Anserine syndrome

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ABSTRACT

Knee pain is a common complaint in clinical practice, and *pes anserinus* tendino-bursitis syndrome (PATB) has been frequently diagnosed based only on clinical features that may cause equivocal interpretations. Patients complain of characteristic spontaneous medial knee pain with tenderness in the inferomedial aspect of the joint. Studies with different imaging modalities have been undertaken during the last years to identify whether these patients suffer from bursitis, tendinitis, or both. Nevertheless, little is known regarding the structural defect responsible for this disturbance. Due to these problems and some controversies, we suggest the term "anserine syndrome" for this condition. Diabetes Mellitus is a known predisposing factor for this syndrome. Overweight and osteoarthritis seem to represent additional risk factors; however, their role in the pathophysiology of the disease is not yet understood. Treatment includes non-steroidal anti-inflammatory drugs, physiotherapy, and injections of corticosteroid, with highly variable responses, from 10 days to 36 months to achieve recovery. The lack of knowledge about its epidemiological, etiological, and pathophysiological aspects requires future studies for this common and intriguing disorder.

Keywords: anserine bursitis, anserine tendinitis, *pes anserinus* tendino-bursitis, *pes anserinus*.

INTRODUCTION

The combined insertion of the sartorius, gracilis, and semitendinous tendons, approximately 5 cm distally of the medial portion of the knee, forms a structure that resembles the natatory membrane of the goose, therefore it has been called "goosefoot" or, from the Latin, *pes anserinus*. Those muscles are primarily flexors of the knee with a secondary influence on the internal rotation of the tibia, protecting the knee against rotation and valgus stress.[1] The anserine bursa is one of 13 bursae found around the knee, being located immediately below the *pes anserinus* (Figures 1 and 2). Usually, it does not communicate with the knee.

The first description of changes in this region in the literature dates back to 1937 when Moschcowitz reported knee pain almost exclusively in women, who complained of pain when going downstairs or upstairs, upon rising from a chair, or referred difficulty when flexing the knees.[2]

The distinction between anserine bursitis and tendinitis is clinically difficult due to the proximity of the tissues; however, it is not significant because treatment is the same for both conditions.

This syndrome has been observed in long-distance runners.[3] Diabetes mellitus (DM) has been identified in a substantial proportion of those patients.[4] Cases of chronic bursitis have been documented in patients with rheumatoid arthritis and osteoarthritis.[5,6]

Etiology also includes trauma, retraction of posterior thigh muscles, bone exostosis, irritation of the suprapatellar plica, damage to the medial meniscus, *pes planus*, *genu valgum*, infection, and foreign body reaction.[7]

Although the majority of authors call this condition "anserine bursitis", the structure responsible for the symptoms is not identified in most cases. More recent studies have questioned the anatomical validity of anserine disease as an inflammatory condition of the bursa and/or tendon.[8] Little is known about its pathology. A prospective study to determine whether these patients really suffer from tendinitis or bursitis has not been undertaken.

To date, the diagnosis is based on the symptoms, which include pain in the medial aspect of the knee when going upstairs or downstairs, sensitivity to palpation (digital pressure) on the area of insertion, and, occasionally, local

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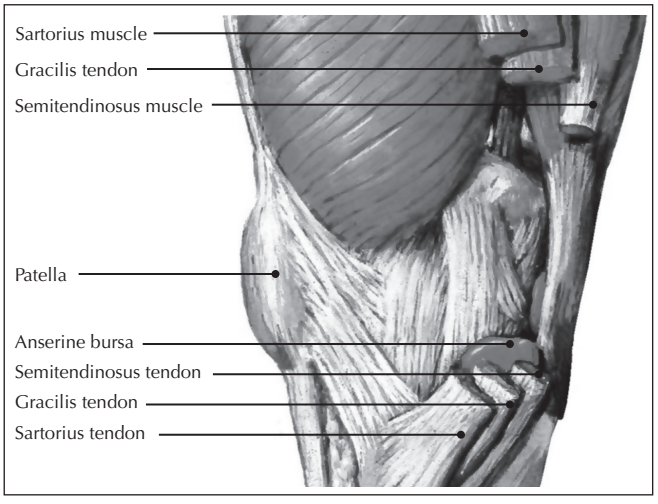

Figure 1
Medial view of the knee.
Shows the intimate relationship between the tendons that form the *pes anserinus* and the anserine bursa, implying on a difficult clinical and imaging diagnosis.

