Comparison of symptoms and clinical findings in subgroups of individuals with patellofemoral pain

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Patellofemoral pain syndrome (PFPS) is one of the most common musculoskeletal disorders. However, no consensus on the definition, classification, assessment, diagnosis, or management has been reached. We evaluated symptoms and clinical findings in subgroups of individuals with PFPS, classified on the basis of the findings in radiological examinations and compared the findings with knee-healthy subjects. An orthopedic surgeon and a physical therapist consecutively examined 80 patients clinically diagnosed as having PFPS and referred for physical therapy. The examination consisted of taking a case history and clinical tests. Radiography revealed pathology in 15 patients, and scintigraphic examination revealed focal uptake in 2 patients indicating pathology (group C). Diffusely increased uptake was present in 29 patients (group B). In the remaining 29 patients radiographic and scintigraphic examinations were normal (group A). Knee-healthy controls (group D) reported no clinical symptoms. No symptom could be statistically demonstrated to differ between the three patient groups. Knee-healthy subjects differed significantly from the three patient groups in all clinical tests measuring pain in response to the provocations; compression test, medial and lateral tenderness, passive gliding of the patella, but they also differed in Q angle. Differences in clinical tests between the patient groups were nonsignificant. The main finding in our study on patients clinically diagnosed with PFPS is that possible pathologies cannot be detected from the patient’s history or from commonly used clinical tests.

Introduction

The knee region is a common anatomical site of musculoskeletal pain. Self-reported knee pain has prevalences between 7% and 19% in an adult general population of 15–75 years (Urwin et al, 1998; Bergman et al, 2001). Eleven percent of young children report incidents of everyday knee pain (Fearon, McGrath, and Achat, 1996). Patellofemoral pain syndrome (PFPS), one of the most common musculoskeletal disorders (Kannus, Natri, Paakkala, and Jarvinen, 1999), is reported to affect 15%–33% of an active adult population and 21%–45% of adolescents (Lindberg, Lysholm, and Gillquist, 1986).

Anterior knee pain (AKP), a symptom common in traumatic and overuse knee disorders, is often used synonymously with PFPS (Table 1) because a major constituent of the anterior knee pain is the PFPS (Van Tiggelen et al, 2004; Lorberboym et al, 2003). Most authors think of AKP as a symptom in patients with PFPS (Holmes and Clancy, 1998), but the term has also been used to denote a certain syndrome

Accepted for publication 11 April 2005.
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The true cause of PFPS is unknown, and the pain syndrome has been variously named (Table 1).

Some practitioners who find no identifiable cause to the pain use both the term PFPS as well as the term AKP, but the terms are best reserved to describe the patient who has yet to be evaluated. If no causative explanation for the pain is found, despite a thorough investigation, the term idiopathic anterior knee pain (IAKP) seems reasonable (Holmes and Clancy, 1998; Stanitski, 1994; The international patellofemoral study group, 1997). PFPS is often a diagnosis of exclusion.

The most widely accepted theory regarding the etiology behind PFPS suggests that the symptoms are the result of excessive patellofemoral joint stress (force per unit area), owing to abnormal patellar tracking, resulting in inflammatory pain (Merchant, 1988). However, a different etiology that takes the pain mechanism into consideration has been proposed. Ischemic pain, caused by high intraossseous pressure has been suggested as a pain mechanism (Hejgaard and Diemer, 1987). Contributions of a neurogenic mechanism are also mentioned (Wojtys, Beaman, Glover, and Janda, 1990). Mori, Fujimoto, Okumo, and Kuroki (1991) and Sanchis-Alfonso, Rosello-Sastre, and Martinez-Sanjuan, 1999; Stanitski, 1994; Van Tiggelen et al, 2004

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Studies on PFPS show no homogeneity in regard to inclusion and exclusion criteria, and several diagnostic symptoms and tests have been proposed (Table 2). Although a wide spectrum of PFPS symptomatology is reported (Table 2), patients who present a typical history of pain together with positive findings in different clinical tests have been included in previously published studies (Cutbhill et al, 1997). Because no consensus on the definition, classification, assessment, or management has been reached, no validation of clinical tests and signs is possible (The International Patellofemoral Study Group, 1997). PFPS is often a diagnosis of exclusion.

