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Magnetic resonance imaging in patellar lateral femoral friction syndrome (PLFFS): Prospective case-control study

B. Barbier-Brion a,∗, J.-M. Lerais a, S. Aubry a,b, D. Lepage c, C. Vidal d, E. Delabrousse e, M. Runge a, B. Kastler f,b

a Osteoarticular Imaging Department, CHU Jean-Minjoz, 3, boulevard Alexandre-Fleming, 25000 Besançon, France
b Health Intervention, Imaging, Engineering, and Innovation Laboratory, Université de Franche-Comté, 25000 Besançon, France
c Anatomy Laboratory, Université de Franche-Comté, 25041 Besançon cedex 3, France
d Clinical Research Center for Technological Innovation, CHU Saint-Jacques, 2, place Saint-Jacques, 25000 Besançon, France
e Gastrointestinal and Urogenital Imaging Department, CHU Jean-Minjoz, 3, boulevard Alexandre-Fleming, 25000 Besançon, France
f Heart, Chest, and Breast Imaging and Pain Treatment Department, CHU Jean-Minjoz, 3, boulevard Alexandre-Fleming, 25000 Besançon, France

Key words
Friction syndrome; Patellar pain syndrome; 3 Tesla MRI; Knee; Instability criteria

Abstract
Objectives: To describe morphologic abnormalities and signs of patellar lateral femoral friction syndrome (PLFFS) detected by magnetic resonance imaging (MRI).

Materials and methods: Prospective study of 56 knees (21 patients and 30 controls) studied by 3Tesla MRI. Comparative analysis of clinical data, quantitative and qualitative imaging criteria in a population of patients with anterior knee pain associated with an abnormal MRI signal along the lateral aral folds of the infrapatellar fat pad, a characteristic sign of PLFFS, and a control population with no anterior knee pain or abnormal signal from the infrapatellar fat pad.

Results: Patients with PLFFS have anterior and/or lateral knee pain. Their knee has anatomical predispositions for instability, primarily with patella alta (P<0.0001), patellar tilt more than 13.5° (P<0.0001), a patellar nose length less than 9 mm (P=0.0037), a patellar nose ratio less than 0.25 (P<0.0001), a TT-TG distance more than 10 mm (P<0.0001), and a trochlear prominence more than 4 mm (P=0.0056). In 35% of patients, patellar chondropathy is visible, and 48% of patients have patellar or trochlear subchondral abnormalities.

∗ Corresponding author.
E-mail address: b-barbier-brion@hotmail.fr (B. Barbier-Brion).

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Patellofemoral disease is commonly characterized by two main symptoms: patellofemoral pain and patellar instability. In most cases, a well-conducted interview and physical examination, along with a thorough radiographic workup, will determine the nosologic framework of the patellofemoral disease. This has been classified by the Lyon school into three well-defined entities [1]:

- objective patellar instability (OPI), which is defined by the presence of at least one episode of true patellar dislocation or radiological sequelae and at least one anatomical abnormality confirmed by imaging: trochlear dysplasia, patella alta, patellar tilt, TT-TG abnormality;
- potential patellar instability (PPI), which is characterized by the presence of subjective patellar pain and/or instability (reflex) with no history of true patellar dislocation and the same anatomical abnormalities as in OPI;
- patellar pain syndrome (PPS) where no major morphologic abnormality is found on the x-rays. The symptoms are a combination of patellar pain, reflex buckling, and sometimes pseudolocking.

Patellar tendon-lateral femoral condyle friction syndrome has recently been described in the literature and opens up a nosological framework on the border between PPS and potential patellar instability. Patellar tendon-lateral femoral condyle friction syndrome is one of the causes of anterior knee pain. This clinical syndrome and its MRI findings were described for the first time in the literature in 1999 by Brukner et al. [2], who called it infrapatellar fat pad impingement, in a study of eight patients, and then by Chung et al. in a study of 42 patients [3], where the term patellar tendon-lateral femoral condyle friction syndrome was introduced. This syndrome is currently not reported in literature reviews [2–11]: it is probably widely underdiagnosed and underestimated because it is unknown. The clinical signs of this syndrome are the same as those of PPS. They are characterized by anterior pain that is exacerbated by hyperextension and tenderness in the lower pole of the patella. Patients may also complain of tenderness in the infrapatellar fat on either side of the patellar tendon, but not in the tendon itself. The disease centers around focal impairment of the fat pad of the anterior compartment of the knee at the external alar fold (Fig. 1). It is the MRI that confirms the clinical diagnosis, with mainly a focal signal abnormality in this fold (Fig. 2).