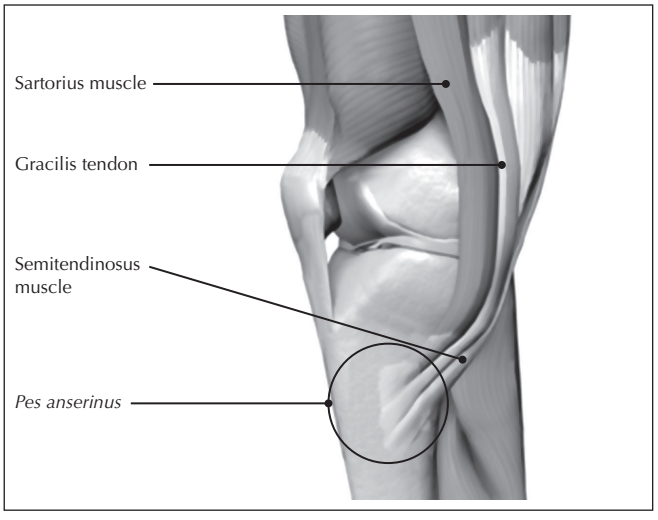

Figure 2
Pes anserinus tendons.
Shows the distance between the insertion of the *pes anserinus* and the knee joint, which should be considered in the physical examination.

edema.[9] Disappearance of the pain after the injection of local anesthetic can contribute for the diagnosis.[10] In the majority of the cases, ultrasound and MRI studies do not confirm the clinical diagnosis.

EPIDEMIOLOGY

This entity inspite of being relatively common is frequently neglected. The exact incidence is unknown. Among 600 consecutive individuals referred to a rheumatology outpatient clinic, 108 received the diagnosis of "soft tissue rheumatism"; among those, 43 had a diagnosis of anserine bursitis.[11]

Studies on the real prevalence and risk factors do not exist. However, reports suggest that anserine bursitis/tendinitis syndrome is more common in overweight females with osteoarthritis of the knees.[1,8,12] It is believed that it is more common in females because women have a wider pelvis, resulting in angulation of the knee, which leads to more pressure in the area of insertion of the *pes anserinus*.

Asian authors evaluated 62 patients with knee osteoarthritis and verefied the diagnosis of anserine bursitis, made by one rheumatologist, in 29 (46%) patients, all females. The authors indicated the frequent association between anserine bursitis and knee osteoarthritis. This study results are debatable as the diagnosis of anserine bursitis was not confirmed by any imaging exam.[13]

Brookler and Morgan[6] reported radiographic changes of osteoarthritis in 20 out of 24 patients with anserine bursitis.

In another study,[11] of 68 patients with presumed osteoarthritis, 41 had anserine bursitis, of whom 38 were females and 37 were overweight.

The presence of fluid in the anserine bursa was documented in 5% of asymptomatic knees.[14] Therefore, the presence of fluid in the anserine bursa in imaging exams does not allow the diagnosis of bursitis.[15]

In a retrospective review of 509 MRIs of the knees of 488 patients of an orthopedic outpatient clinic with suspected "internal derangement", a prevalence of 2.5% of anserine bursitis was observed.[16] The most common complaint was pain in the medial aspect of the knee simulating injury of the medial meniscus. Similar to other studies, the axial image was considered essential to differentiate accumulation of fluid, especially in the semimembranosus bursa and in Baker's cyst; however, other fluid collections, such as meniscal cyst, bone cysts, and bursitis close to the collateral ligament can make the differential diagnosis difficult.[17,18]

Cohen *et al*. observed a 34% prevalence of anserine bursitis in 96 non-insulin dependent diabetics.[19]

When evaluating 48 patients with DM, other authors[20] made a clinical diagnosis of tendinitis or bursitis in 23 (23.9%) knees of 14 (29.1%) symptomatic patients. However, only four (8.3%) diabetic patients had ultrasound changes compatible with tendinitis. The results also suggest that structural changes, such as meniscal lesions, which occur as a consequence of osteoarthritis, can have a role in the origin of pain in the medial compartment of the knee in diabetic patients.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Typical signs and symptoms include pain in the medial aspect of the knee along with pain on palpation and edema of the anatomical site of insertion of the *pes anserinus*. It may be exacerbated when going upstairs or downstairs. However, many patients complain of pain in the posteromedial region or in the midline of the knee, without edema, making the differential diagnosis with meniscal lesion a challenge.