The most widely accepted theory regarding the etiology behind PFPS suggests that the symptoms are the result of excessive patellofemoral joint stress (force per unit area), owing to abnormal patellar tracking, resulting in inflammatory pain (Merchant, 1988). However, a different etiology that takes the pain mechanism into consideration has been proposed. Ischemic pain, caused by high intraossseous pressure has been suggested as a pain mechanism (Hejgaard and Diemer, 1987). Contributions of a neurogenic mechanism are also mentioned (Wojtys, Beaman, Glover, and Janda, 1990). Mori, Fujimoto, Okumo, and Kuroki (1991) and Sanchis-Alfonso, Rosello-Sastre, Monteagudo-Castro, and Esquerdo
found neuromas and nerve injury in the lateral retinaculum. Merchant (1988) suggested one subgroup of AKP to be reflex sympathetic dystrophy of the patella. Butler-Manuel (1992) reported involvement of the sympathetic nervous system in PFPS. Pain in the subchondral bone can be caused by dynamic metabolic adaptations characterized by increases in bone turnover and remodeling (McCarty, 1997; Dye and Boll, 1986). By making us rethink of the pathogenesis of PFPS this information could lead, ultimately, to changes in the assessment, management, and prognosis of this pain syndrome.

Previous studies on PFPS have most probably been undertaken on several different diagnoses with different pathophysiology. This clearly calls for further studies on the pain mechanism involved. Clinical diagnosis of pain syndromes are common and sometimes only based on anatomical locations. A mechanism-based classification of pain could have profound implications: drugs may be prescribed with target distinct mechanisms, scientists may have new guidelines for experimental design, and clinicians may be eventually armed with more reliable and valid

<table>
<thead>
<tr>
<th>Clinical assessment</th>
<th>References</th>
</tr>
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<tbody>
<tr>
<td>Patient history</td>
<td>Arrol, Ellis-Pegler, Edwards, and Sutcliffe, 1997; Cutbill et al, 1997; Garrick, 1989; Goldberg, 1991; Powers, 1998</td>
</tr>
<tr>
<td>Insidious onset of pain</td>
<td>Arrol, Ellis-Pegler, Edwards, and Sutcliffe, 1997; Crossley, Bennell, Green, and McConnell, 2001; Crossley et al, 2002; Davidson, 1993; Cutbill et al, 1997; Powers, 1998</td>
</tr>
<tr>
<td>Activity-induced anterior knee pain</td>
<td>Arrol, Ellis-Pegler, Edwards, and Sutcliffe, 1997; Crossley, Bennell, Green, and McConnell, 2001; Crossley et al, 2002; Davidson, 1993; Cutbill et al, 1997; Powers, 1998</td>
</tr>
<tr>
<td>Unilateral anterior knee pain</td>
<td>Kannus, Natri, Paakkala, and Jarvinen, 1999; Werner, Knutsson and Eriksson, 1993</td>
</tr>
<tr>
<td>Bilateral anterior knee pain</td>
<td>Finestone et al, 1993; Goldberg, 1991; Vahasarja, 1995</td>
</tr>
<tr>
<td>Pain when sitting for long time</td>
<td>Crossley et al, 2002; Davidson, 1993; Galanty, Matthews, and Hergenroeder, 1994; Garrick, 1989; Ireland, Wilson, Ballantyne, and McClay Davis, 2003; Powers, 1998; Powers et al, 2003; Wilson, Carter, and Thomas, 2003</td>
</tr>
<tr>
<td>Pain when palpating peripatellar structures</td>
<td>Cutbill et al, 1997; Garrick, 1989; Goldberg, 1991; Guzzanti, Gigante, Di Lazzaro, and Fabbriciani, 1994; Ireland, Wilson, Ballantyne, and McClay Davis, 2003; Laprade and Culham, 2003; Post, 1993</td>
</tr>
<tr>
<td>Positive compression test</td>
<td>Powers, 1998; Strobel and Stedtfelt, 1990; Welsh, 1985</td>
</tr>
<tr>
<td>Positive apprehension test</td>
<td>Cutbill et al, 1997; Davidson, 1993; Goldberg, 1991; Kannus and Niittyri, 1994; McConnel, 1986; Messier et al, 1991; Percy and Strother, 1985; Reid, 1993</td>
</tr>
</tbody>
</table>
diagnostic tools for treatment and clinical investigation.

