Conservative Treatment of Inflamed Knee Bursae

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Knee bursitis often mimics other pathologies, making correct diagnosis necessary to initiate appropriate treatment. Most commonly affected are the prepatellar, pes anserine, tibial collateral ligament, and two infrapatellar bursae. Other common bursitic conditions include Baker's cysts and posttraumatic adventitious hemorrhagic bursitis. Most of these can be treated conservatively with aspiration of fluid from the bursa, rest, ice, immobilization, and injection of a corticosteroid and analgesic combination. Some chronic bursitic conditions may require surgical excision of the bursa.

Many disorders involving the flexor and extensor mechanisms of the knee and associated bursae are being seen because the growing number of athletes tend to place high levels of stress on their knees. These bursitic conditions can be characterized both by etiology (acute overuse, chronic overuse, traumatic, or infective)

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The stress of repetitive kneeling leads to a relatively high incidence of prepatellar bursitis among wrestlers.
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Figure 1. The clinical appearance of prepatellar bursitis in a 17-year-old male wrestler.

Figure 2. Surgically exposed prepatellar bursa of a 20-year-old college wrestler.

and by location. Most often bursitis can be treated conservatively in a physician’s office. However, familiarity with the anatomic positions of the commonly affected bursae is essential to correctly diagnose and treat the condition (see “Identifying Bursae,” page 72).

Other conditions that must be differentiated from bursitis, such as crystal-induced bursitis, rheumatoid arthritis, other chronic inflammatory arthritides, and tumors or foreign bodies are seldom seen in young, otherwise healthy athletes. If these conditions are of concern in a given patient, routine x-rays of the affected joint can generally distinguish them from the types of bursitis that more commonly affect athletes. Note that not all knee bursae are affected by bursitis, but knowing the location of all the bursae may aid diagnosis in those often affected.

Prepatellar Bursitis

Acute Nonpyogenic. Commonly known as coal miner’s, carpet layer’s, or housemaid’s knee, acute nonpyogenic bursitis of the prepatellar bursa develops in response to frictional stress directly over the bursa, such as that caused by repetitive kneeling. It is common among wrestlers and trampolinists (figure 1). Patients who develop acute nonpyogenic prepatellar bursitis generally present with tenderness and swelling directly over the patella. They have decreased range of motion of the knee because pain limits movement as the skin tightens over the patella during maximum flexion.

Pyogenic. Pyogenic bursitis of the prepatellar bursa develops as a result of penetrating or integumental injuries, including recent aspiration of the bursa. The patient’s history can thus alert the physician to the diagnosis. Patients who have pyogenic prepatellar bursitis present with red, tender swelling directly over the patella, and occasionally with associated cellulitis. Patients may also have inguinal lymphadenopathy. Old wounds, puncture sites, or abrasions may be evident. Pyogenic bursitis may be indistinguishable clinically from nonpyogenic bursitis; thus, microbiological study of the aspirate is necessary.

Treatment. Using aseptic technique and a wide-bore needle, physicians should initiate treatment for prepatellar bursitis by aspirating the fluid-filled bursa. In nonpyogenic bursitis, the aspirate is a clear, serouslike fluid. Following aspiration, physicians can inject the bursa with a combined corticosteroid and local analgesic, and prescribe rest, ice, compression, and elevation, which can be augmented with a posterior splint or knee immobilizer.

In pyogenic bursitis, the aspirate is cloudy and viscous. Culture of this fluid is always indicated. Commonly, the pathogen is staphylococcus; streptococcus is involved less frequently.
Because penicillin-resistant bacteria may be involved, physicians should treat these infections with a penicillinase-resistant penicillin until antibiotic sensitivities are available.

If a patient's pyogenic bursitis does not respond to antibiotics, or the bursa is extremely large, open drainage with excision of the bursa may be necessary.

**Chronic Inflammatory Prepatellar Bursitis.**
Chronic inflammatory prepatellar bursitis may result from repeated episodes of nonpyogenic prepatellar bursitis or from a single resolved episode of pyogenic bursitis. Repetitive mild traumas or previous infection cause thickening of the bursal wall.

