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Absent Posterior Tibial Artery Associated with Idiopathic Clubfoot

A Report of Two Cases

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The presence of vascular anomalies in the lower extremities of patients with idiopathic clubfoot has been well documented1-6. Absence or a substantial reduction of the anterior tibial artery occurs in approximately 90% of limbs with clubfoot1,2,4,5,7,8. However, an absent or anomalous posterior tibial artery is a very uncommon finding in patients with clubfoot3,6. In patients in whom the posterior tibial artery is absent and the anterior tibial artery is deficient, the peroneal artery becomes the dominant artery to the foot and therefore must be carefully protected during surgical correction of the clubfoot. We report the cases of two patients in whom the posterior tibial artery was found to be absent at the time of an operative release procedure for clubfoot. We also report the arteriographic appearance of an absent posterior tibial artery associated with clubfoot, which has not been reported previously in the orthopaedic literature, to our knowledge. The families of both of our patients were informed that data concerning the cases would be submitted for publication.

Case Reports

CASE 1. A newborn infant with bilateral idiopathic clubfoot was initially treated with serial manipulations and corrective casts. The application of the casts started when the patient was two weeks of age and continued on a weekly basis until the patient was ten weeks of age. Incomplete correction was obtained, and the patient was scheduled for a bilateral complete soft-tissue release at nine months of age. Preoperative clinical evaluation revealed the absence of a palpable dorsalis pedis pulse bilaterally and the absence of a palpable posterior tibial pulse on the right side.

The operation was begun on the right foot with use of the Cincinnati incision9 under a tourniquet pressure of 200 mm Hg. The tibial nerve was identified in the sheath posterior to the posterior tibial tendon and was of normal size; however, the posterior tibial artery was not present. Additionally, an accessory flexor digitorum longus muscle was present immediately adjacent to the tibial nerve. The Achilles tendon was then lengthened with use of a z-lengthening technique. After medial, posterior, and lateral release of the ankle and subtalar joints, a small artery (1 mm in diameter) located on the posterior surface of the ankle joint was noted to have been transected. The tourniquet was immediately deflated, and circulation to the foot returned instantaneously. Intraoperative consultation with a vascular surgeon was obtained, and repair of this small artery was not attempted because of the technical difficulty and the unlikelihood of maintaining patency after repair. The remaining surgical release was completed without difficulty. After the release, the talonavicular joint was held reduced with a Kirschner wire. The same procedure was then performed on the left foot, where normal vascular anatomy was noted. An accessory flexor muscle was not present. Both feet healed uneventfully and the correction was found to have been maintained at the time of the most recent follow-up, seventeen years postoperatively.

CASE 2. A two-week-old boy with bilateral idiopathic clubfoot was treated with serial manipulations and corrective casts. Incomplete correction was obtained, and the patient underwent bilateral complete soft-tissue release operations at the age of ten months. Clinical examination revealed the absence of a palpable dorsalis pedis pulse bilaterally and the absence of a palpable posterior tibial pulse in the left limb. The left foot was operated on first with use of the Cincinnati incision under a tourniquet pressure of 200 mm Hg. An accessory flexor digitorum longus muscle was identified immediately adjacent to the tibial nerve and attaching distally to the flexor digitorum longus tendon. The posterior tibial artery was noted to be absent from the sheath containing the tibial nerve. However, a vascular bundle was identified just anterior to the Achilles tendon and adjacent to the posterior ankle capsule, and it was protected. The soft-tissue release was completed without incident. Full correction of the equinus deformity was not attempted because of the amount of stretch that would have been placed on the seemingly important posterior vascular structures. The same procedure was then performed on the right foot, in which normal vascular and muscular anatomy was noted. Postoperatively, the serial application of casts was used to gradually dorsiflex the left foot.
Hindfoot varus and equinus deformities as well as forefoot adduction recurred on the left side, and revision clubfoot surgery was performed under tourniquet control when the patient was seven years of age. Again, the complete absence of a posterior tibial artery was observed. During the medial, posterior, and lateral releases of the ankle and subtalar joints, the anomalous vascular structures overlying the posterior aspect of the ankle joint were carefully preserved. The tourniquet was deflated, the extremity had excellent perfusion distally, and the pulse over the anomalous vasculature was good. Intraoperative continuous-wave Doppler assessment indicated the absence of the anterior tibial artery. A planned concomitant midfoot osteotomy was deferred until the exact vascular anatomy could be delineated with use of arteriography.

