Skeletal Radiol
DOI 10.1007/s00256-013-1582-y

CASE REPORT

Adhesive capsulitis of the knee

Marcelo Rodrigues de Abreu · Mateus Falcão · Clarice Sprinz · Roque Furian · Luis Roberto Marczyk

Received: 4 July 2012 / Revised: 7 January 2013 / Accepted: 13 January 2013

Abstract The authors describe the case of a 42-year-old woman presenting with significant knee pain and disability. Her imaging findings using contrast MR imaging and FDG PET/CT suggested adhesive capsulitis, which was confirmed by arthroscopy, histology, and the clinical outcome.

Keywords Adhesive capsulitis · Frozen knee · Capsular · Inflammation · Synovial · Fibrosis

Introduction

Adhesive capsulitis (AC) is a common disorder. It usually affects the shoulder and causes chronic pain that can last for several months or for a period of years. AC can be primary (unknown etiology), or secondary to trauma, surgery or immobilization. Imaging findings of adhesive capsulitis have been previously described. Conventional arthrography demonstrates reduction of the joint volume and obliteration of the axillary fold [1]. Magnetic resonance (MR) imaging may show thickening and signal alteration of the joint capsule and surrounding tissues with or without gadolinium enhancement [2, 3]. Adhesive capsulitis has also been reported in the hip, wrist and ankle joints [4, 5].

M. R. de Abreu (✉) · M. Falcão · C. Sprinz · L. R. Marczyk
Hospital Mãe de Deus and Mãe de Deus Center, Rua José de Alencar 286,
Porto Alegre, RS, Brazil
e-mail: marcelorad@hotmail.com

R. Furian
FFCMPA, Porto Alegre, Brazil

L. R. Marczyk
Hospital de Clínicas, Universidade Federal do Rio Grande do Sul (UFRGS), Rua Ramiro Barcelos 2350,
Porto Alegre, RS, Brazil

Published online: 23 February 2013

To our knowledge, adhesive capsulitis of the knee has not been reported in the literature. This report describes a patient with spontaneous and progressive chronic incapacitating knee pain and restricted movement. Imaging findings were compatible with joint capsule and synovial disease, suggesting a diagnosis of adhesive capsulitis, which was later supported by arthroscopy, histological examination, and clinical outcome.

Case report

A 42-year-old woman presented at the orthopedic clinic with moderate/severe knee pain on 21 September 2010. The symptoms were initially mild and progressed to moderate/severe over the course of 3 months. Upon clinical examination, no effusion or inflammatory signs were found. She had limitations of both flexion (90°) and extension (85°) of the knee, (normal knee range of motion: flexion 130°, extension 120°). Laboratory examinations for a systemic rheumatological condition were negative (FAN, rheumatoid factor, HLA-B27). Radiographs of both knees were normal. The patient was treated initially with oral anti-inflammatory medication and underwent 40 sessions of physiotherapy; no significant improvement was observed.

Eight months later the patient was re-evaluated and continued to report moderate/severe knee pain. Magnetic resonance imaging was performed using a low-field magnet (0.3 T Magnetom C, Siemens, Erlangen, Germany). STIR sequences (TR/TE 3,500–120) revealed high signal intensity in the suprapatellar fat pad, the anterior cruciate ligament, and the popliteal tendon insertion into the lateral condyle. No knee effusion was observed; in fact, the amount of joint fluid was thought to be reduced. No menisci, cartilage or ligament injuries were found.

Six weeks later, another set of MR images were obtained using a high field strength (1.5 T, Magnetom Spree; Siemens, Erlangen, Germany) with gadolinium contrast agent administration (14 ml). High signal intensity on T2-weighted images (TSE; TR/TE: 3,000/100) and contrast enhancement were noted in the same regions that had been abnormal in the previous examination (the suprapatellar fat pad, the anterior cruciate ligament, and the popliteal tendon insertion) and also at the posteromedial capsule. No menisci, cartilage or ligament injuries were found. Additionally, T2 mapping of the patellar cartilage was performed, with normal findings. The radiologist suggested the possibility of adhesive capsulitis of the knee (Fig. 1), and recommended a complementary study with PET/CT for confirmation.