To the best of our knowledge, this is the first descriptive prospective 3 Tesla study of patellar tendon-lateral femoral condyle friction syndrome. The objective of this study is to improve the description of the MRI signs found in patellar tendon-lateral femoral condyle friction syndrome. We particularly studied the predisposing morphological variants of the trochlea and patella in patients with anterior pain with a focal signal abnormality of the infrapatellar fat pad compared with a control population.

**Patients and methods**

**Patients**

We performed a prospective follow-up on patients with anterior knee pain for more than 3 months who had undergone a 3 Tesla MRI at our institution between November 2004 and June 2009. Patients with a focal signal abnormality in the anterolateral fat (lateral alar fold) of the anterior compartment of the knee were included. 21 patients (23 knees) were included in this study out of a total of 409 MRIs of the knee performed during that period, 67 of which were for anterior knee pain.

Thirty control subjects with no anterior knee pain were also selected in order to screen for any morphological abnormalities and compare them to those found in cases of PLFFS. Those subjects, with no known patellofemoral history, underwent an exploratory MRI for posterior pain ($n=4$) or suspected internal meniscus lesions ($n=26$). Patients with a history of patellar dislocation, arthroscopy, or surgery of the knee, and indirect MRI signs of a history of patellar dislocation were excluded.

Each enrolled patient underwent a clinical and radiological workup including an MRI. During the MRI appointment, the investigator routinely conducted an interview for the purpose of recording the patient’s history, history of the present illness, and functional symptoms. He performed a physical examination of the knee and the patellofemoral compartment in particular.

He looked for classic signs of patellofemoral disease with Smillie’s test and Zohlen’s test. If there was associated lateral pain, Renne’s test and Noble’s test were done.

**MRI protocol**

All studies were performed with a 3 Tesla MRI (Signa Excite, General Electric Medical Systems, Milwaukee, WI) and a four-channel transmit and receive antenna with dedicated quadrature knee HR coil to allow parallel acquisitions. The knee was positioned in extension with the foot at the zenith. We did three fat-saturated proton-density (fat-sat PD) sequences: axial sequences of the lower third of the quadriceps tendon at the anterior tibial tuberosity perpendicular to the anterior cortical surface of the femur, sagittal sequences at the external edge of the trochlea, with phase encoding in the cephalocaudal direction to minimize artefact from flow in the popliteal artery, and coronal sequences parallel to the bicondylar plane (TR between 2600 and 3500 ms, TE between 30 and 60 ms, number of excitations = 4 for the sagittal plane and 2 for the other planes, FOV between 14 and 16 cm, matrix $= 512 \times 512$, slice thickness $= 3$ mm, interslice gap $= 0.3$ mm, acquisition time $= 4 \text{ min} \ 5 \text{ sec}$ in the sagittal plane, $3 \text{ min} \ 4 \text{ sec}$ in the coronal and axial planes).
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Figure 1. The lateral alar fold (arrowheads) of the infrapatellar fat pad, located between the inferolateral edge of the patella and the lateral facet of the femoral trochlea. Radiologic/anatomic correlation: a: lateral parasagittal MRI slice using fat-saturated proton-density-weighted (fat-sat PD) images; b: axial fat-sat PD slice through the inferior part of the patella; c, d: cadaver slices (Anatomy Laboratory, Medical and Pharmaceutical Sciences Teaching Unit, Université de Franche-Comté).

A T1-weighted sagittal sequence was also done (TR = 860 ms, TE = 21 ms, number of excitations = 2, FOV between 14 and 16 cm, matrix = 512 × 512, slice thickness = 3 mm every 0.3 mm, acquisition time = 4 min 37 sec). All images were stored on our network (PACS) for post-processing of quantitative data.

MRI analysis

All MRIs were independently read by two observers. A rereading was done, when necessary, to reach a consensus.