There is a consensus in the literature that standard radiographic testing is important for evaluation and diagnosis, providing confirmatory or additional information regarding pathology (Arrol, Ellis-Pegler, Edwards, and Sutcliffe, 1997). However, the literature also disagrees about the correlation between symptoms and physical and radiographic findings, and this diagnosis is often made from a clinical examination only (The International Patellofemoral Study Group, 1997).

Bone scintigraphy is used to detect changes in bone metabolism and has proved to be a more sensitive diagnostic modality in comparison with radiography in evaluation of knee osteoarthritis (Boegård et al, 1999). Scintigraphy has been recommended in the evaluation of patients with PFPS and AKP because it depicts physiological features (i.e., bone remodeling or inflammation), whereas radiography gives biomechanical information such as fractures or osseous loose bodies (Dye, 1993; Hejgaard and Diemer, 1987; Dye and Boll, 1986). A positive bone scintigraphy indicates an increase in bone metabolism, but the method cannot determine whether the final result will be a net loss or a net gain in bone. A pathophysiologically increased bone scintigraph indicates an etiological relationship between the area with increased bone metabolism and the patient’s symptoms. The method can distinguish between localized skeletal conditions such as tumors, bone necrosis, and stress fractures (focal uptake), and increased bone remodeling (diffuse uptake). Dye and Boll (1986), Butler-Manuel (1992), and Leppela et al (1998) have reported diffuse uptake on scintigraphy to be a common finding among patients with PFPS. If bone remodeling is correlated to the etiology of PFPS is not known.

The aim of the present study was to investigate if commonly used symptoms and clinical findings are useful in diagnosing subgroups of individuals with PFPS, according to findings in radiological examinations.

Materials and methods

Patients

All patients and controls gave their informed consent to participate in the study, which was approved by the research ethics committee of the Faculty of Medicine at Lund University. An orthopedic surgeon and a physical therapist examined consecutive patients clinically diagnosed as PFPS and referred for physical therapy. The patients were referred either from primary health care, general practitioners, or orthopedic departments. The following inclusion and exclusion criteria were used to create a study group that were as homogenous as possible.

Inclusion criteria

1. Age 20–50 years. To avoid difficulties in differentiating between PFPS, late symptoms of apophysitis and early symptoms of osteoarthritis, patients between ages 20–50 years were chosen.
2. Pain duration longer than 6 months. Pain duration of more than 6 months has been proposed in studies on PFPS (Cutbill et al, 1997) ruling out short-term pain experiences of other etiology.
3. No functional (symptomatic) instability. Instability in the lower extremity may denote other pathologies.
4. No causative explanation for the pain in clinical examination. PFPS is clinically a diagnosis of exclusion.

Exclusion criteria

Patients were excluded if the clinical examination revealed any symptoms suggesting other pathology of the knee joint such as ligament or meniscus tears, synovial plica, tendinopathy, apophysitis, osteoarthritis, neuroma, fat pad impingement, or patellar instability. Patients were also excluded if they had any previous injuries or operations in the leg or had received any treatment for the pain the last 12 months except for commonly used painkillers, such as paracetamol or nonsteroidal anti-inflammatory drugs.

Control group

The controls were selected from visitors to a health club and were also between 20 and 50 years. These subjects were age and sex matched as near as possible with the pathological groups.
**Exclusion criterion**

The exclusion criterion was any experience of knee pain in the last 6 months or a history of previous knee trauma.

**Methods**

**Study design (Figure 1)**

Eighty patients who fulfilled the above-mentioned inclusion and exclusion criteria underwent a radiographic examination. Five patients dropped out before the radiographic examination because of spontaneous pain relief (n = 2), rejecting further examinations (n = 2), and one patient moved out of the region.

**Radiographic examination**

Standard anterio and lateral views, together with an axial patellar view (Merchant view), were performed. For ethical reasons, the controls were not examined radiographically.

**Scintigraphy**

A dose of technetium 99 m was given intravenously and was allowed to concentrate in bone. Anterior and lateral static images of both knees using a gamma camera 3 hours after the injection were obtained. The scans were visually evaluated blindly and separately by two experienced observers who then reached a consensus. They assessed the degree of localized uptake in the following groups: normal, diffusely increased, and focal. For ethical reasons, the controls were not examined scintigraphically.