Fluorodeoxyglucose (FDG) PET/CT (with 1-h acquisition delay after intravenous FDG injection), revealed significant enhancement of the suprapatellar fat pad, the anterior cruciate ligament, the posterior cruciate ligament, the popliteus insertion, the posterior capsule and the semimembranosus insertion. The other knee, which was imaged at the same time, was normal (Fig. 2).

Because symptoms persisted, arthroscopy was recommended. Approximately 1 year after she was first seen, arthroscopy revealed inflammatory signs at the suprapatellar synovial membrane (synovitis), with a red coloration and limited distension. A soft tissue sample from this area was submitted for histopathological evaluation. No other joint abnormalities were found at arthroscopy.

Biopsy using hematoxylin–eosin staining revealed synovial hyperplasia (synovial membrane with multiple layers of cells) with synovial papillomatosis, mild capsular and stromal fibrosis (fibroblast infiltration between collagen fibers), and stromal chronic focal inflammatory infiltrate (lymphocyte and histiocyte conglomerations), findings with high specificity value for adhesive capsulitis (Fig. 3).

One month after the arthroscopy, the patient reported that her symptoms were improving. By 24 months after the initial symptoms, she was asymptomatic.

Discussion

Adhesive capsulitis has an incidence of 3–5 % in the general population and up to 20 % in those with diabetes [6]. Adhesive capsulitis of the shoulder is one of the most common musculoskeletal problems encountered in orthopedics [6]. Although the pathophysiology of AC remains unclear, this disease is thought to be caused by a combination of inflammation and a fibrotic reaction in the capsule and synovial membrane that subsequently leads to the formation of adhesions [7–11].

The presentation of adhesive capsulitis generally comprises three distinct stages [6]. The first stage is called the

