CONGENITAL DISLOCATION OF THE PERONEAL TENDONS IN THE CALCANEOVALGUS FOOT

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Congenital dislocation of the peroneal tendons is a rare and infrequently reported deformity of the foot in the neonate. Four cases of this deformity associated with a congenital calcaneovalgus deformity of the foot have been treated and followed to the resolution of both of the deformities. The calcaneovalgus foot proved more resistant to correction and required more prolonged and aggressive treatment than was usual when it was found as an isolated deformity. All four patients demonstrated other stigmata of intra-uterine malposition and oligohydramnios or both. Our anatomical studies suggested that the superior peroneal retinacular ligament was the critical stabilising structure for the peroneal tendons. A concept of the pathogenesis of this deformity is discussed and a proven regimen for its treatment presented.

The calcaneovalgus deformity of the foot is a common and relatively benign disorder of the newborn. Clinically, it is characterised by an increased range of dorsiflexion of the foot with a subsequent decreased range of plantar flexion and a valgus deformity of the heel. The dorsum of the foot may actually lie against the anterior aspect of the tibia, but contractures of the skin and other soft tissues are generally not severe. The foot is usually quite supple and can easily be corrected to a neutral position. Normally, radiographic bony abnormalities are absent and permanent deformities do not occur.

In contrast, congenital dislocation of the peroneal tendons is a rare disorder. Estor and Aimes (1923) reviewed the French literature and found nine cases. It has been described infrequently in the English literature and has not been reported in association with calcaneovalgus deformities of the foot in the neonate.

Over the past three years, we have treated four cases (five feet) of dislocation of the peroneal tendons associated with a calcaneovalgus deformity of the foot. All were diagnosed shortly after birth, were similarly treated, and were followed to resolution of the deformities. All proved initially resistant to full passive correction and required a more prolonged and aggressive treatment than one normally expects with an isolated congenital calcaneovalgus deformity.

In the anatomy of the peroneal tendons and their fascial restraints were studied in cadaveric dissections of a foetus and a four-month-old baby. Based on these studies, we propose a concept of the pathogenesis of this deformity and a proven regimen for its treatment.

CASE REPORTS

Case 1. A 2670-gram caucasian girl was born after 37 weeks' gestation to a 29-year-old primigravida. The pregnancy had been complicated by Hashimoto's thyroiditis diagnosed at three weeks' gestation which was treated with 150 micrograms of Synthroid daily. The remainder of the pregnancy was unremarkable with a normal vertex delivery. There was no familial history of orthopaedic anomalies.

At birth she was found to have bilateral calcaneovalgus deformities of the foot that were resistant to passive correction. The peroneal tendons were dislocated anteriorly onto the lateral surface of the fibula. The calcaneovalgus deformity could be manipulated to a neutral position with difficulty, but the peroneal tendons remained dislocated. With manipulation of the foot into an equinovarus position and with posteriorly directed pressure on the peroneal tendons, reduction of the tendons could be achieved. However, when the foot resumed its calcaneovalgus deformity, the peroneal tendons redislocated. The patient was also noted to have a subluxatable right hip which subsequently resolved without treatment. Manipulative reduction of the foot in equinovarus, and manual reduction of the peroneal tendons was started shortly after birth, and maintained with immobilisation in a cast. The casts were changed at weekly intervals. An improvement of the deformity was noted at six weeks but the tendons remained dislocatable. After eight weeks of manipulation and casting, a stable reduction of the tendons was achieved. Thereafter, splitting was continued for another eight weeks. At the two-year follow-up the peroneal tendons remained reduced and stable behind the fibula, and both feet and ankles were normal and without residual deformity.

Case 2. A 1480-gram caucasian girl was born after 33 weeks' gestation to a 24-year-old woman who had had three previous pregnancies, in two of which she had miscarried. The pregnancy had been complicated at 23 weeks by premature rupture of the membranes, but had been allowed to progress until 33 weeks. A vertex presentation and normal delivery ensued. Initial examination of the baby revealed a calcaneovalgus deformity of the left foot and dislocation of the peroneal tendons. The foot was markedly dorsiflexed and everted and was resistant to passive correction. When plantar flexion and inversion of the foot was attempted, the peroneal tendons became bow-stringed laterally across the lateral malleolus. Passive stretching exercises were begun immedi-
Table 1. Clinical findings and results of treatment in four patients with congenital dislocation of the peroneal tendons and a calcanovalgus deformity of the foot