The radiographic examination of the 75 subjects revealed pathology in 15 of the patients (group C). Examples of pathology found in the radiographic examination were gonarthrosis. 

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**Figure 1. Study design.**

PFPS = Patellofemoral pain syndrome; IAKP = Idiopathic anterior knee pain
(n = 9), osteochondritis dissecans (n = 2), anomalies (n = 2), and infarction (n = 2).

Scintigraphy was performed in the remaining 60 patients, and 2 patients showed pathological focal uptake and were included in group C. Diffuse uptake on scintigraphy appeared in 29 patients (group B), leaving 29 patients with IAKP (group A) (Figure 1).

Once the radiological examination was completed, dividing the subjects into three groups, a clinical examination composed of a patient history and clinical test was performed. Crossley, Bennell, Green, and McConnell (2001) and Cutbill et al (1997) summarized commonly used clinical symptoms and tests, and the ones proposed by Bizzini, Childs, Piva, and Delitto (2003) and Gerrard (1989) were adopted.

Patient history

Patients were asked about pain onset and duration and pain provoking events, (i.e., activity induced pain, pain on stair climbing, pain after sitting for a long time, and pain when squatting). Pain was recorded as a dichotomous variable, “yes or no,” both in the patient’s history and in the clinical examination. If patients had bilateral pain, in the results the knee with the worst pain is shown.

Tegner’s activity score (0–10) (Tegner and Lysholm, 1985)

The patients and controls filled in their activity score to assess their level of work and sport activities.

Clinical tests

Compression test

The subjects were lying supine, and the examiner compressed the patella toward the femur with the quadriceps muscle relaxed and the knee extended. The subjects were asked to report any experience of pain during the compression.

Medial and lateral tenderness

The subjects were lying supine with the quadriceps relaxed and the knee extended. The medial and lateral borders of the patella were palpated for local tenderness. The subjects were asked to report any experience of pain during the palpation.

Passive gliding of the patella

The subjects were lying supine with the quadriceps relaxed. Passive gliding of the patella medially and laterally was investigated. The subjects were asked to report any experience of pain during the gliding.

Quadriceps angle

The subjects were lying supine with the quadriceps relaxed and the knee extended. With a long axis goniometer, the quadriceps (Q) angle was measured from the spina iliaca anterior superior (SIAS) to the middle of the basis patellae and from there to the tibial tuberosity.

Statistical analysis

All data were analyzed with Statistica 6.0 (StatSoft, Inc). The mean and standard deviation (SD) were calculated for quantitative variables: age, pain duration, and Q angle. To control for the overall significance level, the chi-square test was used to study differences for ordinal categorical data in the Tegner’s activity scale and between nominal (dichotomous) data (i.e., pain-provoking activities reported in the patient’s history and manual clinical tests). This analysis was followed by pairwise post hoc comparisons also with the chi-square test. For quantitative values, the Kruskal-Wallis test was used to test between all four populations, and in corresponding post hoc comparisons, the Mann-Whitney was used to test between pairs. All tests were two-sided and at the 5% significance level. Sensitivity and specificity for the clinical tests were calculated in a 2 by 2 contingency table.

Results

Twenty-nine patients with IAKP (group A), 29 patients with diffuse uptake on scintigraphy indicating slow bone turnover but not regarded as pathological (group B), 17 with diagnosed pathology according to radiography and scintigraphy (group C), and 48 controls (group D) are included in the results (Figure 1). Subjects’ characteristics are displayed in Table 3.
Patient history

Group D experienced no pain in any of the pain-provoking activities, which confirmed the patients’ history, because inclusion criteria excluded such symptoms. Group D differed significantly from all patient groups in the symptoms reported. No symptom could be statistically demonstrated to differ between groups A, B, and C (Table 4).