Arteriography was performed the next day with use of the Seldinger technique and a left femoral artery approach. Frontal and lateral digital arteriograms of both lower extremities were made. No complications occurred. Arteriography of the left lower extremity showed that the external iliac, common femoral, deep femoral, superficial femoral, and popliteal arteries were normal. A hypoplastic anterior tibial artery was shown originating from the popliteal artery and terminating as a small branch to the ankle. No posterior tibial artery was identified on either the frontal or the lateral projection (Figs. 1-A and 1-B). Lastly, a dominant peroneal artery, originating from the distal popliteal artery, was found to cross the ankle posterior to the distal tibiofibular joint to form a lateral plan tar arch. No vessel posterior to the medial malleolus was visu-
alized. No anomalous venous structures were seen.

Arteriography of the right lower extremity showed normal arterial anatomy superior to the knee. A hypoplastic anterior tibial artery was noted. The posterior tibial artery arose from a normal popliteal artery, crossed the ankle posterior to the medial malleolus, and formed a normal plantar arch. The peroneal artery was normal in its origin and course. There was no additional surgical management. At the time of this writing, the patient had persistent, mild metatarsus adductus of the left foot.

**Discussion**

Although gross anatomic studies have failed to demonstrate an association of vascular anomalies with clubfoot, several arteriographic studies have shown a strikingly high prevalence of associated arterial anomalies in the limbs of patients with clubfoot. The most common abnormality has been an absent anterior tibial artery, which has been found in up to 85% of children with clubfoot compared with 12% of the general population. In these children, the anterior tibial artery was shown to terminate at the level of the distal tibial epiphysis. In contrast, the posterior tibial artery was normal in all patients and served as the dominant vessel of the foot, supplying the deep plantar arch by means of the lateral plantar artery. Because of the high prevalence of deficient anterior blood supply in patients with clubfoot, the argument has been made that, at the time of preoperative planning and surgical correction of clubfoot, it is best to assume the complete absence of the anterior tibial artery. As a result, great emphasis has been placed on protecting the posterior tibial artery and its terminal branch, the lateral planatar artery, during clubfoot release operations, to prevent ischemia of the foot.

Because the posterior tibial artery is often the principal arterial supply to the foot in patients with clubfoot, anomalies involving this vessel are of particular concern when these patients are managed surgically. To our knowledge, only one case of an absent posterior tibial artery associated with clubfoot has previously been reported in the orthopaedic literature. It was concluded that the peroneal artery was the major source of blood flow to the foot in that patient. Anomalies of the posterior tibial artery are estimated to occur in 5% of the general population, although the complete absence of that vessel is very rare. Anatomists have shown that, when the posterior tibial artery is absent in the general population, an embryonic precursor of the peroneal artery persists and performs the function of the posterior tibial artery. This artery, known as the “great peroneal artery,” usually occupies the position of the posterior tibial artery at the ankle, just posterior to the medial malleolus, before terminating in the sole of the foot.

In our two patients, however, no vessel was present posterior to the medial malleolus. In both patients, small vessels lay along the posterior ankle joint capsule; in our second patient, arteriography showed that those vessels were branches of the peroneal artery.

The question arises regarding the utility of preoperative assessment of circulation to the clubfoot. The absence of a palpable pulse in the typical location posterior to the medial malleolus should serve as a warning of the possible absence of the posterior tibial artery. In such instances, noninvasive imaging such as Doppler ultrasound has not proved to be consistently reliable in delineating vascular anatomy in patients with clubfoot deformities. Preoperative arteriography is an effective way to delineate the vascular anatomic relationships in patients with clubfoot, although the indications for and the cost-effectiveness of identifying the rare condition of an absent posterior tibial artery cannot be ascertained on the basis of the two cases reported here. However, the absence of a posterior tibial artery during surgical dissection should serve as a warning of a potentially precarious circulation to the foot. Posterior and lateral dissection must be done with extreme caution so as not to injure the peroneal artery or its branches. The potential to injure the peroneal artery also exists during a percutaneous Achilles tenotomy to correct residual equinus contracture in patients with clubfoot deformity who have been treated with the increasingly popular Ponseti method.

Both of our patients were noted to have an accessory flexor digitorum longus muscle in one foot, which was the foot that had the absent posterior tibial artery. The accessory flexor digitorum longus muscle has been reported to occur in up to 6.6% of patients with clubfoot. However, its association with vascular anomalies of the foot has not, to our knowledge, been previously reported. A review of a larger series of patients with clubfoot may shed additional light on this association and its potential role in the etiology of this disorder.

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