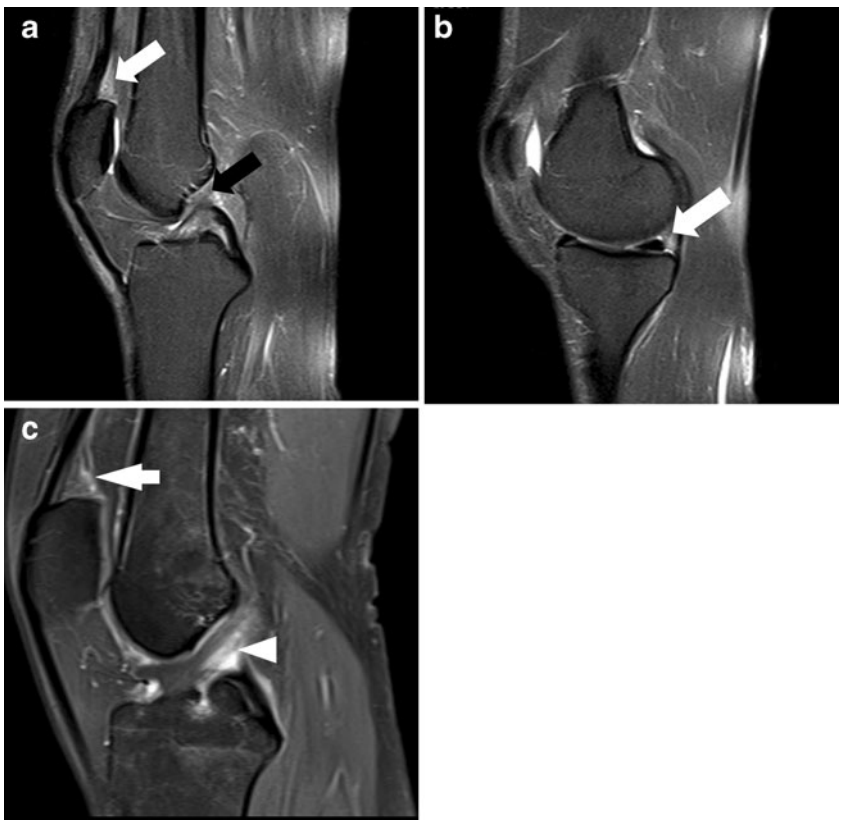

Fig. 1 Second MR imaging examination performed using a high-field 1.5-T magnet. **a** Sagittal T2-weighted FSE sequence (TR 3000/TE 100) with fat suppression demonstrating a high signal intensity at the suprapatellar fat pad (*white arrow*) and at the anterior cruciate ligament (*black arrow*). **b** Sagittal T2-weighted FSE sequence (TR 3000/TE 100) with fat suppression demonstrating a high signal intensity at the posteromedial capsule zone (*white arrow*). **c** Sagittal T1-weighted sequence (TR 500/TE 13) with fat suppression with contrast agent demonstrating enhancement of the suprapatellar fat pad (*white arrow*) and the pericruciate ligament zone (*white arrowhead*)

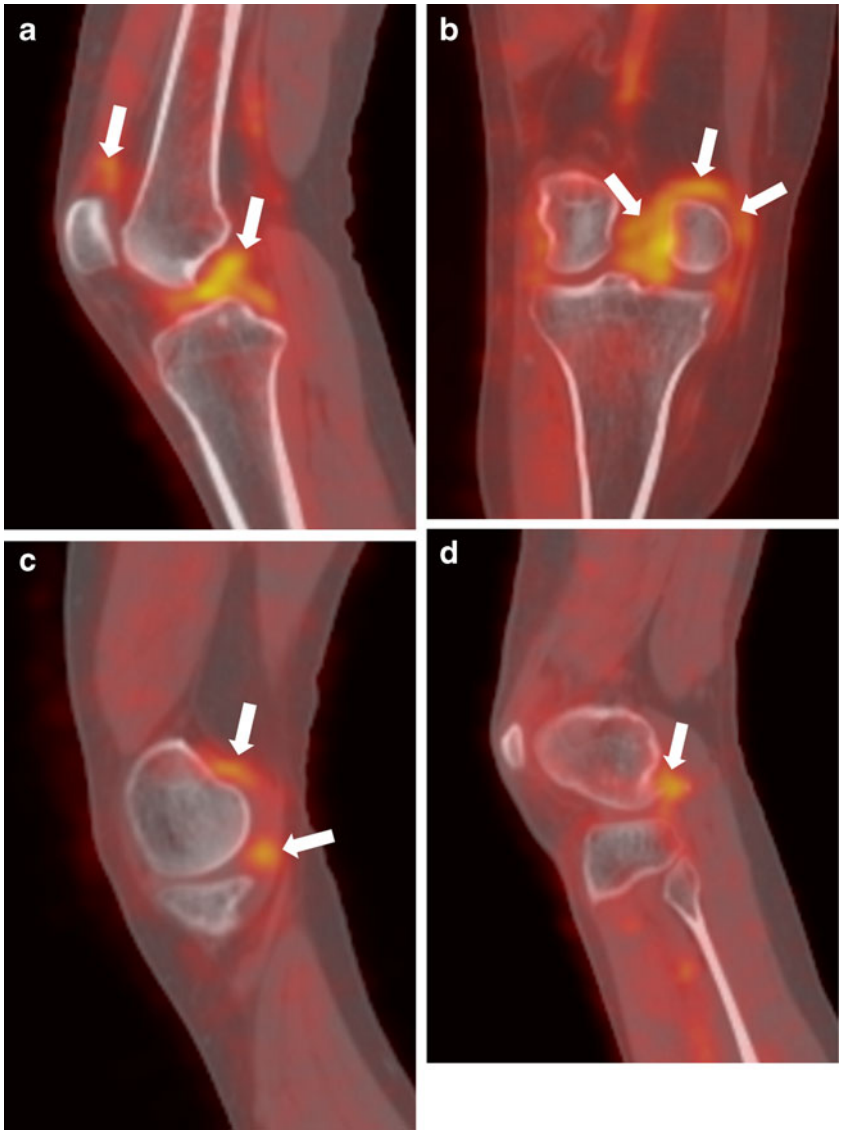

Fig. 2 Fluorodeoxyglucose (FDG) PET/CT (with 1-h delayed acquisition) of the knee with multiplanar reconstruction demonstrating enhancement (*white arrows*) of **a** the cruciate ligaments and suprapatellar fat pad, **b**, **c** the articular capsule, and **d** the popliteal tendon insertion

