Lateral hindfoot impingement (LHI) is a subtype of ankle impingement syndrome with classic MRI findings. Biomechanically, LHI is the sequela of lateral transfer of weight bearing from the central talar dome to the lateral talus and fibula. The transfer occurs due to collapse of the medial arch of the foot, most commonly from posterior tibial tendon (PTT) and spring ligament (SL) insufficiency. Clinical features include lateral hindfoot pain, deformity, and overpronation on gait analysis. MRI changes continuously reflect the altered biomechanics as the syndrome progresses over time, including typical and often sequential changes of PTT and SL failure, increasing heel valgus, talocalcaneal and subfibular impingement, and finally lateral soft tissue entrapment. In addition to diagnosis, MRI is a useful adjunct to plan surgical treatment.

Biomechanics

The PTT, a powerful invertor and plantar flexor, elevates the talonavicular joint by its contraction and thus maintains the medial longitudinal arch of the foot. It stabilizes the hindfoot against valgus deformity. Hence, PTT insufficiency causes flatfoot deformity and heel valgus (6).

Most cases of LHI originate in the medial compartment from PTT dysfunction and less commonly from isolated tears of the plantar calcaneonavicular ligament (pCNL) or spring ligament (SL) (7). The PTT is the primary dynamic stabilizer of the talonavicular joint or the keystone of the foot, thus maintaining the medial arch. The SL is the primary static stabilizer. The two structures are synergists and most cases of PTT failure result in adjacent SL
Degeneration. PTT failure leads to repetitive talar head descent, thereby straining and injuring the overloaded SL. Thus, PTT failure leads to a cascade of peritalar subluxation, talar head descent, flatfoot, lateral weight transfer, heel valgus, and finally LHI (8).

Disproportionate lateral eversion from PTT failure causes abutment of the lateral process of the talus and the calcaneum at the angle of Gissane, referred to as talocalcaneal impingement (TCI). Left uncorrected, continuous eversion forces further cause the calcaneum to abut the distal fibula, referred to as subfibular impingement (SFI). Subfibular impingement can rarely be seen in isolation without TCI. TCI alone or with SFI constitutes LHI. Endstage LHI leads to soft tissue changes in the lateral gutter including peroneal tendinosis and subluxation, sural nerve compression, entrapment of the calcaneofibular ligament (CFL), and lateral adventitial bursitis (9).

Clinical features

Patients of LHI syndrome present with chronic lateral hindfoot pain, instability, deformity, and gait dysfunction with overpronation. Occasionally a remote history of medial foot pain in the past is elicited. The clinical presentation of foot pain migrating from the medial to lateral compartment over several years follows the biomechanics of PTT dysfunction in the medial compartment leading to lateral weight transfer. On clinical examination, pes planus and hindfoot valgus are noted (10). The deformity is initially flexible but turns rigid and constant as the disorder evolves. Patients with long standing history of lateral hindfoot pain and deformity may have loss of sensation along the distribution of the sural nerve with a positive Tinel’s sign on examination.

Differential diagnosis

Lateral hindfoot pain may have several etiologies other than talocalcaneal and subfibular impingement. Entrapment of fat and joint capsule in the sinus tarsi has similar clinical features. Sinus tarsi syndrome may also be secondary to PTT dysfunction and occasionally precedes LHI (11). Other clinical differential diagnoses include subtalar degenerative arthritis, indolent infections, occult fractures, chondromatosis, talar osteochondritis dissecans, primary sural neuropathy and isolated peroneal tendinosis. In the clinical circumstance of a wide differential diagnosis, MRI contributes to a single, comprehensive diagnosis as well as to triage of patients into conservative and surgical management options.

Imaging of LHI

Weight-bearing frontal and lateral radiographs of the ankle and foot (Fig. 1) with possible addition of hindfoot alignment views are often the first line of assessment in patients with persistent hindfoot pain (12). If the diagnosis is evident and trial of conservative management is favored, cross-sectional imaging is not obligatory. Computed tomography (CT) is useful for pre-operative planning as it displays osseous anatomy to an advantage. For the measurements of various angles, weight-bearing cone beam CT may be superior to radiographs due to lack of superimposed bones and greater reproducibility (Fig. 2) (13). Although ultrasonography (US) does not accurately depict bone alignment and articular changes, it can depict tendon abnormalities in LHI syndrome. Tenosynovitis, tendinosis and tears of the PTT are easily diagnosed with US (14). The dynamic capability of US is useful to demonstrate tendon instability. US is also employed for guiding intra-articular injections of local anesthetics for confirmation of diagnosis and for directing suture anchors. US-guided steroid injection is a palliative option for intractable pain.

