CONGENITAL SNAPPING KNEE
HABITUAL ANTERIOR SUBLUXATION OF THE TIBIA IN EXTENSION

BARRY D. FERRIS, ANDREW M. JACKSON

From University College Hospital and the Hospital for Sick Children, London

We describe a rare form of congenital snapping knee. In six knees in four children, the tibia subluxed anteriorly on the femur when the knee was extended and reduced spontaneously on flexion. The abnormal movements were seen and felt as sudden snaps or clunks at about 30° of flexion.

All six knees showed similar dysplastic features, although the patients had different clinical syndromes. The mechanism of the subluxation and its management are discussed.

The term congenital dislocation of the knee includes a spectrum of conditions from genu recurvatum through subluxation to complete dislocation (Laurence 1967). We report a very rare form of congenital instability of the knee which does not fit into this classification. There is anterior subluxation of the tibia on the femur every time the knee extends. Reduction occurs spontaneously on flexion and is accompanied by a dramatic and disturbing clunk at about 30°. Knee flexion is not restricted by quadriceps fibrosis and there is no marked recurvatum deformity.

We could find only one reference to this type of knee subluxation in the English literature. Curtis and Fisher (1970) describe 10 knees in five patients showing “heritable congenital tibiofemoral subluxation”. Since the most striking physical sign in our patients was the clunk which accompanies both subluxation and reduction, we have preferred the simpler term ‘congenital snapping knee’. We describe four patients with six snapping knees.

PATIENTS

Three of our four patients had recognisable congenital syndromes; the fourth had multiple congenital abnormalities that did not fit a known pattern. Details are given in Table I.

Table I. Some clinical features and management of the four patients

<table>
<thead>
<tr>
<th>Case</th>
<th>Side</th>
<th>Management</th>
<th>Follow-up (yr)</th>
<th>Valgus alignment (degrees)</th>
<th>Range of movement (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>L</td>
<td>Tenodesis at 1 year</td>
<td>2</td>
<td>5</td>
<td>0 to 120</td>
</tr>
<tr>
<td>2</td>
<td>L</td>
<td>Tenodesis at 8 months</td>
<td>4</td>
<td>15</td>
<td>+ 30 to 135</td>
</tr>
<tr>
<td></td>
<td>R</td>
<td>Tenodesis at 15 months</td>
<td>4</td>
<td>15</td>
<td>+ 30 to 135</td>
</tr>
<tr>
<td>3</td>
<td>R</td>
<td>Supracondylar osteotomy at 13 years</td>
<td>20</td>
<td>20*</td>
<td>5 to 135</td>
</tr>
<tr>
<td>4</td>
<td>L</td>
<td>Division of fascia lata at 11 years</td>
<td>11</td>
<td>10</td>
<td>10 to 130</td>
</tr>
<tr>
<td></td>
<td>R</td>
<td>Nil</td>
<td>11</td>
<td>Neutral</td>
<td>10 to 130</td>
</tr>
</tbody>
</table>

* this knee also showed laxity of the medial collateral ligament

Case 1. Larsen’s syndrome, left congenital snapping knee. This patient had an irreducible congenital dislocation of the right knee, a snapping left knee, and other features of the syndrome. This combination of signs suggests that both knee abnormalities are closely related manifestations of a similar dysplasia (Fig. 1).

Case 2. Catel–Manzke syndrome, bilateral snapping knees. Catel–Manzke syndrome includes the Pierre Robin anomaly, congenital heart disease and toe anomalies (Dignan, Martin and Zenni 1986). Our patient also had bilateral snapping knees, which were obvious at birth.

Case 3. Congenital short tibia with intact fibula and absent fifth ray, right snapping knee. This patient with lower limb dysplasia also had talocalcaneal fusion and a ball and socket ankle joint. The knee subluxation was associated with increasing valgus deformity; a supracondylar osteotomy was required at 13 years of age. Arthroscopy at this time confirmed that there was
complete absence of the anterior cruciate ligament. Radiographs of the mature knee show the extent of the bony dysplasia (Fig. 2).