"freezing" or "painful" stage. Patients may not present during this stage because they think that eventually the pain will resolve if self-treated. As the symptoms progress, pain worsens and both the active and the passive range of motion becomes more restricted, eventually resulting in the patient seeking medical consultation. This stage typically lasts between 3 and 9 months and is characterized by acute synovitis [12]. Most patients will progress to the second stage, the "frozen" or "transitional" stage. Because of pain at the end of the range of motion (ROM), use of the limb may be limited. causing muscular disuse. The common capsular pattern of limitation has historically been described as diminishing motions. with external joint rotation being the most limited, followed closely by joint flexion and internal rotation. There eventually becomes a point during the frozen stage when the pain does not occur at the end of ROM. During this stage (which lasts anywhere from 4 to 12 months), shoulder pain does not necessarily worsen. The third stage begins when the range of motion begins to improve; this stage (lasts anywhere from 12 to 42 months) is termed the "thawing" stage and is defined by a gradual return of joint mobility. The treatment options for AC have included, but are not limited to, physical therapy, corticosteroid injections, closed manipulations, and capsular release [10–13].

Analyzing our presented case retrospectively, the chronology of symptoms was similar to that described above, and the duration of the disease was approximately 24 months. In the classic description of AC, pain presents first, followed by progressive loss of motion; the pain then subsides, and motion may or may not be slowly restored. Symptoms of AC may progress so slowly that the patient does not even seek medical attention until range of motion and pain severely limit their daily activities. It is not uncommon for a patient to present with pain as their only complaint and not realize that there is a loss of motion [6].

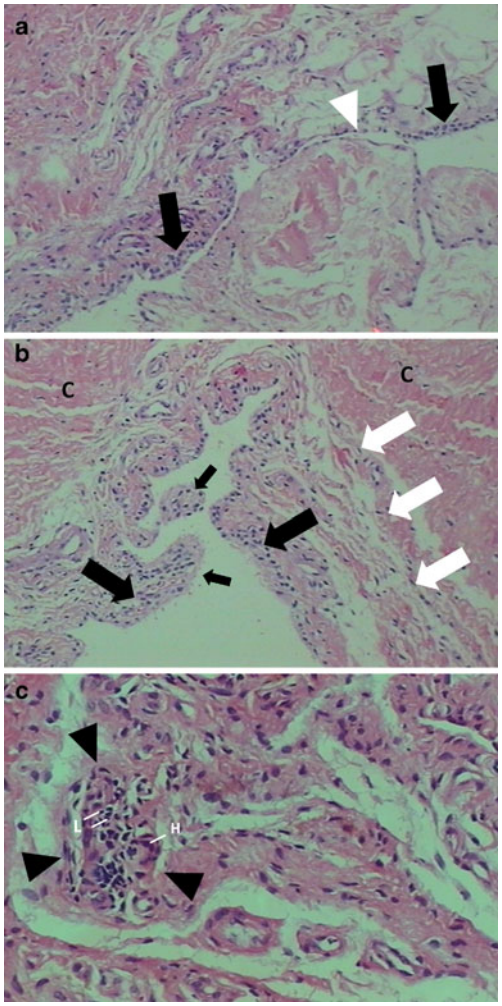

Fig. 3 Histology of the soft tissue sample removed by arthroscopy at the suprapatellar pouch/suprapatellar fat pad. Hematoxylin/eosin staining demonstrated synovial hyperplasia (synovial membrane with multiple layers of cells; *black arrows*). **a** Synovial hyperplasia (*black arrow*), synovial papillomatosis (*small black arrows*), and mild stromal fibrosis (*white arrows*). **b** Stromal chronic focal inflammatory infiltrate with lymphocyte and histiocyte conglomerates (*black arrowheads*). **c** Normal synovial membrane with a single layer of cells (with *arrowheads*). *L* lymphocytes, *H* histiocytes, *C* joint capsule

In the knee, the loss of motion is probably more difficult to assess because of its large synovial recesses compared with those in the shoulder joint.