Functional test

In Tegner’s activity score, group D reported higher levels in sport activities than groups A,

Table 3. Subjects’ characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Group A (IAKP)</th>
<th>Group B (slow bone turn over)</th>
<th>Group C (diagnoses of pathology)</th>
<th>Group D (controls)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>29</td>
<td>29</td>
<td>17</td>
<td>48</td>
</tr>
<tr>
<td>Age (mean years, range, SD)</td>
<td>34 (20–49, 9.0)</td>
<td>34 (20–49, 8.9)</td>
<td>39 (20–49, 9.7)</td>
<td>31 (20–49, 7.5)</td>
</tr>
<tr>
<td>Female (n)</td>
<td>19</td>
<td>15</td>
<td>12</td>
<td>30</td>
</tr>
<tr>
<td>Male (n)</td>
<td>10</td>
<td>14</td>
<td>5</td>
<td>18</td>
</tr>
<tr>
<td>Pain duration (years, range, SD)</td>
<td>7 (1–20, 5.3)</td>
<td>9 (1–25, 6.4)</td>
<td>8 (1–20, 6.8)</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 4. Symptoms and clinical tests.

<table>
<thead>
<tr>
<th></th>
<th>Group A (IAKP)</th>
<th>Group B (slow bone turn over)</th>
<th>Group C (diagnoses of pathology)</th>
<th>Group D (controls)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects (n)</td>
<td>29</td>
<td>29</td>
<td>17</td>
<td>48</td>
</tr>
</tbody>
</table>

**Patient’s history**

<table>
<thead>
<tr>
<th></th>
<th>Group A (IAKP)</th>
<th>Group B (slow bone turn over)</th>
<th>Group C (diagnoses of pathology)</th>
<th>Group D (controls)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activity-induced pain (n)</td>
<td>29</td>
<td>28</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>Stair climbing (n)</td>
<td>29</td>
<td>29</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>Squatting (n)</td>
<td>28</td>
<td>27</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>Sitting for a long time (n)</td>
<td>23</td>
<td>23</td>
<td>14</td>
<td>0</td>
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</tbody>
</table>

**Functional test** (median, range)

<table>
<thead>
<tr>
<th></th>
<th>Group A (IAKP)</th>
<th>Group B (slow bone turn over)</th>
<th>Group C (diagnoses of pathology)</th>
<th>Group D (controls)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tegner: sport</td>
<td>4 (1–9)</td>
<td>3 (1–6)</td>
<td>3 (1–5)</td>
<td>5 (2–9)**</td>
</tr>
<tr>
<td>Tegner: work</td>
<td>3 (1–4)</td>
<td>3 (1–4)</td>
<td>2 (1–5)</td>
<td>3 (1–6)</td>
</tr>
</tbody>
</table>

**Clinical tests**

<table>
<thead>
<tr>
<th></th>
<th>Group A (IAKP)</th>
<th>Group B (slow bone turn over)</th>
<th>Group C (diagnoses of pathology)</th>
<th>Group D (controls)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compression test (n)</td>
<td>24</td>
<td>24</td>
<td>14</td>
<td>1</td>
</tr>
<tr>
<td>Palpation, medial (n)</td>
<td>14</td>
<td>13</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Palpation, lateral (n)</td>
<td>12</td>
<td>13</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Patella gliding (n)</td>
<td>14</td>
<td>13</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>Quadriceps angle (degrees, SD)</td>
<td>14 (4.6)</td>
<td>15 (3.4)</td>
<td>13 (4.9)</td>
<td>10 (2.9)***</td>
</tr>
</tbody>
</table>

*p Group D differed significantly from all patient groups in the clinical signs and symptoms (*p < 0.001).

**Group D reported significantly higher levels in sport activities than all patient groups (*p < 0.001).

***Group D differed significantly from group A and B (*p < 0.001), and group C (*p = 0.074) in Q angle.
B, or C (Table 4, Figure 2). There was no difference in work between groups. Differences between groups A, B, and C were nonsignificant for sport and work activities.

Clinical tests

Group D differed significantly from group A, B, and C in all clinical tests: compression test, medial and lateral tenderness, passive gliding of the patella, and Q angle (Table 4). Group D had a mean Q angle of 10° which differed from group A (14°), group B (15°), and group C (14°) (Table 4, Figure 3). Differences in clinical tests between groups A, B, and C were nonsignificant.

The sensitivity for the clinical tests to differentiate between group B and C ranged from 0.29 to 0.83 and the specificity from 0.17 to 0.69 (Table 5), indicating that these tests could not predict findings seen in radiographic examinations.