Characteristic slices were used for axial measurements (Figs. 3 and 4): the cartilage slice was the first axial slice where it was possible to view cartilage on the lateral aspect of the upper part of the trochlea, with the slice through the trochlea outlining a “Roman arch” and on the slice 30 mm above the femorotibial space. We measured the angle of the lateral trochlear inclination (LTI), the depth of the trochlea (D) at the Roman arc hand 3 cm from the joint space, the trochlear joint facet asymmetry (L/M), patellar lateral lateralizarion (or patellar translation), patellar tilt, and the TT-TG or distance between the tibial tuberosity and the bottom of the trochlear groove, now increasingly used in MRI studies [12].

In the sagittal plane, we calculated the height of the patella (patella alta) (Fig. 5) using different methods: Insall and Salvati [13], modified Insall [14], and Caton and Deschamps [15]. We recorded the patellar nose length, the
patellar nose ratio (N/P), and the ventral trochlear prominence: projection X (Fig. 6).

The usual and normal values in the literature are reported in Table 1.

Qualitatively, the type of patella is analyzed on the axial slices based on its morphology and Wiberg classification. Type II patellae with a very small, convex internal face were considered dysplastic. The type of supratrochlear/trochlear junction is recorded. It is classified into two categories: patients with an oblique, square, "stair step" type junction that is aggressive for the patella at the entry to the trochlear notch, and all other types. It was studied on the median sagittal slice through the bottom of the trochlea, corresponding to the point of engagement of the patella (Fig. 7).

We noted signal hyperintensity in the soft tissue below the iliotibial (IT) band (fascia lata). Those high-intensity areas had to be visible in at least two planes of different slices. Then we looked for abnormal intratendinous signals in the patellar ligament and, finally, cartilage abnormalities using the Outerbridge staging system, and we recorded focal or diffuse signal abnormalities of the subchondral bone at the patellar and trochlear facets.

**Statistical tests**

For all statistical tests in this study, we set a significance threshold of 5% (two-tailed). We used two nonparametric statistical tests, since the sample sizes were small: Wilcoxon’s test to compare the mean of a variable between
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Figure 3. Quantitative axial morphologic data: a: bicondylar plane; b: maximum measurement of the internal condyle; c: maximum measurement of the external condyle; d: measurement of the trochlear groove; e: measurement of the external trochlear facet; f: measurement of the internal trochlear facet; g: measurement of lateral patellar displacement or tilt. Trochlear depth $D = [b + c/2] - d$; facet ratio $R = e/f$.

Two groups and Fisher’s test to compare the percentage of a variable between two groups.

Results

Patients

Our population had 67% (14/21) women in the patient group and 33% (10/20) in the control group. The mean age was 32.5 years (range 20–54) in the patient group and 35 years (range 18–69) in the control group.

The friction syndrome was revealed by clinical signs of PPS in 18 out of 19 cases. In eight cases, the pain was peripatellar, but more marked laterally [$\times$ 4] and medially [$\times$ 4]. In five cases, the Renne’s and Noble’s tests were positive with very lateralized peripatellar pain: those patients had symptoms that were essentially peripatellar on exertion. It should be noted that 4 patients reported other functional signs such as pseudo locking or instability.

The syndrome evolved over a period of eight months on average. Regular athletic activity was noted in 14 cases (especially walking [$\times$ 7], skating and skiing [$\times$ 3], handball, football, bicycling, and track and field once each).

Morphologic criteria

Quantitative data

The data are reported in the Table 2.

Figure 5. Patellar height indexes: a: Insall and Salvati (T/P); b: modified Insall (TA/SA); c: Caton and Deschamps (PA/SA). P: length of patella; T: length of patellar ligament; SA: measured joint surface area; TA: measured TTA and inferior edge of SA; PA: measured anterior tibial rim and inferior edge of SA.
We essentially report that the most significant qualitative criteria ($P < 0.0001$) between the patient population and the control population were patellar tilt (threshold $> 13.5^\circ$), TT-TG (threshold $> 10$ mm), and patella alta.

**Figs. 8 and 9** summarize the main abnormalities found. Only one patient had an abnormal intratendinous signal in the patellar ligament near its proximal patellar insertion site. The control had this type of abnormality.

The different cartilaginous and subchondral abnormalities in patients and controls are shown in **Fig. 10**. Fifteen patellae (65%) and 21 trochleae (91%) were found to be free of cartilage damage. Eight patellae had cartilage abnormalities (35%) (**Fig. 11**). Eleven bone abnormalities were seen. The four cases of patellar subchondral bone abnormalities were associated with the aforesaid chondral abnormalities in all cases.