Prolonged immobilization of a joint after injury or surgery has been shown to have several detrimental physiological consequences, including the following: decreased collagen length, fibrofatty infiltration into the capsular recess, ligament atrophy resulting in decreased stress absorption, collagen band bridging across recesses, random collagen production, and altered sarcomere number in the muscle tissue, consequences that should disappear after recovery time [14]. The development of AC after trauma or surgery (secondary AC) could represent an exacerbation of this physiological consequences following immobilization. In primary AC immobilization (joint restriction) is probably due to primary contracture (fibrosis).

The histological analysis of AC usually reveals the following: hypertrophic synovitis, hypervascular synovitis, and rare inflammatory cell infiltrates during phase 1; hypertrophic and hypervascular synovitis with perivascular scar, subsynovial scar, fibroplasias, and scar formation in the underlying capsule during phase 2; and dense scar formation of the capsule during phase 3 [15]. In our case, the histological findings were compatible with phase 1–2 AC, showing synovial hyperplasia, mild capsular fibrosis (scar), stromal fibrosis (scar), and chronic focal inflammatory infiltrate.

Magnetic resonance imaging can be useful at all stages of adhesive capsulitis, including stage 1, during which the clinical examination findings may be subtle [14]. Early research in a small study population showed that joint capsule measurements that are greater than 4 mm in the axillary recess on T1 oblique coronal MR images suggest a diagnosis of adhesive capsulitis with reasonable sensitivity and excellent specificity [1]. Several MR imaging studies have demonstrated the high prevalence of gadolinium enhancement of the joint capsule and synovial membrane in stage 2 disease [1, 3, 7, 16].

A recent study published by Ahn et al. [16] demonstrated that the pain intensity was linked to the contrast enhancement grades of the joint capsule. Our case showed hyperintensity of the capsule and synovial membrane on T2-weighted images, as well as contrast enhancement, suggesting that it represented stage 2 disease, which was compatible with the clinical examination of the patient. The knee capsule and synovial enhancement by gadolinium was corroborated by avid FDG consumption on PET/CT, suggesting a hypermetabolic state of the periarticular tissues. Positron emission tomography with computed tomography (PET/CT) with [18F] fluorodeoxyglucose (FDG) can be used to evaluate inflammatory joint disease by visualizing the glucose consumption at the inflamed areas [17]. However, we believe that our case represents the first report of this imaging method in AC. PET/CT imaging with FDG is not routinely indicated for the diagnosis of AC, but provided useful confirmation of synovial and joint capsule abnormalities. The degree of enhancement with FDG PET/CT was quite marked, and disproportionately more prominent than seen on the comparable post-gadolinium MR imaging examination. This is probably due to increased contrast resolution inherent to each method, considering the background tissue signal and enhancement with contrast compounds.

Abnormal rotator interval fat signal has been described in the literature as a manifestation of AC of the shoulder [1, 3, 7, 16]. In our case there was abnormal MR signal and

contrast enhancement in the suprapatellar fat pad. Isolated signal alteration of the suprapatellar fat pad has been described by Schweitzer et al. [18] as a condition that is analogous to Hoffa's disease, an inflammatory process of the fat pad. Histological examination of Hoffa's disease reveals fibrous tissue, leukocytes, perivascular cells, and fibrin-occluded blood vessels [19]. These findings are thought to cause pain at the infrapatellar fat pad and result in signal alterations on MR imaging [20]. The authors suggested that these periarticular fat pads are often a window to articular disorders or synovial abnormalities because of their anatomical positions. In one patient from the study who underwent an imaging-guided biopsy of the suprapatellar fat pad, histological analysis revealed vasculitis with obliteration of the blood vessels and an inflammatory response, which is very similar to the histological changes described in Hoffa's disease. It is possible that isolated signal alteration on the suprapatellar fat pad could represent the beginning of AC of the knee, or a "forme fruste" of the disease.