In 1985, Larson and Baum described criteria for the diagnosis of this entity, which include: pain in the anteromedial region of the knee, especially going upstairs or downstairs, morning pain and rigidity for more than one hour, nocturnal pain, and difficulty raising from a chair of getting out of the car, frequently associated with local sensitivity and edema in the area of the anserine bursa.[5]

The incidence of this syndrome is higher in women, overweight individuals, with osteoarthritis of the knees, *valgus* deformities, *pes planus*, and between 50 and 80 years of age, but younger obese women can also be affected.

Diagnosis is imminently clinical and it can be corroborated by imaging exams. History is typical and it is characterized by pain in the proximal medial region of the knee, approximately 5 cm below the medial joint interline of the knee, especially in overweight people with signs of degenerative joint disease. Knee X-rays are usually normal, but can also show bony exostosis or signs of osteoarthritis of the medial compartment. This syndrome is not related to the degree of the degenerative process, *i.e.*, if more advanced, it does not predict the presence of anserine bursitis/tendinitis.

Whether pain and sensitivity to digital pressure are caused by bursitis or tendinitis of the *pes anserinus* or by a possible panniculitis or fasciitis, is unknown, and histopathological studies are necessary.

Few studies systematically assessed the morphological characteristics of the insertion of the *pes anserinus* and respective bursa in patients with the clinical diagnosis of anserine syndrome using imaging exams.

Uson *et al*.[21] undertook an ultrasonographic analysis of 37 female patients with suspected anserine bursitis or tendinitis performed up to 48 hours after the clinical diagnosis. This imaging technique is an excellent way to evaluate superficial soft tissues, such as tendons and bursae. They analyzed: the thickness of the insertion of the *pes anserinus*; intratendinous morphological characteristics; the presence of fluid collection greater than 2 mm in the bursa; and changes in the subcutaneous fat of the medial aspect of the knee, among others. Only one patient had anserine tendinitis. Bursitis was observed in a symptomatic unilateral knee and also in one with bilateral pain, but also in one asymptomatic knee. In the group of six patients with bilateral pain, a significant thickening of the subcutaneous tissue was observed when compared with six asymptomatic controls. A change in the subcutaneous tissue in the medial compartment was not observed. Injury if the medial collateral ligament was observed in four asymptomatic knees. Lesions of the medial meniscus were observed in three symptomatic knees and in one asymptomatic knee. The authors concluded that the majority of patients diagnosed with anserine tendinitis/bursitis does not have morphological changes in the ultrasound and that the etiology of the pain most likely results from a complex interaction between structural changes secondary to osteoarthritis and peripheral and central pain processing mechanisms.[21]

In the same line of research, more recently, other authors[20] have observed ultrasound evidence suggestive of *pes anserinus* tendinitis in only 8.3% of 48 patients with DM type II evaluated with a 10 MHz transducer and with a clinical diagnosis of anserine tendinitis or bursitis, emphasizing that none of the patients showed inflammation of the bursa.

Studies have demonstrated that only a minority of patients with the clinical diagnosis of anserine syndrome have compatible ultrasound changes. This discrepancy can be explained by three mechanisms: 1) the ultrasound might not detect the abnormalities of anserine tendinitis/bursitis. In this case, the MRI might present a contribution. However, since tissues com few mobile protons emit little or no signal, the internal architecture of the tendons of the *pes anserinus* might also not be well demonstrated by this imaging method; 2) the sensitivity to digital pressure of the anserine region might not be originated in the tendons or bursa, but in other tissues that are not well visualized by the ultrasound; and 3) the point of pain sensitivity during deep palpation could be a tender point with atypical pain threshold. Note that 30% of asymptomatic individuals experience pain during deep palpation of this area.

