Injury to the proximal deep medial collateral ligament

A PROBLEMATICAL SUBGROUP OF INJURIES

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Most injuries to the medial collateral ligament (MCL) heal well after conservative treatment. We have identified a subgroup of injuries to the deep portion of the MCL which is refractory to conservative treatment and causes persistent symptoms. They usually occur in high-level football players and may require surgical repair.

We describe a consecutive series of 17 men with a mean age of 29 years (18 to 44) who were all engaged in high levels of sport. Following a minor injury to the MCL there was persistent tenderness at the site of the proximal attachment of the deep MCL. It could be precipitated by rapid external rotation at the knee by clinical testing or during sport. The mean time from injury to presentation was 23.6 weeks (10 to 79) and none of the patients had responded to conservative treatment. The surgical finding was a failure of healing of a tear of the deep MCL at its femoral origin which could be repaired. After a period of post-operative protective bracing and subsequent rehabilitation the outcome was good. All the patients returned to their sports and remained asymptomatic at a mean of 48 weeks (28 to 60) post-operatively.

Recognition of this subgroup is important since the clinical features, the course of recovery and surgical requirement differ from those of most injuries to the MCL.

Injury to the medial collateral ligament (MCL) is the most common injury to knee ligaments.1,2 Fortunately, compared with other major ligaments in the knee, the MCL has the greatest potential to heal after injury.3-5 As a result most isolated injuries to the MCL are managed conservatively with an excellent outcome. Early surgery may, however, be indicated when a major injury to the MCL is part of a multiple ligamentous injury, especially when associated with complete disruption of the posterior cruciate ligament.6

However, there is a small subgroup of injuries which specifically involve the deep portion of the MCL. They occur particularly in elite football players who, despite a seemingly uneventful early recovery, develop persistent pain which prevents a return to high-level sports. A characteristic pathological lesion can be identified at surgery. We present the clinical course and outcome of treatment in this subgroup of patients.

Patients and Methods

Our series was drawn from professional or high level sportsmen. Having recognised this problematical subgroup of MCL injuries, data were prospectively collected over a period of three years from 2004 from 17 men, with a mean age of 29 years (18 to 44). There were 15 football players, of whom 12 were professional and three high-level amateurs, one rugby player and one cyclist (Table I).

Detailed histories were obtained from the players and medical staff of the sports clubs involved. When available, video recordings of the injuries being sustained were viewed to establish the mechanism of injury. We also recorded the athlete's sport and level of proficiency, the initial and continuing symptoms, exacerbating or relieving factors, the findings at clinical examination, MRI findings, the initial and current treatment including use of injection therapy with steroids or sucrose, the operative findings, the length of recovery from surgery and the time to return to sport.

The MCL injuries were classified according to their degree of disruption,3 which was assessed clinically, in comparison with the uninjured knee, by detecting the extent of opening of the medial compartment during application of a valgus force with the knee in 30° of flexion. The following grading system was used: grade I, an ‘endpoint’ with < 5 mm of excess opening, grade II, an ‘endpoint’, but a medial opening of between 5 mm and 10 mm.
Surgery was only offered when symptoms persisted despite a programme of rehabilitation which included a period of rest and often one or more injections. All of our patients were initially given options for conservative treatment such as injections or physiotherapy.

The mean time to surgery was 23.6 weeks (10 to 79) after injury. No previous surgery had been undertaken. All operations were undertaken by a single surgeon (AW) under general anaesthesia. The detailed surgical findings, which included the site and exact anatomy of the lesion, were recorded. A standard surgical technique was followed. Preliminary arthroscopy was undertaken to rule out occult injuries and in all cases none was found. A medial longitudinal incision was then made over the site of pain on the medial epicondyle while the knee was flexed to 30° and the hip abducted and externally rotated with the foot supported against a rest or sandbag. The saphenous nerve and its infrapatellar branch were identified whenever possible and protected. Occasionally, they had to be dissected free from scar tissue. The superficial layer of fascia was incised in the line of its fibres to identify the superficial MCL as the second layer. The superior 1 cm to 2 cm of the latter were usually thickened and incised to expose the deep MCL as the third layer. Any persisting disruption to the proximal deep MCL was closed with interrupted No. 1 absorbable sutures using a horizontal mattress technique. In some patients the deep MCL was advanced proximally to the site of attachment of the superficial MCL by taking the suture from the deep MCL through the attachment and tying the sutures on the superficial surface of the superficial MCL. Occasionally, after ‘freshening’ the bone surface, bone anchors were needed for bony reattachment. Once the ligament repair was complete, the layers were closed separately using interrupted absorbable sutures and with ‘double-breasting’ of the layers, if sufficient laxity allowed, in order to reinforce the deep repair. At this stage any excess laxity present initially should have been abolished.