A similar condition of the knee was described by Paulo et al. in patients after knee surgery or injury. They named it infrapatellar contracture syndrome (IPCS) [21, 22]. Patients with IPCS present with anterior knee pain, decreased patellar mobility, a decreased range of motion and quadriceps atrophy following knee surgery [23]. The diagnosis is purely clinical and not obvious unless the problem is sought. A comparison of patellar excursion relative to the contralateral knee is critical for diagnosing this condition. Occasionally, there is a significant loss of the range of motion, but for the majority of the patients, the main complaints are anterior knee pain and difficulty with rehabilitation. Arthroscopic release results in significant improvement in the range of motion [23, 24].

We think that in our case, the absence of a meniscal tear, or any other findings of internal derangement, argue strongly against other diagnoses to explain the patient's symptoms. In addition, no joint effusion was observed, which is not compatible with pure synovitis because joint effusion is usually abundant. Although isolated signal alteration of the anterior cruciate without discontinuity can be found in patients with partial ligament tear, mucoid degeneration or gout [25, 26], in our case, there was no clinical evidence of these conditions, and the appearance on MRI was not typical of mucoid degeneration. Therefore, we believe that the diagnosis that best fits the patient's condition is adhesive capsulitis of the knee.

Adhesive capsulitis is accepted to occur in joints other than the shoulder. AC of the wrist can be found in patients who have undergone prolonged wrist immobilization [5]. AC of the hip has been reported in the literature [4] with clinical characteristics similar to the shoulder, principally consisting of painful restricted motion and a clear predilection for middle-aged women. This report is the first description of AC of the knee. Similar to AC of the hip [4], this condition is possibly underdiagnosed. One of the reasons for this may be that the very large synovial recesses of the knee may undergo contraction with little or no loss of motion, which is the main physical examination finding in AC.

In summary, we presented the case of a middle-aged woman with incapacitating chronic knee pain that persisted for 24 months and ultimately underwent spontaneous and complete resolution. Imaging findings on MR and FDG PET/CT were compatible with capsule and synovial disease, but there was no joint effusion, and no clinical or laboratory findings of rheumatological disease. Arthroscopy revealed capsular joint restriction and synovitis. Biopsy showed synovial hyperplasia, mild capsular fibrosis, and chronic focal inflammatory infiltrate. All of those findings, together with the exclusion of other pathology, suggest the diagnosis of adhesive capsulitis of the knee.

References

1. Connell D, Padmanabhan R, Buchbinder R. Adhesive capsulitis: role of MR imaging in differential diagnosis. Eur Radiol. 2002;12:2100–6.
2. Emig EW, Schweitzer ME, Karasick D, Lubowitz J. Adhesive capsulitis of the shoulder: MR diagnosis. AJR Am J Roentgenol. 1995;164:1457–9.
3. Tamai K, Yamato M. Abnormal synovium in the frozen shoulder: a preliminary report with dynamic magnetic resonance imaging. J Shoulder Elbow Surg. 1997;6:534–43.
4. Byrd JW, Jones KS. Adhesive capsulitis of the hip. Arthroscopy. 2006;22(1):89–94.
5. Brandser EA, Renfrew DL, Schenck RR. Adhesive capsulitis of the wrist. Can Assoc Radiol J. 1995;46(2):137–8.
6. Manske RC, Prohaska D. Diagnosis and management of adhesive capsulitis. Curr Rev Musculoskelet Med. 2008;1:180–9.
7. Carrillon Y, Noel E, Fantino O, Perrin-Fayolle O, Tran-Minh VA. Magnetic resonance imaging findings in idiopathic adhesive capsulitis of the shoulder. Rev Rheum Engl Ed. 1999;66:201–6.
8. Bunker TD, Anthony PP. The pathology of frozen shoulder. A Dupuytren-like disease. J Bone Joint Surg Br. 1995;77:677–83.
9. Neviaser JS. Adhesive capsulitis of the shoulder: a study of the pathological findings in periarthritis of the shoulder. J Bone Joint Surg Am. 1945;27:211–22.
10. Lundberg BJ. The frozen shoulder. Clinical and radiographical observations. The effect of manipulation under general anesthesia. Structure and glycosaminoglycan content of the joint capsule. Local bone metabolism. Acta Orthop Scand Suppl. 1969;119:1–59.
11. Rodeo SA, Hannafin JA, Tom J, Warren RF, Wickiewicz TL. Immunolocalization of cytokines and their receptors in adhesive capsulitis of the shoulder. J Orthop Res. 1997;15:427–36.
12. Harryman DT, Lazurus MD, Rozencwaig R. The stiff shoulder. In: Rockwood Cam Matsen FA, Wirth MA, Lippitt SB, editors. The shoulder. Philadelphia: Saunders; 2004.
13. Grey RG. The natural history of "idiopathic" frozen shoulder. J Bone Joint Surg Am. 1978;60:564.