According to Sharrard, the surrounding vascular network bleeds into the bursa, resulting in distention. However, the aspirate is often serous, without evidence of blood staining. A normal bursal wall has a characteristic frond pattern and synovial lining containing secretory cells (figure 3). Histologically, we (G.C.R., M.J.B., P.W.C.) have been unable to demonstrate secretory cells in chronically inflamed bursal linings (figure 4). We speculate that distension is caused by transudation of fluid from tissue surrounding the bursa.

Many patients feel little or no pain, and thus do not seek medical attention until the bursa has become grossly distended. They present with large, fluctuant, nontender masses directly over their patellas. These masses may extend several inches up the thigh or several inches down the anterior proximal tibia.

**Treatment.** Initial treatment for chronic prepatellar bursitis involves aspiration of fluid from the bursa and injection of a corticosteroid to reduce the inflammatory reaction. Compression may help limit further distention of the bursa. Repetitive aspirations may be necessary.

If this conservative approach fails, surgical excision of the bursa is indicated. Quayle and Robinson have described a surgical procedure in which only the posterior wall of the bursa is removed, preventing the skin from sticking and scarring to the underlying tissue. This procedure is easier to perform than excision of the entire bursa, and it is less traumatic for patients, resulting in fewer complications. In the senior author's (G.C.R's) experience, this procedure gives excellent results, and leaving the anterior wall of the bursa intact does not appear to predispose patients to recurrence of bursitis. Following the operation, the surgeon should institute suction drainage and prescribe a posterior extension splint to immobilize the knee for at least 10 to 14 days.

To help prevent recurrent chronic prepatellar bursitis and the other prepatellar bursitis conditions, physicians should recommend that patients use protective knee pads.
Infrapatellar Bursitis

Infrapatellar bursitis may involve either the superficial infrapatellar bursa or the deep infrapatellar bursa.

Superficial Infrapatellar Bursitis. Bursitis of the superficial bursa, like that of the prepatellar bursae, develops following repetitive mild trauma, such as kneeling on the bursa. In contrast to the theory that the most consistently occurring bursae develop during fetal life, Sharrard has postulated that the superficial infrapatellar bursa is not an inborn structure, but one that develops only in response to repetitive kneeling. Unlike prepatellar bursitis, though, infrapatellar bursitis usually arises from kneeling in an upright position and therefore is commonly known as vicar's knee. Generally, it heals well with compression and ice packs and does not require complete immobilization or drainage.

Deep Infrapatellar Bursitis. Bursitis of the deep infrapatellar bursa may follow direct trauma over the patellar tendon, such as striking the knees on a hard surface during a fall. This bursitis may be difficult to distinguish clinically from posttraumatic rupture of the patellar tendon or hemorrhage into the retropatellar fat pad. Patients who have these conditions all have pain and tenderness over the patellar tendon, and they are unable to fully flex or extend their knees. MRI is frequently necessary to confirm the diagnosis.

Frictional or overuse injuries may also cause deep infrapatellar bursitis, which is then characterized by pain deep in the patellar tendon just proximal to its insertion on the tibial tuberosity. Fluid accumulation in the bursa may cause the sac to bulge on either side of the patellar tendon.

The symptoms of Osgood-Schlatter disease and patellar tendinitis may closely mimic those of frictional or overuse deep infrapatellar bursitis. Osgood-Schlatter disease may in fact coexist with the bursitis. Radiography, which can demonstrate apophyseal and residual ossicles present in the tendon, may help establish the diagnosis. Distinguishing patellar tendinitis from deep infrapatellar bursitis is difficult, although patients with the latter condition generally feel pain deep to the tendon.

Identifying Bursae

Bursae are closed sacs or cystic spaces lined with a synovial membrane that closely resembles that found in synovial joints. The anatomic locations of the knee bursae vary—as do the descriptions of their locations. Some bursae are inconsistently found; some frequently form communications with adjacent bursae or coalesce to form larger sacs.

Bursae that occur most consistently develop during fetal life and are found interposed between tendon or ligament sheaths and bony prominences. These bursae, which promote joint mobility and protect adjacent muscular and skeletal tissues by reducing friction, include bursae located circumferentially around the knee joint.

Anterior Bursae. Three bursae are located on the anterior aspect of the knee: the large prepatellar bursa, which lies between the patella and the skin, is the most commonly involved in bursitic conditions. The second, the small superficial infrapatellar bursa, lies between the skin and the proximal portion of the patellar tendon. The third, the smaller deep infrapatellar bursa, is found between the distal patellar tendon and the proximal tibia (figures A and B).