Immediately after operation the knee was compressed with a heavy bandage which was exchanged the next day for an elasticated compression bandage, and supported in a brace. The patient mobilised non-weight-bearing with a hinged brace allowing flexion of 30° to 60° for two weeks followed by partial weight-bearing with the brace allowing flexion from 10° to 90° for a further two weeks. Use of the brace was thereafter continued in an unlocked manner with full weight-bearing for two weeks. Strengthening, agility and proprioceptive exercises were encouraged after a further four weeks, allowing a return to training from 12 weeks. All patients were reviewed at two, six, 12, 24 and 48 weeks post-operatively.

Results

There was a characteristic pattern. Commonly, the mechanism of injury involved not only a valgus stress but also a concomitant external rotation force. In football this usually occurred during ‘tackling’ and was the case in 13 of the 15 football players. Initially, there appeared to be, at most, minor MCL laxity (grade I in II and grade II in one). The firm belief of the treating sports physicians and physiotherapists at the early stages after these injuries was that these athletes had sustained only a minor injury to the MCL. As a result they were usually given a rehabilitation programme which would have allowed a rapid return to running. As a ‘straight-line’ activity presenting little stress to the MCL it was tolerated well. However, in time it became apparent, as more challenging drills were encoun-

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yrs)</th>
<th>Sport</th>
<th>Mechanism of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>44</td>
<td>Amateur football</td>
<td>Valgus and external rotation in a tackle</td>
</tr>
<tr>
<td>2</td>
<td>32</td>
<td>Professional football</td>
<td>Valgus and external rotation in a tackle</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>Professional football</td>
<td>Tackle with leg in air which was forced into valgus</td>
</tr>
<tr>
<td>4</td>
<td>27</td>
<td>Professional football</td>
<td>Valgus force in ‘block tackle’</td>
</tr>
<tr>
<td>5</td>
<td>31</td>
<td>Professional rugby</td>
<td>Valgus and external rotation</td>
</tr>
<tr>
<td>6</td>
<td>23</td>
<td>Professional football</td>
<td>Valgus and external rotation</td>
</tr>
<tr>
<td>7</td>
<td>37</td>
<td>Elite amateur cycling</td>
<td>Valgus and external rotation force (associated with ACL rupture) while skiing</td>
</tr>
<tr>
<td>8</td>
<td>32</td>
<td>Professional football</td>
<td>Valgus and external rotation in ‘block tackle’</td>
</tr>
<tr>
<td>9</td>
<td>31</td>
<td>Professional football</td>
<td>Valgus and external rotation in a tackle</td>
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<td>Professional football</td>
<td>Valgus and external rotation in a tackle</td>
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<tr>
<td>11</td>
<td>18</td>
<td>Professional football</td>
<td>Valgus in ‘block tackle’</td>
</tr>
<tr>
<td>12</td>
<td>18</td>
<td>Professional football</td>
<td>Valgus in ‘block tackle’</td>
</tr>
<tr>
<td>13</td>
<td>26</td>
<td>Professional football</td>
<td>Valgus and external rotation</td>
</tr>
<tr>
<td>14</td>
<td>28</td>
<td>Professional football</td>
<td>Valgus and external rotation in a tackle</td>
</tr>
<tr>
<td>15</td>
<td>23</td>
<td>Amateur football</td>
<td>Valgus in ‘block tackle’</td>
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<tr>
<td>16</td>
<td>22</td>
<td>Professional football</td>
<td>Valgus in tackle</td>
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<td>17</td>
<td>29</td>
<td>Professional football</td>
<td>Valgus and external rotation in a tackle</td>
</tr>
</tbody>
</table>
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In all 17 patients the pain was in the region of the proximal half of the MCL and in 13 it was very well localised to a small area just inferior to the medial epicondyle of the femur. In eight patients there was a palpable thickening of the proximal part of the MCL. In kicking sports such as football, the persisting pain typically occurred when the ball was directed on a curved trajectory such as in a cross-field pass or from kicking a stationary ball when a sharp external rotation force was applied to the knee. However, symptoms were not provoked when kicking hard with the dorsum of the foot as in simple extension of the knee without axial rotation.

Application of pure valgus stress resulted in no excess medial joint opening in five patients, in one of which it increased to grade II after a ‘prolotherapy’ injection of concentrated sucrose solution. Minor excess medial opening (grade-I laxity) was present in 11. One patient presented with grade-II laxity at 30°, but not at 0°.

While valgus stress applied to the affected knee only reproduced the pain in one patient, rapid external rotation of the leg with the knee flexed to 30° did so in all but two in whom it was not possible to reproduce the pain. When the test was positive the pain was present above the joint line in the region of the medial epicondyle, which helped to differentiate the lesion from meniscal pathology.