14. Sofka CM, Ciavarra GA, Hannafin JA, Cordasco FA, Potter HG. Magnetic resonance imaging of adhesive capsulitis: correlation with clinical staging. HSS J. 2008;4:164–9.
15. Mangine RE, Heckmann T, Eifert-Mangine M. Alternative techniques for the motion-restricted shoulder. In: Andrews J, Wilk K, editors. The athletes shoulder. New York: Churchill Livingstone; 1994.
16. Ahn K-S, Kang CH, Oh Y-W, Jeong W-K. Correlation between magnetic resonance imaging and clinical impairment in patients with adhesive capsulitis. Skeletal Radiol. 2012;41:1301–8.
17. Takata T, Taniguchi Y, Ohnishi T, Kohsaki S, Nogami M, Nakajima H, et al. (18)FDG PET/CT is a powerful tool for detecting subclinical arthritis in patients with psoriaticarthritis and/or psoriasis vulgaris. J Dermatol Sci. 2011;64(2):144–7.
18. Schweitzer ME, Falk A, Pathria M, Brahme S, Hodler J, Resnick D. MR imaging of the knee: can changes in the intracapsular fat pads be used as a sign of synovial proliferation in the presence of an effusion? AJR Am J Roentgenol. 1993;160:823–6.
19. Hoffa A. Influence of adipose tissue with regard to the pathology of the knee joint. JAMA. 1904;43:795–6.
20. Jacobson JA, Lenchik L, Ruhoy MK, Schweitzer ME, Resnick D. MR imaging of the infrapatellar fat pad of Hoffa. Radiographics. 1997;17:675–91.
21. Paulos LE, Wnorowski DC, Greenwald AE. Infrapatellar contracture syndrome: diagnosis, treatment and long-term follow-up. Am J Sports Med. 1994;22(4):440–9.
22. Paulos LE, Rosenberg TD, Drawbert J, et al. Infrapatellar contracture syndrome: an unrecognized cause of knee stiffness with patella entrapment and patella infera. Am J Sports Med. 1987;15:331–41.
23. Richmond JC, al Assal M. Arthroscopic management of arthrofibrosis of the knee, including infrapatellar contraction syndrome. Arthroscopy. 1991;7(2):144–7.
24. Vaquero J, Vidal C, Medina E, Baena J. Arthroscopic lysis in knee arthrofibrosis. Arthrosc J Arthrosc Relat Surg. 1993;9(6):691–4.
25. McIntyre J, Moelleken S, Tirman P. Mucoid degeneration of the anterior cruciate ligament mistaken for ligamentous tears. Skeletal Radiol. 2001;30(6):312–5.
26. Melloni P, Valls R, Yuguero M, Sáez A. An unusual case of tophaceous gout involving the anterior cruciate ligament. Arthroscopy. 2004;20(9):e117–21.