Rennie and Saifuddin[16] observed a prevalence of only 2.5% (13 knees among 488 evaluated by MRI) of anserine bursitis. Disorders that can cause diagnostic confusion on imaging exams included: popliteal cyst, semimembranosus bursitis, and meniscal cyst. Axial incidence was considered primordial to differentiate the anserine bursa from other posteromedial fluid collections. The authors considered that identifying the presence of fluid in the anserine bursa associated with the classical symptoms allow the diagnosis without the need of invasive arthroscopy.[16]

Another study[22] using MRI demonstrated the presence of effusion in the anserine bursa in 3.7% of 451 symptomatic

patients over 45 years of age. Among the 59 patients who had some changes in MRI and underwent clinical evaluation, a correlation between physical and imaging findings was not observed, and they concluded that the majority of patients with a clinical diagnosis of anserine bursitis or tendinitis do not have changes in this imaging exam.

It is believed that the MRI can be useful in the diagnosis of acute anserine bursitis when fluid accumulation associated with synovial proliferation are observed.[23] It can also be useful in the evaluation of undetermined masses in the medial region of the knee.[24]

In a case presentation, axial and coronal T1- and T2-weighted images indicated the presence of a cystic, lobulated lesion adjacent to the posteromedial aspect of the distal femur, superficially between the anserine tendons and the medial collateral ligament, without communication with the knee. The authors considered it a good method to detect and differentiate cystic masses within and around the knee.[25]

Another description that used computed tomography showed a well-defined cystic image of low attenuation immediately below the *pes anserinus*, in a patient with a painless mass in the anteromedial aspect of the knee below the medial joint interline. The administration of IV contrast did not show any highlights. The authors indicated that the distension of the anserine bursa is not synonymous of bursitis, as the patient did not have any symptoms, and the syndrome could be a case of tendinitis or fasciitis affecting the insertion of the *pes anserinus*.[26]

Analysis of the fluid, although rarely documented in the literature, showed mononuclear cells or the absence of inflammatory cells and absence of crystals.[26,27]

Summarizing, the literature shows that ultrasound, CT, and MRI findings usually do not allow the identification of which structure is responsible for the symptoms in anserine tendinitis/bursitis syndrome.

DIFFERENTIAL DIAGNOSIS

Several possibilities should be considered in the differential diagnosis, especially lesions in the medial meniscus, osteoarthritis of the medial compartment of the knee, L3-L4 radiculopathy, and lesions in the medial collateral ligament.

Pain and sensitivity are present in the medial compartment in medial meniscus lesion and osteoarthritis, while in the anserine syndrome they are located inferomedial to the medial joint interline. In some situations, different disorders coexist. Larsson and Baum observed that several patients referred to a tertiary rheumatology outpatient clinic with articular degenerative disease and knee pain also had anserine syndrome.[5]

Knee pain secondary to L3-L4 radiculopathy is associated with lumbar pain without pain on digital pressure of the anserine region. Stress maneuvers of the medial collateral ligament, with or without instability, contribute for the diagnosis of lesions of this ligament.

Cystic periarticular knee lesions include physiological increase of the bursa and recesses, pathological cysts (meniscal and ganglion cysts), as well as benign and malignant soft tissue masses that simulate cysts. These lesions can result in symptom overlap or suggest internal knee derangement. Some cystic lesions can present diagnostic difficulties.

Bursae inflammation should always be included in the differential diagnosis. Suprapatellar bursitis is one of them. This bursa is a midline structure located between the pre-femoral and suprapatellar fat pad. It is better visualized in sagittal planes of the MRI. Unlike other bursae located around the knee, it usually communicates with the joint, except when the suprapatellar plica, which corresponds to a normal embryonic septum, does not undergo degeneration as it should. Lipo-hemarthrosis may develop in the bursa as a consequence of an intra-articular fracture, both of the femur and patella, resulting in the formation of layers of fat from the bone marrow over a layer of blood. Free bodies originated in the joint can migrate into the bursa.

Pre-patellar bursitis, located between the patella and the skin, is commonly secondary to repetitive trauma, such as that observed in people who kneel frequently. Gout and infections represent other possible causes. Similar to the suprapatellar bursa, it is also better analyzed in sagittal views of the MRI.

Superficial infra-patellar bursitis, located between the anterior aspect of the distal pole of the patellar tendon and the subcutaneous fat, is less common, but it should be considered. Direct trauma can cause hemorrhagic bursitis. Fluid in this bursa can be associated with Osgood-Schlatter disease.