**Discussion**

Anterior knee disease includes many conditions whose nosological framework is still not fully standardized. Thus, the language used in the literature is vague. The distinctions must be clear in order to fit the treatment to the patient as effectively as possible. The advent of MRIs and 3 Tesla high magnetic fields is a crucial contribution to knee imaging. They provide a wealth of information essential for understanding this disease.

Patellofemoral disease is thus the patellar lateral femoral friction syndrome (PLFFS) is a female disease, and our series is in agreement with that data, with two-thirds of our patients being female. In our patients, we found the classic symptoms of PPS and patellar tendon-lateral femoral condyle friction syndrome to be clinically indistinguishable.

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<table>
<thead>
<tr>
<th>Table 1</th>
<th>Normal values and abnormal MRI threshold value for study criteria.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Measurements</strong></td>
<td></td>
</tr>
<tr>
<td>Lateral troclear inclination angle (LTI) (Fig. 4)</td>
<td>16.93° (12.17—21.69)</td>
</tr>
<tr>
<td>Troclear depth (Fig. 3) D 3 cm</td>
<td>5.2 mm (2.4—10.5)</td>
</tr>
<tr>
<td>Troclear projection (Fig. 6) X</td>
<td>4.2 mm (1.7—7.3)</td>
</tr>
<tr>
<td>Troclear facet ratio (Fig. 3) L/M</td>
<td>1.75 (1.1—5.8)</td>
</tr>
<tr>
<td>Lateral patellar displacement (Fig. 3) Tilt</td>
<td>2.5 mm (0—11.2)</td>
</tr>
<tr>
<td>Patellar tilt (Fig. 4) TT-TG</td>
<td>9.2° (0—20)</td>
</tr>
<tr>
<td>Extension to 15° flexion</td>
<td>15 mm (10.5—19.5)</td>
</tr>
<tr>
<td>30° flexion</td>
<td>9 mm (5—13)</td>
</tr>
<tr>
<td>Insall index (Fig. 5)</td>
<td>1 (0.9—1.1)</td>
</tr>
<tr>
<td>Modified Insall index</td>
<td>1.25</td>
</tr>
<tr>
<td>Caton and Deschamps index</td>
<td>1 (0.8—1.2)</td>
</tr>
<tr>
<td>Patellar ratio (NP/P)</td>
<td>11.9 (4—21.7)</td>
</tr>
<tr>
<td>Patellar nose (NP/P)</td>
<td>0.3 (0.15—0.56)</td>
</tr>
</tbody>
</table>
Most patients have anterior peripatellar knee pain, sometimes more pronounced laterally or medially, and in some cases associated with pseudo block or instability phenomena. Patients often work out regularly. In addition, it is not uncommon to find very lateral pain symptoms mimicking ilio Tractus Band Syndrome (ITBS). A tight IT band may be associated with PPS [16]; the intertwining of these syndromes explains the difficulty of the physical examination and justifies doing an MRI, the only test capable of clarifying a clinical picture that has low sensitivity and is thus tricky.

Dysplasia of the trochlea is one of the most important anatomical factors involved in instability and lack of patellar engagement. The MRI was introduced in order to diagnose trochlear dysplasia [17]. The quantitative criteria used for dysplasia are trochlear lateral inclination, trochlear depth, and ventral prominence of the trochlea [17]. With regard to trochlear lateral inclination, and for a threshold value of 11° [18], the means for the patients are significantly below the controls, with a single control case found to be actually dysplastic. For trochlear depth, the mean values for

