145.
30. Ege Rasmussen KJ, Fanø N: Trochanteric bursitis: Treatment by corticosteroid injection. *Scand J Rheumatol* 1985;14(4):417-420.
31. Alvarez-Nemegyei J, Canoso JJ: Evidence-based soft tissue rheumatology: III. Trochanteric bursitis. *J Clin Rheumatol* 2004;10(3):123-124.
32. Shbeeb MI, Matteson EL: Trochanteric bursitis (greater trochanter pain syndrome). *Mayo Clin Proc* 1996;71(6):565-569.
33. Schapira D, Nahir M, Scharf Y: Trochanteric bursitis: A common clinical problem. *Arch Phys Med Rehabil* 1986;67(11):815-817.
34. Kagan A II: Rotator cuff tears of the hip. *Clin Orthop Relat Res* 1999;(368):135-140.
35. Bunker TD, Esler CN, Leach WJ: Rotator-cuff tear of the hip. *J Bone Joint Surg Br* 1997;79(4):618-620.

36. Govaert LH, van der Vis HM, Marti RK, Albers GH: Trochanteric reduction osteotomy as a treatment for refractory trochanteric bursitis. *J Bone Joint Surg Br* 2003;85(2):199-203.

37. Glassman AH: Complications of trochanteric osteotomy. *Orthop Clin North Am* 1992;23(2):321-333.

38. Free SA, Delp SL: Trochanteric transfer in total hip replacement: Effects on the moment arms and force-generating capacities of the hip abductors. *J Orthop Res* 1996;14(2):245-250.

39. Leitze Z, Sella EJ, Aversa JM: Endoscopic decompression of the retrocalcaneal space. *J Bone Joint Surg Am* 2003;85(8):1488-1496.

40. Stephens MM: Haglund's deformity and retrocalcaneal bursitis. *Orthop Clin North Am* 1994;25(1):41-46.

41. Theobald P, Bydder G, Dent C, Nokes L, Pugh N, Benjamin M: The functional anatomy of Kager's fat pad in relation to retrocalcaneal problems and other hindfoot disorders. *J Anat* 2006;208(1):91-97.

42. Canoso JJ, Liu N, Traill MR, Runge VM: Physiology of the retrocalcaneal bursa. *Ann Rheum Dis* 1988;47(11):910-912.

43. Schepsis AA, Jones H, Haas AL: Achilles tendon disorders in athletes. *Am J Sports Med* 2002;30(2):287-305.

44. Harwell JI, Fisher D: Pediatric septic bursitis: Case report of retrocalcaneal infection and review of the literature. *Clin Infect Dis* 2001;32(6):E102-E104.

45. Watson AD, Anderson RB, Davis WH: Comparison of results of retrocalcaneal decompression for retrocalcaneal bursitis and insertional achilles tendinosis with calcific spur. *Foot Ankle Int* 2000;21(8):638-642.

46. Hugate R, Pennypacker J, Saunders M, Juliano P: The effects of intratendinous and retrocalcaneal intrabursal injections of corticosteroid on the biomechanical properties of rabbit Achilles tendons. *J Bone Joint Surg Am* 2004;86(4):794-801.

47. Angermann P: Chronic retrocalcaneal bursitis treated by resection of the calcaneus. *Foot Ankle* 1990;10(5):285-287.

48. Pauker M, Katz K, Yosipovitch Z: Calcaneal ostectomy for Haglund disease. *J Foot Surg* 1992;31(6):588-589.

49. Schneider W, Niehus W, Knahr K: Haglund's syndrome: Disappointing results following surgery. A clinical and radiographic analysis. *Foot Ankle Int* 2000;21(1):26-30.

50. Miller AE, Vogel TA: Haglund's deformity and the Keck and Kelly osteotomy: A retrospective analysis. *J Foot Surg* 1989;28(1):23-29.

51. Ortmann FW, McBryde AM: Endoscopic bony and soft-tissue decompression of the retrocalcaneal space for the treatment of Haglund deformity and retrocalcaneal bursitis. *Foot Ankle Int* 2007;28(2):149-153.