Palpation revealed soft tissue thickening in the region of the proximal MCL in eight patients and a thickened infrapatellar branch of the saphenous nerve in four.

All patients were investigated by MRI with a typical finding of oedema in the proximal MCL in all. Disruption to the deep MCL (Fig. 1) can usually be identified if sought on the coronal images and was found in 13 patients.

A total of 15 patients had received injections of local anaesthetic and corticosteroid under ultrasound guidance before operation. All had only a short period of benefit before recurrence of the symptoms by four weeks. Four had repeated injections of local anaesthetic and corticosteroid and one had ‘prolotherapy’ in which a highly concentrated sucrose solution was injected in an attempt to provide sclerosis. In this patient (case 8) the initial laxity actually increased after the injections. He also received two injections of corticosteroid at around the same time.

At surgery a characteristic lesion was observed (Fig. 2) in all patients. With the proximal superficial MCL exposed, in those patients with excess laxity on valgus stress testing scarring was visible in the proximal superficial MCL, indicating that it had been involved in the original injury. The infrapatellar branch of the saphenous nerve was involved in the scarring in five patients. When the superficial MCL was incised the characteristic lesion was revealed. The deep MCL was torn just distal to its proximal attachment. In longer-standing injuries the torn ends were smooth and retracted. In more recent injuries there was often granulation tissue present at the defect.

At 12 weeks after operation, all patients had made a complete recovery allowing a return to full training, and remained asymptomatic at their last follow-up at a mean of

Fig. 1a
Coronal fat-suppressed T2 MR scans (a & b) showing oedema and ligament disruption (arrows) at the proximal attachment of the deep medial collateral ligament in two patients.

Fig. 1b
48 weeks (28 to 60). The mean time to return to competitive sport was 17 weeks (12 to 28). In four patients a sensory deficit over the area of the leg supplied by the infrapatellar branch of the saphenous nerve occurred postoperatively. This persisted in two patients by the last follow-up.

Discussion
In recent years the importance of the medial soft-tissue structures has been appreciated with better recognition of the patterns of injury. Surgery may be indicated in some of these injuries particularly if there is damage to the meniscotibial ligament and destabilisation of the meniscus, which usually accompanies dislocation with multiple ligamentous injury to the knee. Most medial soft-tissue injuries can be treated conservatively.

However, there is a subgroup of patients who remain symptomatic despite appropriate treatment. They have a localised refractory injury of the proximal deep MCL, which arises from the medial epicondyle, 15 mm to 17 mm proximal to the margin of the femoral articular cartilage. It inserts into the tibia 2 mm to 3 mm distal to the margin of the articular surface and its fibres run deep to and parallel to the superficial MCL to which they are adherent. Both parts of the MCL resist valgus stress, but the deep portion also resists external rotation, which is a key factor in the understanding of this injury. In 12 of our patients the recorded mechanism of injury included external rotation as well as valgus. This role of the deep MCL in resisting external rotation at the knee is in accordance with the history of the pain being reproduced when applying rapid external rotation to the knee. Since the superficial MCL heals, simple valgus stress rarely causes pain and it is only with the addition of external rotation, which is usually resisted by the deep MCL, that symptoms arise.

It is our experience that sometimes the condition settles with targeted injection of corticosteroid. Therefore such injections are not inappropriate although it could be argued they may predispose to failure of healing of the deep MCL since steroids interfere with healing and the inflammatory response. Although pre-operative MRI characteristically illustrated localised oedema or disruption of the proximal MCL in most of our patients, we could not establish a firm prognosis from the MRI findings.

Recently, it has been reported that injection of platelet-rich plasma has been of value in the treatment of sports injuries and this may have a role in treating the injury which we describe. This is currently being evaluated.

It is interesting that injury to the deep MCL should be associated with poor healing when the superficial MCL heals so readily. A possible explanation is that the proximity of the deep MCL to the joint results in the leaking of synovial fluid between the torn ends of the ligament thereby delaying or preventing healing. Additionally, rapid rehabilitation may impede healing.

Unfortunately, it is not possible to comment on the true incidence of the lesion which we describe since most tears of the MCL recover with conservative treatment. It is possible that some of our cases would have settled if left longer. In fact, all were initially left longer than the usual time that it takes for MCL injuries to heal.

Our series represents a small but defined group of MCL injuries with damage specifically to its deep portion. Appreciation of this is important since failure to do so will result in persistent symptoms and if it fails to recover this injury is amenable to surgical treatment.

Supplementary material

A further opinion by Dr K. Sisak and a table showing the clinical details of all the patients is available with the electronic version of this article on our website at www.jbjs.org.uk

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References