### Table 2 Statistical analysis of quantitative parameters.

<table>
<thead>
<tr>
<th>Quantitative criteria</th>
<th>Patients</th>
<th>Controls</th>
<th>P (Wilcoxon test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral trochlear inclination (LTI)ₐ</td>
<td>17.13 ± 5.09</td>
<td>19.80 ± 4.25</td>
<td>0.0345</td>
</tr>
<tr>
<td>Trochlear depth at 3 cm (D₃)ᵇ</td>
<td>3.56 ± 1.59</td>
<td>4.34 ± 0.86</td>
<td>0.0657</td>
</tr>
<tr>
<td>Trochlear depth at arch slice (D arch)ᵇ</td>
<td>4.55 ± 1.04</td>
<td>5.14 ± 0.95</td>
<td>0.0297</td>
</tr>
<tr>
<td>Trochlear projection (X)ᵇ</td>
<td>4.22 ± 1.43</td>
<td>2.82 ± 1.03</td>
<td>0.0012</td>
</tr>
<tr>
<td>Lateral and medial trochlear facet ratio (L/M)ᵇ</td>
<td>1.93 ± 0.40</td>
<td>1.57 ± 0.23</td>
<td>0.0012</td>
</tr>
<tr>
<td>Patellar laterizationᵇ</td>
<td>2.90 ± 3.84</td>
<td>-5.2 to 7.5</td>
<td>0.0294</td>
</tr>
<tr>
<td>Patellar tiltᵇ</td>
<td>14.43 ± 6.88</td>
<td>0 to 30</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>TT-TGᵇ</td>
<td>13.64 ± 3.96</td>
<td>7.5 to 22.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Insall index</td>
<td>1.26 ± 0.12</td>
<td>0.96 to 1.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Modified Insall index</td>
<td>1.93 ± 0.13</td>
<td>1.6 to 2.1</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Caton index</td>
<td>1.18 ± 0.15</td>
<td>0.95 to 1.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Patellar nose (NP)ᵇ</td>
<td>9.08 ± 1.94</td>
<td>4.8 to 12.1</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Patellar nose ratio (NP/P)</td>
<td>0.22 ± 0.05</td>
<td>0.13 to 0.3</td>
<td>0.0003</td>
</tr>
</tbody>
</table>

ₐ Measurements in millimeters.
ᵇ Measurements in degrees.
control subjects were 5.2 mm on axial slices 3 cm from the joint space and 4.2 mm at the arch [19]. In the absence of volume acquisition, this proximal measurement lacks accuracy and reproducibility; in our opinion, it is more reliable at the arch where the trochlea is shallower in the patient group ($P = 0.0297$), a source of instability. In cases of trochlear dysplasia, the connection between the femoral shaft and the trochlea is more abrupt. It was Guilbert [20,21] who came up with this original concept. The flat, round, square, and oblique types (by order of severity) are dependent on

![Figure 8](image-url)

**Figure 8.** Example of qualitative and quantitative criteria for control knees (BC: bicondylar plane): a: knee No. 30. No trochlear projection and type 1 trochlear/supratrochlear junction; b: knee No. 12. L/M joint facet ratio of 1.6; c: knee No. 5. No lateral patellar displacement and 8° patellar tilt; d: knee No. 9. Superimposition of two reference slices. TT-TG 5.3 mm; e: knee No. 9. Normal modified Insall index of 1.8; f: knee No. 19. Patellar nose of 15.8 mm.
Figure 9. Example of qualitative and quantitative criteria for patient knees (BC: bicondylar plane): a: knee No. 9. Trochlear projection 6.6 mm and type 3 trochlear/supratrochlear junction; b: knee No. 21. L/M joint facet ratio of 2.4; c: knee No. 6. Lateral patellar displacement (→) of 6.9 mm and 17° patellar tilt; d: knee No. 17. Superimposition of two reference slices. TT-TG 20 mm; e: knee No. 3. Elevated modified Insall index of 2; f: knee No. 5. Patellar nose of 7.5 mm.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Number of knees (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patella</td>
</tr>
<tr>
<td></td>
<td>Internal</td>
</tr>
<tr>
<td>0</td>
<td>1 5</td>
</tr>
<tr>
<td>1</td>
<td>0 0</td>
</tr>
<tr>
<td>2</td>
<td>1 3</td>
</tr>
<tr>
<td>3</td>
<td>0 0</td>
</tr>
<tr>
<td>4</td>
<td>1 3</td>
</tr>
<tr>
<td>SCB</td>
<td>1 3</td>
</tr>
</tbody>
</table>

including 4 above trochlea

Figure 10. Cartilage abnormalities according to the Outerbridge staging classification and subchondral bone (SCB) abnormalities in patients.
the projection of the trochlea. According to that author, 80% of case of OPI or PPI had an oblique or square junction, while 72% of controls had a flat junction. "Stair step" junction areas are aggressive when the patella engages in the trochlea. They are associated with greater mechanical instability, more patellar pain, more marked projection, and more severe dysplasia. At the same time, in the context of OPI, the ventral trochlear prominence, projection (X), the quantitative reflection of this data, has a threshold value of 4 mm [19] and 6.9 mm for a diagnosis of trochlear dysplasia [17]. In our study, whether quantitatively with respect to mean projections, with a 4 mm threshold, or qualitatively (type of junction), there was a significant difference between our patients and our controls: 0.0012, 0.0056, and 0.0064, respectively. With regard to patellar joint facet asymmetry, the control value was 1.75 or 57%; a threshold of 2.5 or 40% gives 100% sensitivity and 96% specificity for the diagnosis of trochlear dysplasia 3 cm from the joint space [17]. In our series, there was no clear statistical power vis-à-vis the threshold at 2.5, but the trochlea of patients had more marked asymmetry than those of the controls and therefore no true trochlear dysplasia, which may nevertheless contribute to patellar instability.