Discussion

Many of the tests being used today are regarded as essential in the diagnosis of PFPS. Because clinical practice differs between orthopedic clinics and between clinicians, we wanted to study symptoms and clinical findings in patients with PFPS referred from different clinicians. We are unaware of any study that has previously discussed this issue. Galanty, Matthews, and Hergenroeder (1994) reported that the only finding of a physical examination of students between 10 and 18 years that was significantly related to AKP was pain during isometric quadriceps contraction. However, in their study, two physicians independently examined only 15 subjects. Reider, Marshall, and Warren (1981) reported characteristic findings in different groups of patients complaining of patellar pain, but only one of the authors had examined the subjects. Harrison, Magee, and Quinney (1996) used a questionnaire survey to study how three groups of clinicians rated the importance of 21 clinical tests related to PFPS, but no patients were examined.

Our study shows that both patients with IAKP and patients with similar clinical symptoms, showing diffusely scintigraphic uptake, but also patients with diagnosed pathology on radiological examinations may have identical patients’ histories. Our study also shows that commonly proposed clinical tests are of no value.
differentiating between patients with or without pathological findings on radiographic or scintigraphic examinations. The importance of a radiographic examination in the evaluation of PFPS, however, is obvious because it may reveal biomechanical pathology. If standard radiographic testing is normal and no other diagnosis is possible, the report of typical pain is sufficient for a diagnosis of PFPS.

It is important to remember that pain in the anterior region of the knee is a common problem not always related to PFPS. Posttraumatic articular injury is a common cause of anterior knee pain. Major blunt trauma to the patella may produce articular injury that results in a spectrum of anatomical manifestations from cartilage softening to osteochondral fracture. Other intra-articular problems, such as loose bodies, meniscal tears, ligament ruptures, symptomatic plica semilunaris, neoplasm, and synovitis, may also produce symptoms suggestive of patellofemoral pathology (Merchant, 1988). In the peripatellar region, ligament injuries, tendinopathy, and bursitis must be excluded. Among children and adolescents, apophysitis (i.e., Osgood-Schlatter disease and Sinding-Larsen, Johansson disease) must also be listed. Mild AKP is a common finding following anterior cruciate ligament reconstruction with a bone-patellar tendon-bone autograft (Jarvela, Kannus,

Table 5. Calculations of sensitivity and specificity for the clinical tests.

<table>
<thead>
<tr>
<th>Clinical test</th>
<th>Group B</th>
<th>Group C</th>
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<tbody>
<tr>
<td></td>
<td>Sensitivity</td>
<td>Specificity</td>
</tr>
<tr>
<td>Compression test</td>
<td>0.83</td>
<td>0.25</td>
</tr>
<tr>
<td>Palpation, medial</td>
<td>0.45</td>
<td>0.69</td>
</tr>
<tr>
<td>Palpation, lateral</td>
<td>0.45</td>
<td>0.63</td>
</tr>
<tr>
<td>Patella gliding</td>
<td>0.45</td>
<td>0.50</td>
</tr>
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</table>
and Jarvinen, 2000). Referred pain, particularly from hip pathology and from lumbar radiculopathy must be considered. Tumors, infections, or metabolic processes rarely accompany AKP complaints (Percy and Strother, 1985). A thorough clinical examination involving the patient’s history and a physical and radiological examination performed by experienced clinicians may reveal these different diagnoses.

There is a consensus among authors that standard radiographic testing is important for the evaluation and diagnosis of AKP to rule out serious pathology but not for identifying malalignment (Arrol, Ellis-Pegler, Edwards, and Sutcliffe, 1997). Nimon, Murray, Sandow, and Goodfellow (1998) found, in girls with AKP, that clinical examination plus standard radiographs are adequate to exclude serious pathology. Our study supports this. We also found 39% of our PFPS patients to have diffuse uptake on scintigraphy, which is in agreement with Dye and Boll (1986). Diffuse uptake on scintigraphy has been translated as a change in bone turnover and could be caused by ischemia or overuse (McCarthy, 1997).

Apart from a typical patient history, the most commonly used tests are inspections of malalignment, measurement of the Q angle, and the compression test (Cutbill et al, 1997). The apprehension test has also been recommended, but because this is a test for patellar instability, it is unsuitable for PFPS.