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Affected foot</th>
<th>Associated conditions</th>
<th>Age at start of treatment</th>
<th>Duration of casting</th>
<th>Duration of splintage</th>
<th>Age at follow-up</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>Bilateral</td>
<td>Subluxatable right hip</td>
<td>1 week</td>
<td>8 weeks</td>
<td>8 weeks</td>
<td>2 years</td>
<td>Resolution of calcanovalgus deformity. Tendons reduced and stable</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>Left</td>
<td>Oligohydramnios Premature birth</td>
<td>1 week</td>
<td>8 weeks</td>
<td>None</td>
<td>0.5 years</td>
<td>Recurrence of calcanovalgus deformity. Tendons redislocated</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6 months*</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6 weeks</td>
<td>8 weeks</td>
<td>Resolution of calcanovalgus deformity. Tendons reduced and stable</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>Right</td>
<td>Subluxatable right hip Caesarian section</td>
<td>10 days</td>
<td>8 weeks</td>
<td>None</td>
<td>2.5 years</td>
<td>Mild planovalgus foot. Tendons reduced and stable</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>Left</td>
<td>Breech Caesarian section</td>
<td>4.5 months</td>
<td>6 weeks</td>
<td>4 months</td>
<td>0.5 years</td>
<td>Mild planovalgus foot. Tendons reduced and stable</td>
</tr>
</tbody>
</table>

*Treatment reinitiated after recurrence of deformity
†Case 4 is currently being treated by splinting the foot at night

ate and continued by the mother after the baby was discharged from hospital. At seven weeks the deformity was still present but the foot was more supple, and weekly manipulation and casting were instituted. After eight weeks of casting, a stable reduction of both the calcanovalgus deformity and the peroneal tendons was accomplished. At this time the family was temporarily lost to follow-up. When the child was next seen at the age of six months, she had a recurrence of the calcanovalgus deformity and the peroneal tendons had redislocated. Manipulation and immobilisation in a cast was again initiated and continued for a total of six weeks. This was followed by daily manipulation at home and splinting in a position of equinovarus for eight weeks. At review, when the baby was one year old, the deformity had remained corrected and the peroneal tendons were reduced and stable.

The clinical findings and results of treatment of four patients, including Cases 1 and 2, are summarised in Table 1. Striking similarities were noted in the clinical presentation and response to treatment. All four patients showed other evidence of intra-uterine malposition.

ANATOMICAL STUDY

The normal anatomy of the peroneal compartment at the ankle was studied on cadavers of a 25-week foetus and a four-month-old infant.

The peroneal tendons take a spiral course around the posterior aspect of the lateral malleolus. Originating in the lateral compartment of the leg, the peroneus longus and brevis curve posterior to, and inferior to, the lateral malleolus to emerge on the lateral aspect of the foot (Fig. 1). The posterior surface of the lateral malleolus contains two grooves or sulci. The lateral retromalleolar groove contains the peroneus longus and the medial groove contains the peroneus brevis. These grooves were already present in the 25-week foetus and well formed in the four-month-old infant.

The major restraining structure of the peroneal tendons is the external annular ligament. This ligament originates from the lateral malleolus and the calcaneus and extends posteriorly to insert onto the calcaneal tendon and the lateral surface of the calcaneus. Henley (Estor and Aimes 1923) described two thickenings within the external annular ligament which reinforce it and provide the major stabilising restraint for the tendons.

These thickenings are the superior and the inferior peroneal retinacula (Fig. 2). The superior peroneal retinaculum extends from the lateral aspect of the fibula and the lateral retromalleolar groove, crosses the peroneal tendons posteriorly and inserts into the calcaneus and the calcaneal tendon. The inferior peroneal retinaculum is a more complex structure composed of both superficial and
deep fibres which originate and insert into the calcaneus forming tunnels that hold the peroneal tendons against the lateral aspect of the calcaneus. The deep portion of the inferior retinaculum has a septum which divides the common sheath into two compartments, each holding one peroneal tendon.

Fig. 2
Dissection of the lateral aspect of the ankle in a cadaver of a four-month-old infant. The superior and inferior peroneal retinacula are each demonstrated by a looped suture.