Deep infra-patellar bursitis, located between the posterior margin of the distal patellar tendon and the proximal tibia, is another possibility. The presence of a small amount of fluid can be a physiological finding and cause diagnostic confusion.[28] This bursitis is usually a consequence of overload of the extensor tendon of the knee, especially in jumpers and long-distance runners.

Semimembranosus bursitis, also called semimembranosus-tibial collateral ligament bursitis, is located within the superficial and deep layers of the medial collateral ligament, and involves the anterosuperior margin of the semimembranosus tendon.

Pain affects the midline of the knee. In sagittal and coronal planes, the fluid collection can be seen in the posteromedial portion of the articular line.

Iliotibial bursitis, which is located between the distal portion of the iliotibial band (near its insertion in the Gerdy tuberculum) and the adjacent tibial surface, can be associated with iliotibial tendinitis and it is usually due to overload and varus stress. It can cause pain in the anterolateral region of the knee and simulate lesion of the lateral meniscus. The MRI can show distension of the bursa close the insertion of the iliotibial band. An increase in T2-weighted signal within the iliotibial band allows the concomitant diagnosis of iliotibial bursitis.

Cysts within the knee are part of the differential diagnosis. Baker's cyst, also known as popliteal cyst or gastrocnemius-semimembranosus recess, is aligned with the synovia and extends between the medial head of the gastrocnemius and semimembranosus muscles. Its peculiar anatomical position helps to differentiate it from other periarticular cystic lesions of the knee. Approximately 5% to 32% of patients with pain in the knee might have a Baker's cyst, two distinct age groups are observed in a bimodal distribution model: between 4 and 7 years and between 35 and 70 years.[29] In the elderly, they are usually associated with some articular abnormality (chronic articular effusion secondary to inflammatory articular disease, osteoarthritis, internal derangement, deposits of crystals, and etc.). Larger cysts are seen in rheumatoid arthritis, with a prevalence of 47.5%, which might not be detected at the physical examination.[30] They might be asymptomatic or, when large, they might cause compression of the popliteal vein, causing deep venous thrombosis. Ruptured Baker's cyst is a potential complication, being more common in the distal portion. Acute rupture can simulate deep venous thrombosis.

Meniscal cyst, also known as cyst of the semilunar fibrocartilage, originates from parameniscal soft tissue and results from extrusion of fluid from a meniscal fissure/laceration. The patient presents with pain, blocked movement, and a palpable mass in the articular line. A study showed that the cyst is twice more common in the medial than in the lateral compartment,[31] is commonly adjacent to the posterior horn and tends to be larger and to dissect. It has a tendency to reoccur after the surgery (excision and aspiration), except if the meniscal lesion is repaired.

The intra-articular synovial cyst (ganglion) can originate in the articular capsule, a ligament, a synovial tendon sheath, or bursa. One study demonstrated that this cyst more commonly originates from the cruciate ligament, and in 61% of the cases in the intercondylar notch.[32] On MRI, the ganglion cyst is seen as an oval, septate fluid collection by the cruciate ligament, and it can be differentiated from a meniscal cyst by the absence of meniscal fissure/laceration and also by its location. Erosion of the femoral condyle or dissection along the plane of the tissue involved in the synovial cyst can be observed.

Synovial osteochondromatosis, which is characterized by cartilaginous bodies originated within a metaplastic synovial tissue in a bursa of articulation, is among the lesions that simulate cysts. It can present with arthralgia, palpable mass, and restriction of movement. Free bodies can undergo calcification and ossification. In those cases, the diagnosis can be made by conventional X-rays. Magnetic resonance imaging can be useful in the earlier phases, before the development of calcification or ossification, observing an intermediate pattern of cartilaginous bodies, in T1-weighted images, and increased signal, in T2. Fatty marrow inside ossified bodies produces a central area of high signal in T1-weighted images.

Malignant tumors that are detected more commonly adjacent to the knee include fibrous histiocytoma, liposarcoma, and synovial sarcoma. They might contain areas of necrosis or myxoid degeneration and, therefore, possibly simulate a cyst. Those tumors typically have heterogeneous soft tissue components that can be highlighted with the administration of IV contrast. The margins of those tumors can be irregular and indicate the presence of infiltration of adjacent tissues.

Deposits of hemosiderin can be seen in the hypertrophied synovial tissue in pigmented villonodular synovitis, which, in T1- and T2-weighted images show low signal, besides demonstrating the exuberance in echo-gradient sequences due to the susceptibility of paramagnetic blood products.