In previous studies on PFPS, lower extremity alignment is evaluated in the search for biomechanical abnormalities (i.e.,, femoral antversion, patella alta and baja, internal and external patella, excessive knee varus, valgus and recurvatum, heel varus, valgus and hyperpronation, and pes planus and cavus) (Dye, 2001; Reid, 1993). We found two knees with patella alta in the radiographic examination. However, we are not sure whether these anomalies cause pain.

The concept that is most widely accepted is that some form of malalignment between patella and femur is of primary causal significance in the genesis of PFPS. An exact determination of the terms patellar alignment, knee alignment, and lower extremity alignment does not exist but would be necessary as they are often used synonymously and contradictory results about malalignment have been presented (Arrol, Ellis-Pegler, Edwards, and Sutcliffe, 1997). Fairbank, Pynsent, van Poortvliet, and Phillips (1984) reported that in pain-free subjects, between 60% and 80% of the population fall into what is generally classed as lower extremity malalignment. The Q angle was previously used as a sign of patellar maltracking but also by using x-ray as an indicator of lower extremity malalignment (Post, 2001; Fulkerson and Hungerford, 1990). Messier et al (1991) reported that a Q angle of over 20° should be regarded as maltracking of the patella. In our study, only three persons would then have had a patellar malalignment syndrome. Post (1993) infers that a positive compression test should be seen as an extremity malalignment indicator. Eighty-three percent of our patients should then suffer from extremity malalignment. Because no large population studies have been conducted to determine normal values for lower extremity alignment or patellar alignment, the presence and degree of malalignment is subjective and what constitutes normal is under debate. Fitzgerald and McClure (1995) studied four different manual clinical tests for patellofemoral alignment where measurement reliability ranged from poor to fair. Their report indicated that such tests did not provide valid information, and they were unable to find a reliable clinical method for assessing alignment. Reid (1993) states that we have an unacceptable use of the terms that is misleading because they are applied to all variations of normal alignment in the lower limbs.

As discussed above, the Q angle might no longer be regarded as reliable in diagnosing PFPS. We found a significant difference between the patient groups and the controls. The differences were, however, only a few degrees and are of limited clinical value because even radiographic measurements of knee alignment (Q angle) show great inter- and intraobserver variability (Ilahi, Kadakia, and Huo, 2001).

The compression test is often positive and regarded as essential by some authors (Guzzanti, Gigante, Di Lazzaro, and Fabbbriciani, 1994; Goldberg, 1991). However, compression and apprehension tests have been reported to be positive in pain-free controls (Harrison, Magee, and Quinney, 1996). In our control group, only one subject had a positive compression test. This might be because our controls, who were highly active physically, were used to excessive
mechanical loading. The apprehension test is a test for patellar dislocation, and poor sensitivity has been reported (Malanga, Andrus, Nadler, and McLean, 2003). Leppala et al (1998), Powers (1998), and Johnson et al (1998) felt that patellar grinding and apprehension tests are characteristic clinical signs. However, the patellofemoral grinding test was intended to indicate pathological changes to retropatellar cartilage, and no studies have documented its sensitivity or specificity in the diagnosis of PFPS (Malanga, Andrus, Nadler, and McLean, 2003).

To diagnose our PFPS patients, we used a typical patient’s history including experience of pain when ascending or descending stairs, when squatting, or during or following a prolonged period of sitting. This agrees with many authors who have used the same pain-provoking activities in their inclusion criteria for PFPS, although the backgrounds for the criteria used differ (Bizzini, Childs, Piva, and Delitto, 2003; Gerrard, 1989). Galanty, Matthews, and Hergenroeder (1994) reported 95% sensitivity to pain from these activities. Crossley, Bennell, Green, and McConnell (2001) found in a systematic review that, in general, PFPS was defined as the presence of pain around the patella in association with activities that load the patellofemoral joint. Although anterior knee pain during or following prolonged period of sitting with flexed knees is reported to be a typical symptom in PFPS (Arrol, Ellis-Pegler, Edwards, and Sutcliffe, 1997), Duri, Aichroth, Wilkins, and Jones (1999) reported that the most consistent symptom related to jumper’s knee was a feeling of deep-seated discomfort during prolonged sitting. All patients reported such symptoms in their study.