The relative importance of the component parts of the annular ligament complex to the stability of the peroneal tendons was studied by sequentially sectioning and manipulating the foot. With sectioning of the inferior half of the external annular ligament, including the superficial and deep portions of the inferior peroneal retinaculum, and manipulation of the foot into a calcaneovalgus position, we were unable to subluxate or dislocate the peroneal tendons. If in addition the superior peroneal retinaculum was sectioned, the peroneal tendons would readily dislocate. If only the superior peroneal retinaculum was sectioned, the peroneal tendons were dislocatable when the foot was manipulated into a calcaneovalgus position. Thus, it appears that the superior peroneal retinaculum is the key restraining force holding the peroneal tendons in their normal anatomic location.

DISCUSSION
An association between dislocation of the peroneal tendons and planovalgus feet was noted by Estor and Aimes (1923) in four of their nine cases of congenital dislocation of the peroneal tendons. They postulated that the planovalgus deformity was the cause rather than the effect of the dislocation. Patterson, Fitz and Smith (1968) and Sharrard (1979) noted an association between dislocation of the peroneal tendons and a convex valgus deformity of the foot but not with a calcaneovalgus deformity. Mankinen, Sears and Alvarez (1976) reported bilateral dislocation of the peroneal tendons associated with pes planus and severe pronation deformity of the heel in a three-year-old girl with multiple chromosomal and congenital anomalies. We were unable to find any cases of congenital dislocation of the peroneal tendons associated with calcaneovalgus deformity in the neonate.

Congenital dislocation of the peroneal tendons has been attributed to either aplasia of the retromalleolar grooves or weakening of the peroneal sheath or both. Erlich (Estor and Aimes 1923) felt that the primary cause of dislocation was absence of the superior retinaculum with aplasia of the retromalleolar grooves being unimportant. Edwards (1928) suggested that the sulci may be too shallow and may lack sufficient lateral support to retain the tendons. Estor and Aimes (1923) felt that weakening of the sheath could lead to subluxation and displacement of the tendons without the tendons actually leaving the sheath. Our studies confirm the importance of the superior peroneal retinaculum. With the superior peroneal retinaculum intact, dislocation of the peroneal tendons could not be produced, but when it was divided and the other restraining structures were kept intact, dislocation could be produced. In both specimens the retromalleolar grooves were well developed behind the cartilaginous model of the lateral malleus. We believe that the grooves probably occur secondary to movement of the foetal or infant foot when the tendons are anatomically located. It is likely that the shallow grooves described by others are secondary rather than primary phenomena. In support of this, Patterson et al. (1968) described a groove on the lateral aspect of the fibula associated with a subluxation of the peroneal tendons in a six-week-old infant with a convex planovalgus deformity of the foot and dislocation of the peroneal tendons.

Congenital calcaneovalgus deformity of the foot is thought to be the result of intra-uterine malposition of the foetus occurring late in pregnancy. This deformity is frequently found in association with oligohydramnios. Signs of uterine malposition or oligohydramnios occurred in all four patients with dislocated peroneal tendons.

Our four cases represent a more severe type of congenital calcaneovalgus deformity than is usually found as evidenced by their pronounced loss of mobility requiring an extended period of manipulation and casting. We would agree with the conclusions of Estor and Aimes (1923) that congenital dislocation of the peroneal tendons represents the end-result of the deformity rather than its cause. When the peroneal tendons dislocate, the superior retinacular ligament is probably stretched and its restraining force at the lateral malleolus is compromised. Also, the pulley effect of the lateral malleolus is lost and the axis of pull of the tendons is transferred anterolaterally relative to the fibula rather than to its more normal posterior position. The dislocated tendons thereby tend to resist actively any normal corrective forces and hold the foot in calcaneus and valgus.

The rationale of treatment is to first reduce the calcaneovalgus deformity, and then relocate the peroneal tendons. These reductions should be held long enough to
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permit the superior retinacular ligament to assume its normal restraining length, to provide for the restoration of muscle balance, and to allow the development of the retromalleolar grooves. The normal pulley effect of the lateral malleolus should then be re-established. We have successfully treated four patients (five feet) by manipulation of the foot into equinus and varus, by manual reduction of the peroneal tendons, and by immobilisation in a cast. This treatment was repeated at weekly intervals until the deformity was permanently corrected. After this, the foot was manipulated daily at home and held corrected in a resting splint in equinus and slight varus for two to four months. Follow-up at six months and one year is recommended.

REFERENCES