Deposits of sodium monourate crystal (gout) or amyloid also show low signal in T1- and T2-weighted images, but, unlike pigmented villonodular synovitis, it does not show changes in echo-gradient sequences. Therefore, MRI can have an important role in those situations.

Varicose popliteal vein, popliteal aneurysm, and secular dilation of the popliteal vein can occasionally be the result of trauma. Magnetic resonance images show characteristics of flow and blood products, in addition to identifying the connection with the popliteal blood vessel, allowing a precise diagnosis. Complications include thrombosis, embolism, or rupture. Aneurysm of the popliteal artery can also simulate a cyst in the popliteal fossa. The characteristics on MRI include pulsatile artifacts and thrombus with distinct layers.

A case of anserine bursitis induced by polyethylene after knee arthroplasty has been reported. Initially, it was considered an infectious complication of the surgical procedure. Therefore, this is another possibility of pain and inflammation to be considered in patients who receive prosthesis.[7]

The saphenous nerve can be compressed by an anserine bursitis and cause pain the lateral tibial region and paresthesia, besides simulating a stress fracture.[33]

Spontaneous osteonecrosis of the knee can affect the medial tibial plateau and, in some cases, can cause a difficult diagnosis, but X-rays and scintigraphy, among other methods, can contribute to elucidate the case.[34]

Other conditions that can be considered in the differential diagnosis include: muscle pain, patellofemoral syndrome, patellar chondromalacia, recurring patellar subluxation, Osgood-Schlatter disease, dissecting osteochondritis, patellar tendinitis, synovial plica, lesion of the infra-patellar fat pad, patellar dysplasia, bi- or multipartite patella, patellar fracture, para-articular chondroma or osteochondroma, synovitis, and synovial hemangioma. Frequently, pain in the anserine region is part of the fibromyalgia syndrome.

Imaging exams can contribute to the differential diagnosis, such as, an X-ray assessment showing osteoarthritis of the knee or patellar derangements, as well as an MRI showing patellar chondromalacia, or meniscal or ligament lesions. A careful physical examination can contribute to elucidate of the diagnosis. Beside inspection, static and walking, semiological tests, alignment, hypermobility, and muscular function should also be evaluated.

TREATMENT

The initial treatment should include resting the affected knee, cryotherapy (cold packs for 10 minutes) for acute cases, physiotherapy, and antinflammatory drugs.. The use of a pillow between the thighs at nighttime can be necessary. Weight loss is mandatory in obese patients. Treatment of occasional associated conditions, such as deviated knee and *pes planus*, and control of diabetes should not be forgotten. Elderly patients and those with chronic pain should be advised to avoid muscular atrophy secondary to disuse. Isometric exercises can be used to this end.

Injection of local anesthetic associated with corticosteroids in the bursa is an option in cases of proven bursitis; 20 to 40 mg of methylprednisolone can be injected[6.35.36]. Triamcinolone, 20 to 40 mg, or betamethasone, 6 mg, can also be used[37]. Special care should be taken to avoid injecting the substance in the tendons of the *pes anserinus*. No more than three infiltrations should be done over a one-year period. The time between infiltrations should be greater than one month. Patients who do not show any response to an initial infiltration rarely respond to repeated infiltrations. One injection in the knee joint can be beneficial in refractory cases.[37]

One study included 44 consecutive patients with anserine bursitis treated with 500 mg of naproxen every 12 hours or corticosteroid injection. In the evaluation, only a verbal pain scale was used and patients were followed for only one month. A significant improvement was reported in 58% and resolution in 5% in the naproxen group *vs.* 70% of significant improvement and 30% resolution in the corticosteroid group ($p \leq 0.05$).[38]

In a retrospective study with 29 patients, clinical remission was observed in 11 of 12 patients who were treated with injectable corticosteroids, compared to 7 of 17 who did not receive infiltration with corticosteroids and anesthetics.[39]