Lateral Bursae. Four bursae commonly occur on the lateral aspect of the knee: the first lies on the posterolateral aspect of the knee between the lateral head of the gastrocnemius and the joint capsule. This bursa normally communicates with the knee joint. Two other bursae are located along the fibular collateral ligament. One lies between this ligament and the biceps tendon; the other lies between the ligament and the popliteal tendon. The fourth laterally occurring bursa lies between the popliteal tendon and the lateral condyle of the femur.

Medial Bursae. Four bursae commonly occur on the medial aspect of the knee: The first
lies between the medial head of the gastrocnemius and the capsule. This bursa sometimes includes an extension between the tendons of the gastrocnemius and the semimembranous muscles. The second, the pes anserine bursa, lies between the tibial collateral ligament and the tendon insertions of the sartorius, gracilis, and semitendinosus muscles. The third lies between the tibial collateral ligament and tendon of the semimembranosus. The fourth, the tibial collateral ligament bursa, lies just beneath the tibial collateral ligament and directly over the medial meniscus. Occasionally people have a fifth bursa lying between the tendon of the semimembranosus and the semitendinosus muscles.

Bursae not found consistently include adventitious bursae, which may develop later in life in response to major trauma or to friction or microtrauma over a bony prominence such as a large exostosis.

In fetal life, the suprapatellar bursa is separate, but after birth, it develops into an extension of the synovial cavity. Thus, it is more correct to refer to this entity as the suprapatellar pouch or suprapatellar reflexion.

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If fluid accumulation does cause the bursa to bulge, the physician should aspirate the fluid and inject a combined steroid-lidocaine medication into the bursa. Following injection, however, a certain amount of steroid may be absorbed into the tendon itself, leaving it vulnerable to rupture. Therefore, patients should avoid for 2 weeks any activity that may put undue stress on the patellar tendon.

Pes Anserine Bursitis

Although physicians most often see pes anserine bursitis in older, obese patients, it is becoming more common among younger people, particularly in runners. The pes anserine bursa’s position on the anteromedial aspect of the knee, just beneath the pes anserinus tendons and over the tibial attachment of the tibial collateral ligament, subjects it to heavy frictional loads during activities such as running. This is particularly true for patients who have flatfoot and genu valgum, conditions that result in excessive pronation and valgus stress at the knee. Running on crowned hills or roads may also exacerbate the problem.

To establish a diagnosis of pes anserine bursitis, physicians must distinguish the condition from a partial tear of the tibial attachment of the tibial collateral ligament. Patients who have either condition report point tenderness over the proximal metaphyseal region. However, a patient who has pes anserine bursitis should not feel increased pain when valgus stress is applied to his or her knee at 30° flexion.

Treatment. To treat pes anserine bursitis, physicians should inject a corticosteroid and a local analgesic directly into the bursa and prescribe ice therapy. Larsson and Baum have suggested that lidocaine alone be injected first, followed by a mixture of local anesthetic and corticosteroid if symptoms persist. Surgery is rarely required. However, patients should rest their knees until all symptoms resolve; this form of bursitis may easily become chronic.

Tibial Collateral Ligament Bursitis

Patients who have tibial collateral ligament bursitis present with tenderness over the tibial collateral ligament at the joint line, closely mimicking symptoms associated with a medial meniscus tear. According to Kerlan and Glousman, physicians should consider a diagnosis of tibial collateral ligament bursitis when a patient has these symptoms without a history of trauma to the knee.

Treatment. To treat tibial collateral ligament bursitis, inject lidocaine combined with a steroid directly into the bursa, then encourage a gradual return to full activity. When uncertain of the diagnosis in a patient who has the typical symptoms, the senior author follows this treatment first but proceeds with arthroscopy if treatment is unsuccessful.

Baker’s Cyst

A Baker’s cyst traditionally has been considered a herniation of the synovial membrane at the posterior medial aspect of the knee caused by an increase in intra-articular pressure (figure 5). However, it now appears that distension of the bursa(e) associated with the gastrocnemius and semimembranosus muscles is the same condition as Baker’s cyst. This distention may result from inflammatory joint disease, degenerative joint disease, a meniscus tear, or a congenital defect. A one-way valve mechanism may be at work, allowing fluid into the cyst but preventing it from returning to the knee joint. The pumping action of muscles while running aggravates this problem, literally pumping fluid into the cyst. Marked fluid distention may result and may lead to nerve and vessel impingement. Or the cyst may rupture, causing swelling and inflammation in the calf that resemble the clinical symptoms of deep vein thrombosis. Baker’s cyst and deep vein thrombosis may coexist. A venogram is often necessary to distinguish the pathologies.