**Case 4. Dysmorphia**, bilateral snapping knees. This girl was born with a number of congenital anomalies, including a prenatal form of cerebral palsy. In spite of tight hamstrings and some fixed flexion deformity, snapping knees were diagnosed at the age of seven years. The snapping was more obvious on the left and by the age of 11 years had become painful (Fig. 3). Arthroscopy of the left knee showed an attenuated incompetent anterior cruciate ligament.

**MECHANISM OF THE SNAPPING**

The subluxation and reduction in these knees occurs with a sudden jerk rather than a smooth glide. Lachman's test was positive in all six knees, suggesting that anterior cruciate deficiency is an important component of the dysplasia that allows this displacement to occur. In Case 3 the anterior cruciate ligament was absent; in Case 4 it was present but attenuated. In the patient with Larsen's syndrome a thin ribbon-like anterior cruciate ligament was found in the completely dislocated right knee but the snapping left knee was not explored. The iliotibial band was obviously under tension as snapping occurred, but in no case was there any contracture of the tensor fascia lata as demonstrated by Yount's test.

Radiographs of knees that had reached or were near to skeletal maturity (Figs 2 and 3) showed a true bone dysplasia similar to that found in congenital absence of the anterior cruciate ligament (Thomas, Jackson and Aichroth 1985). The shape of the lower femoral and upper tibial articular surfaces was abnormal. The normal movements of the contact point between femur and tibia, forward during extension and rolling back during flexion, did not occur (Goodfellow and O'Connor 1978). In the younger patients it was harder to be certain of morphological abnormalities since the epiphyses were largely unossified (Fig. 1). In addition to articular abnormalities, three knees in two patients showed a valgus deformity, one requiring a corrective osteotomy. In contrast to complete congenital dislocation of the knee, none of our patients showed hyperextension.

**MANAGEMENT**

In our younger patients the subluxation could be passively reduced in extension, but this was not possible in the older patients. This suggests that if surgical treatment is to be undertaken it should be performed early before the subluxation in extension becomes fixed.

In two patients seen within the first few months of life, three congenital snapping knees were immobilised in plaster at 90° flexion for about eight weeks. This treatment had no effect on the condition.

**Operation.** In three knees in Cases 1 and 2 we tried to control the subluxation by an extra-articular tenodesis. A strip of the iliotibial band was raised, based on Gurley’s tubercle. This was passed back deep to the lateral collateral ligament as in the MacIntosh operation for anterolateral instability (MacIntosh and Darby 1976). The strip of fascia was then passed behind the knee to the medial side in the plane between the popliteal vessels and the posterior capsule. Through a separate incision on the medial side of the knee the fascial band was attached to the tendon of rectus femoris. At the operations, the tenodesis effect gave good control of anterior subluxation in extension: the snapping was prevented.
Fig. 2

Case 3. Lateral radiographs of both knees. The anterior part of the femoral condyle of the snapping knee on the left is markedly flattened and the tibial plateau is abnormally tilted.

Fig. 3

Case 4. Lateral radiographs of the left knee in flexion and extension. There is an abnormal appearance of the tibial plateau and flattening of the femoral condyles. The loss of the normal anteroposterior movement of the femur in the tibia can also be seen.
The knees were then immobilised in flexion for six weeks. After recovery they had improved in that the snapping had ceased, but Lachman's test still remained positive.

In one patient (Case 4) the iliotibial band and intermuscular septum were divided as the only treatment (Curtis and Fisher 1970). This patient was mentally deficient and suffered a degree of spasticity. Her recovery was protracted but ultimately the snapping phenomenon and her pain were relieved.

**DISCUSSION**

Congenital snapping knee due to habitual subluxation of the tibia in extension appears to be associated with other major congenital anomalies and with anterior cruciate ligament insufficiency. The subluxation occurs at about 30° of flexion with a disturbing clunk.

In the older children there was an abnormal relationship and altered morphology of both tibial and femoral epiphyses. It may be possible to limit the development of these abnormalities by early surgery aimed at controlling the ligamentous instability.

Conservative treatment with splintage in early childhood appears to be ineffective, but the soft tissue release described by Curtis and Fisher (1970) relieved pain and stopped the snapping in one case. Our modified MacIntosh procedure produced a similar improvement in function, but did not control the abnormal anteroposterior glide in the long term.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

**REFERENCES**