Self-scoring questionnaires have been used in assessing both subjective symptoms and functional limitations in PFPS (Thomeé, Renström, Karlsson, and Grimby, 1995). Activity level despite pain is of interest. Tegner’s activity score was chosen because this score has been used in previous studies (Kannus, Natri, Paakkala, and Jarvinen, 1999; Thomeé, Renström, Karlsson, and Grimby, 1995).

The diagnosis of biomechanical dysfunction is fundamental to disease classification within the musculoskeletal system. Tenderness on palpation has been the method of choice for many years and in many diseases (Duri and Aichroth, 1995). In recent years, reports have been published on both poor and good inter- and intraobserver agreement for manual tests (Cook et al, 2001). This applies to palpation of pain areas in soft tissue as well as detection of joint movements and alignment (Ellenbecker et al, 2002; Johnson et al, 1998; Jacobs et al, 1995). Awareness of these disadvantages will be useful for future research. Holmes and Clancy (1998) reported that palpation for tenderness on the medial and lateral boarders of the patella, together with measurements of passive gliding of the patella, are highly subjective tests with no reliability. In our study, no clinical test had both good sensitivity and specificity. Positive findings of pain from palpation of different structures in the knee region as well as pain from provoking activities are not uncommon within other diagnoses. Our study did not use magnetic resonance imaging (MRI) or sonography. That is one reason why we are not able to definitely exclude the possibility that some of the patients suffered from tendinopathy. On the other hand, we excluded patients below the age of 20 years with the hope of excluding late symptoms of apophysitis. One also has to bear in mind that false-positive signals using MRI and sonography have been reported in asymptomatic patients (Jarvela, Kannus, and Jarvinen, 2000). Taken together, these findings suggest that pain from palpation will only marginally help in diagnosing PFPS.

PFPS is in reality a diagnosis of exclusion, and the high incidence of PFPS reported may originate from difficulties in excluding other diagnoses. Most obvious is the problem, among adolescents, to differentiate between PFPS, patellar tendinopathy (jumper’s knee), Morbus Osgood-Schlatter, and Morbus Sinding-Larsen, Johansson. Yates and Grana (1986) found in a prospective study that only 9% of knees with PFPS were considered to have idiopathic chondromalacia.

Our choice of control group could be questioned because controls often are randomly selected from an age- and sex-matched population. Because we wanted to study symptoms and clinical findings in a group of patients clinically diagnosed as having PFPS, we expected to find patients with other diagnoses. Knee pain also is commonly found in a general population (Bergman et al, 2001; Urwin et al, 1998), and
choosing a control group from a matched population most probably would have meant that in the control group, subjects with knee pain would have been present. To avoid this, we selected our controls from subjects with no experiences of knee pain for the last 6 months. However, the difference found in the Tegner’s sport activity score could be explained by our way of selecting the control group.

Other limitations of our study are that we did not test the inter- or intrareliability of the tests used. However, our purpose was not to study the reliability of clinical tests; besides, many of the tests used have been investigated by others. As commented above, palpation for tenderness as well as pain provocation tests show poor reliability (Holmes and Clancy, 1998). The patient’s perception of pain and the examiner’s technique influence these subjective clinical tests. Although many authors have used the same Q angle measurement that we did, no universal accepted method with detailed description exists. The reliability is reported to be poor (Biedert and Warnke, 2001; Ilahi, Kadakia, and Huo, 2001). Although Tegner’s activity scale has been used in several studies on PFPS, it is only validated for knee ligament injuries.

From our observational study, we cannot draw any conclusions about what causes the pain. We know that PFPS is a diagnosis containing several subgroups of patients with possible different pain mechanisms. This calls for further studies on the pathophysiology behind PFPS.

Conclusions

The main finding in our study on patellofemoral pain syndrome is that patients, clinically diagnosed with PFPS, on two occasions and by different clinicians, may still have other diagnoses that cannot be detected from the patient’s history or from commonly used clinical tests. If standard radiographs are normal and a thorough clinical examination still reveals unexplained anterior knee pain, serious pathology is eliminated and a diagnosis of PFPS can be made. Diffuse uptake on scintigraphy may be present in nearly half of the patients with PFPS.

Acknowledgments

The study was financed by grants from Research and Development, Kristianstad University, and Näslund’s Physical Therapy AB, Kristianstad, Sweden.

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