As for patellar displacement, a mean of 2.5 mm was found in the control population and 7.7 mm for knees with dysplastic trochlea: a threshold of 6 mm was set for 75% sensitivity and 83% specificity. Patellar tilt has previously been described as a source of anterior knee pain with no clinical instability or history of dislocation [14]. Tilt seems to be the best indicator of a potential imbalance between the vastus medialis and lateralis muscles. Using the figure of 13.5° as the threshold value, as there is a consensus in the literature for trochlear or muscular dysplasia, a significant difference was found between the two groups in our study. Other authors also state that patellar tilt is a source of anterior knee pain with no clinical instability or history of dislocation [12]. Thus the best indicator of a potential imbalance between the vastus medialis and lateralis muscles is patellar tilt.

The lateral displacement force exerted on the patella by quadriceps contraction depends on the position of the anterior tibial tuberosity relative to the groove of the trochlea. In the population with OPI, 56% of cases had a TT-TG greater or equal to 20 mm while in the population of normal subjects, only 3% reached that threshold. The mean value was 9.3 mm in control subjects [12]. A very high patella is likely to miss engaging if the TT-TG angle is wide or if the balance of the forces between the vastus medialis and lateralis is not satisfactory. In our series, there was a significant difference in the mean TT-GT measurements between patients and controls ($P < 0.0001$); event at a threshold of 10 mm, that difference remains highly
significant (P < 0.0001). Our results for the controls are consistent with those in the literature; for example, Guilbert [20] obtained TT-TG measurements of 8.1 ± 3.3 mm for controls, while we obtained 7.93 ± 3.01 mm for our controls. In our patients, we recorded mean measurements of 13.64 ± 3.96 mm while Guilbert obtained 11.1 ± 3.4 mm and 12.8 ± 5.4 mm, respectively, for PPI and OPI. A 2009 study [22] found that patients with anterior knee pain with no history of instability (as in our patients) had a statistically wider TT-TG angle and greater patellar chondromalacia. Finally, in 2006, Wittstein [12] also found a significant difference between TT-GT measurements in patients with anterior knee pain with no history of dislocation or instability and controls (12.6 ± 1.1 mm versus 9.4 ± 0.6 mm). Thus, they all conclude, as we did, that excessive TT-TG angles, even those less than 20 mm, are a source of anterior knee pain.

To the best of our knowledge, patella height is the only morphologic criterion studied in the context of PLFFS. According to the Insall and Salvati index [13], it is normally 1 ± 0.1. We speak of patella alta if it is greater than 1.2. In MRI terms, we speak of patella alta when the value is greater than or equal to 1.3 [23, 24]. The modified Insall index [14] overcomes the limitations of the Insall and Salvati method described by some authors, including false positive in the case of abnormal patellar morphology, such as a short patellar nose (most of the patients in our study). The patella alta threshold is then 2. Finally, the Caton and Deschamps index [15] is normally between 0.8 and 1.2. We speak of patella alta if it is greater than or equal to 1.2 [25]. This index is interesting because it does not depend on the degree of knee flexion. This index would be the most sensitive for diagnosing patella alta by MRI [24]. For example, in a series of 42 patients, Chung et al. [3] found 79% of patients with patella alta with an Insall ratio greater than 1.3. In 2010, Subhawong also found an association between friction syndrome and patella alta in a predominantly female population [26]. Based on the different possible MRI measurements for patella alta, all indexes show a highly significant difference (P < 0.0001), with more cases of patella alta in patients compared with controls. In our smaller series, according to the indexes, 48 to 57% of patients have patella alta. Patella alta is a major instability criterion [25] and source of pain.