A physician may demonstrate a communicating Baker’s cyst simply by injecting radiopaque dye into the patient’s knee joint (figure 6). A communication is best seen after the patient has exercised the knee joint.

When a patient presents with a large, soft mass on the posterior aspect of his or her knee, also look for popliteal aneurysm or soft-tissue
tumor. These conditions are easily differentiated by ultrasound or MRI. However, a biopsy may be necessary to confirm the diagnosis of a soft-tissue tumor.

**Treatment.** Aspiration and injection of a corticosteroid is the first line of treatment for a Baker’s cyst, but the results are usually disappointing. Physicians must take care to avoid neurovascular structures when aspirating the fluid and injecting the medication.

Treatment of a chronic Baker’s cyst caused by meniscal pathology requires correction of the underlying lesion. Meniscal derangement can cause increased synovial fluid production, resulting in increased intra-articular pressure. The latter, in turn, can cause herniation through the posteromedial capsule. Arthroscopic correction of a meniscal tear is often sufficient to normalize intra-articular pressure.

If cysts persist despite correcting the underlying pathology, physicians can excise them and carefully close any communication with the knee joint. Postoperatively, physicians should immobilize patients’ knees in a slightly flexed position for 3 to 4 weeks. The patient should then begin a gradually progressive rehabilitative program to restore strength and range of motion.

For congenital Baker’s cysts in children, observation may be the best treatment; many resolve spontaneously.

**Hemorrhagic Adventitious Bursitis**

As stated previously, adventitious bursae are commonly found between bony prominences and overlying soft tissue where friction is great: for example, bursae seen in hallux valgus. Adventitious bursae may also form in the anterior knee region between the skin and the extensor mechanism following direct blunt trauma and secondary organization of large hematomas. Generally, hematomas undergo organization, lysis, and resorption by the body. However, hematomas around the knee undergo these changes less readily than in other anatomic locations, possibly because they are less exposed to surrounding absorbent tissue and to the circulating flow of blood.

Following direct blunt trauma to the anterior...
Direct, blunt trauma to the knee, such as that incurred in a collision or fall, may cause posttraumatic hemorrhagic adventitious bursitis.

knee, blood pools between the skin, the superficial fascia, and the fascia investing the extensor mechanism, particularly the retinaculum. A large volume of this organizing clot may dissect the fascia, forming a cleft. Repeated flexion and extension of the knee can propel the clot circumferentially within the cleft, where it develops a thick fibrous capsule or pseudosheath around itself, forming a bursa. With subsequent minor trauma, the newly developed adventitious bursa may become more distended, probably as a result of further hemorrhage into the bursa or transudation of fluid from the surrounding tissue. The pseudosheath lining, like the inner bursal lining in chronic bursitis, does not contain any secretory cells.

Generally, traumatic bursae do not communicate with the joint, although one communicating traumatic bursa has been reported. Radioactive dye injected into the bursa and then seen within the joint indicates disruption of the underlying extensor mechanism.

**Treatment.** Early treatment of acute posttraumatic hemorrhagic adventitious bursitis involves sterile aspiration and ice therapy to prevent further bleeding and movement of the pooled blood. The patient's knee should be compressed and placed in a knee immobilizer in extension for 1 to 2 weeks until healing is complete.

For patients who have chronic posttraumatic hemorrhagic adventitious bursitis, the first line of treatment should be aspiration followed by injection of corticosteroids. If the fibrous sheath has formed and fluid remains, surgical excision may be indicated. Following excision, the physician should place a suction drain for 24 to 48 hours and immobilize the patient's knee in a posterior splint in extension for at least 2 weeks.

**In-Office Treatment**

Most of the bursitic conditions that commonly affect athletes can be treated in the office with aspiration, corticosteroids, and immobilization. Specific treatment depends on the etiology and pathogenesis of the condition. However, initial diagnosis is paramount: Prompt, effective treatment yields gratifying results at a low cost and with little morbidity to the patient. It also prevents the need for further invasive procedures.

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References