Twenty-six patients clinically diagnosed with osteoarthritis of the knees and anserine syndrome, were evaluated by Yoon and Kim[40]. Ultrasound was also used to confirm the diagnosis. Triamcinolone acetonide was injected in the bursa of 17 patients. The therapeutic response was evaluated by a visual analogue scale (VAS), Western Ontario and MacMaster Osteoarthritis Index (WOMAC), and by the global evaluation by the patient and the investigator using the Likert scale. Only two patients (8.7%) demonstrated ultrasound evidence of anserine tendinitis/bursitis. The parameters of the VAS, pain index, and functional capacity of the WOMAC showed statistically significant improvement after the injection of corticosteroids. Global evaluation by the patients revealed that two considered their response excellent; six, good; one, moderate; eight, absence of improvement; and no patient declared worsening. The authors indicated that the two patients who referred excellent response were the only ones with ultrasound evidence of anserine tendinitis/bursitis. The study concluded that the ultrasound is a useful diagnostic tool to guide the treatment of anserine syndrome in patients with osteoarthritis of the knee.[40] The same authors believe that bursitis is more frequent and responds more rapidly to treatment than anserine tendinitis.

In the study by Larsson and Baum, 76% of the patients with a diagnosis of anserine bursitis had had symptoms for more than one year. They noticed immediate relief of pain after the injection. Approximately 70% of injected knees showed significant improvement. An initial difference between the isolate administration of lidocaine and the combination of lidocaine and long-acting corticosteroid was not observed. However, after one month, significant improvement was observed in patients who received corticosteroid.[5]

Therefore, studies have demonstrated that infiltration with corticosteroids can be a good option in well-documented and well-selected patients. The risk of complications include atrophy of the subcutaneous tissue, skin depigmentation, and

rupture of the tendon.[37,41] Patients should be warned about discomfort or pain after the injection, which is seen in 30% of the patients, and 10% are at risk of developing a reaction to the corticosteroid. These side effects can be managed with local ice therapy and analgesics.[42]

Physiotherapy does have a role in the treatment of this disorder. Ultrasound has been documented as effective in the reduction of the inflammatory process in anserine syndrome.[6] Transcutaneous electrical stimulation (TENS) has been used in other types of bursitis; however, it has not been documented specifically in this syndrome. Rehabilitation exercises should follow physiotherapeutic principles for derangements of the knees (stretching and strengthening of adductors and quadriceps, especially in the last 30° of knee extension, using the vastus medialis muscle, besides stretching the tendons that comprise the *pes anserinus*). For those cases secondary to restricted flexibility and muscles/tendons retraction, stretching can promote important reduction in the tension on the anserine bursa.

Surgery is indicated in case of failure of the conservative treatment. A simple incision followed by drainage of the distended bursa can provide improvement of symptoms.[5,6,35,43] However, a case in which occurred the removal of the bursa, due to the large size of the lesion, has been reported.[7] In case of bone exostosis, it should be removed.

CONCLUSIONS

Pain in the medial compartment of the knee is a common occurrence. Many patients with inferomedial pain receive the diagnosis of *pes anserinus* tendinitis or anserine bursitis or both conditions based only on the presence of pain on palpation of the anatomic site. However, when submitted to imaging exams, the majority of these patients do not show evidence of an inflammatory process. The deranged structure is not identified, as well as an alternative lesion, to justify the pain of many patients. It has been suggested that pain in these patients is secondary to or associated with a panniculitis of the fat tissue of the medial compartment, despite the scarcity of data in the literature.[44] Due to the anatomical intimacy between the tendons of the *pes anserinus* and the medial collateral ligament,[45] it has also been suggested that the pain in these cases could be due to ligament damage.[21]

On the other hand, changes in imaging exams compatible with anserine bursitis have been observed in asymptomatic individuals.

Diabetes mellitus seems to be a predisposing factor, and the influence of overweight, damage to the medial meniscus, or osteoarthritis, as well as other incriminating factors, is yet to be confirmed. In the majority of patients with the clinical diagnosis of anserine tendinitis/bursitis, the origin of the symptoms would, most likely, result from a complex interaction between structural changes and peripheral and central pain modulation mechanisms.

As this perplexing condition remains to be better understood, epidemiologic, etiologic, and pathophysiological studies are necessary for a better understanding and, consequently, to guarantee adequate therapy.

For all the reasons exposed here, we suggest that the term "anserine syndrome" would be more appropriate and it should be used for these patients.

Its differential diagnosis is very encompassing and it was discussed here to offer subsidies to facilitate the clinical approach of pain, especially in the medial compartment of the knee.

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