The patients in our series had significantly shorter mean patellar noses and lower mean patellar nose ratios than controls (P < 0.0001 and P = 0.0003). A nose length shorter 9 mm and a patellar nose ratio lower than 0.25 should be considered a criterion for patellar dysplasia [19]. Using a threshold below 9 mm for patellar nose length and a value of less than 0.25 for patellar nose ratio, the tests were also significant (P = 0.0037 and P < 0.0001). Thus, the shorter the patellar nose and the lower the ratio, the more the patients in our series had PLFFS. These findings have previously been reported in the literature, but in the specific area of patellar instability with, according to some authors, greater specificity than patellar height; patients with these anatomic predispositions are thus more likely to experience patellar instability [19].

One of the original findings of our study was visualization of signal abnormalities in the form of signal hyperintensity in fat-sat PD sequences in the fat below the IT band. These high-intensity areas are focal and not outright hyperintense, with no thickening of the adjacent IT band or thickening of the fat. They are different from those described in IT band syndrome. The statistical difference between the presence of this signal abnormality in patients and controls in our series was borderline at P = 0.0629 and needs to be confirmed in a larger sample. The etiology of PLFFS is known to be multifactorial. Some authors have comparatively shown that IT band tightness may play a role in anterior knee pain, and should be taken into account when treating these syndromes and possibly treated by reeducation [27]. Since high magnetic fields have come into use, one wonders if this abnormality is not physiological, but a reflection of the subject’s reduced activity before the test is even performed. With that in mind, a larger series might answer these questions. But already we should pay attention to that area when there is anterior knee pain.

Only one patient had an abnormal signal in the patellar ligament near its proximal patellar insertion site, suggesting a partial tear. In a series of 42 patients, Chung et al. [3] found nine injured tendons versus only one in 50 knees found by Subhawong [26]. The low incidence we found in our series is undoubtedly related to the small sample size.

It is commonly accepted that cartilage injuries that expose the adjacent subchondral bone are a source of pain. There is great innervation in that location where there are nociceptive nerve fibers. We have found bone damage without cartilage involvement, probably due to lack of sensitivity of the detection sequences.

In our opinion abnormal fat in the externalalar fold is a source of pain. Due to repeated microtraumas and varying degrees of resulting hyperplasia in that region, there may be a local inflammatory phenomenon. That area is anatomically very nociceptively innervated and has proven to be highly painful on palpation during arthroscopy performed without anaesthesia [28]. Finally, absent any other obvious cause of knee pain, arthroscopic resection of that inflammatory fat results in patient improvement [29].

In conclusion, various hypotheses may explain pain in PLFFS: abnormal fat in the externalalar fold due to a patellofemoral centering defect, patella alta and patellofemoral chondropathy, bone abnormalities, and pseudo IT band syndrome. We are aware that our study has limitations. Our small patient sample size reflects the borderline statistical results of certain study criteria. Due to the small control sample size, we recorded no case where there was an abnormal fat signal in the externalalar fold with no associated symptoms. The study populations were not homogeneous for sex, a bias inherent in the prospective enrollment of controls. Patient height and weight was not entered in the records, which could be a predisposing factor for this syndrome. There was no protocol using sequences dedicated to cartilage analysis, which may explain an underestimation of chondral damage. However, such long sequences have not yet proven their sensitivity and would have required an arthroscopic or CT scan comparison, thus encumbering our protocol on what is a benign disease.

Conclusion

Peripatellar anterior knee pain is common and it is difficult for the clinician to be sure of the diagnosis. Since the

clinical presentation is similar, the diagnosis of PPS or PLFFS is a process of elimination. Thanks to the MRI, we have clearly shown that patellar and trochlear dysplastic morphologic features are found in patients with anterior knee pain with a focal abnormality of the superolateral fold of the infrapatellar fat pad. If MRI shows only minor morphologic and soft tissue abnormalities, we can diagnose PLFFS with certainty. The merit of our study is that it shows the high incidence of morphologic abnormalities, presumed to cause inflammation of the fat of the external alar fold, which is characteristic of PLFFS, when they were thought to be the preserve of OPI or PPI (especially patella alta). Such abnormalities are widely underestimated in the literature; their role in the genesis of pain is certain, but they are not responsible for OPI.

Magnetic resonance imaging is very useful, since it is specific and makes it possible to confirm the diagnosis. It replaces standard x-rays, since it is possible to calculate all anatomic abnormalities with MRI.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References