MRI, with its inherent sensitivity for bone marrow edema and multiplanar capabilities is the modality of choice to narrow the differential diagnosis of lateral hindfoot pain when radiographs are equivocal and for detailed assessment of bone and soft tissue changes.
Figure 2. a, b. Weight-bearing cone beam CT showing commonly used metrics of arch integrity and foot alignment in a case of acquired pes planus (13). Sagittal reconstruction (a) shows reduced calcaneal pitch. Calcaneal pitch is the angle of the plantar calcaneum (solid line) to the horizontal (dotted line) and is normally more than 18°. Coronal reconstruction (b) shows increased heel valgus angle. Heel valgus angle is the angle of the medial margin of the calcaneum (solid line) to the long tibial axis (dotted line) and is normally less than 6°. Image Courtesy: Khushboo Pilania and Bhavin Jankharia.

Figure 4. a–d. Grades of PTT dysfunction (straight arrows) with sprains of the synergistic calcaneonavicular (spring) ligament (curved arrows) (11, 15). Axial PD FS images in different patients. Grade 1a (a) is a thickened tendon without significant longitudinal splitting. Grade 1b (b) is a thickened tendon with significant longitudinal splits. Grade 2 (c) is an attenuated tendon compared to the neighboring FDL and FHL tendons. Grade 3 (d) is tendon rupture and avulsion from the navicular tuberosity insertion (NT). Associated degeneration and interstitial tearing of the superomedial calcaneonavicular ligament (a–c, curved arrows) and medioplantar oblique calcaneonavicular ligament (d, curved arrow) are seen.
Assessment of the entire spectrum of bone and soft tissue changes seen in LHI syndrome.

**MRI features of LHI**

**PTT and SL anatomy**

The PTT originates from the proximal tibia, fibula, and interosseous membrane and enters the foot by sharply changing angulation at the ankle in the tibial retromalleolar groove, where it is protected by the flexor retinaculum forming a fibro-osseous tunnel (6). In the midfoot and hindfoot, the PTT has a principle insertion into the navicular tuberosity and numerous and variable accessory insertions into multiple tarsal bones and metatarsal bases. It is the anterior and medial most tendon in the posteromedial compartment of the ankle and foot (Fig. 3). The flexor digitorum longus (FDL) and flexor hallucis longus (FHL) are successively located posterior and lateral to the PTT.
The SL, a synergist of the PTT has three main components from superomedial to inferolateral: the superomedial calcaneonavicular ligament (smCNL), the medioplantar oblique calcaneonavicular ligament (mpoCNL) and the interplantar longitudinal calcaneonavicular ligament (iplCNL). The smCNL (Fig. 3) creates a hammock-like structure that supports the talar head and talonavicular joint and separates the PTT from the talus bone. It is the largest and most important bundle of the pCNL and the strongest static stabilizer of the longitudinal arch. The mpoCNL and iplCFL are weak stabilizers.

PTT and SL abnormalities

PTT insufficiency as seen on MRI ranges from tenosynovitis, tendinosis and tendon elongation to frank tears, rupture, discontinuity, and tendon retraction. The prevalence of LHI significantly increases with grading of PTT tear (Fig. 4) (11, 15). However, a degenerated tendon might be elongated and dysfunctional in the absence of a visible tear (Fig. 5). The normal excursion of the PTT is merely 1-2 cm and any elongation, even by a centimeter, adversely impacts its ability to maintain the medial longitudinal arch (8). Signal alteration in the SL representing degeneration, sprains, or interstitial tearing is a sequela of PTT abnormality (Fig. 6). Although accessory navicular bones are often incidental imaging findings, type II and III variations can lead to flatfoot and pain, as the more proximal insertion of the PTT reduces the insertion angle, thereby altering biomechanics and escalating stress (Fig. 7) (16). This subset predictably presents at a younger age than PTT degeneration.

Heel valgus angle

Hindfoot valgus malalignment, the prerequisite for LHI can be objectively quantified by the hindfoot valgus angle. It is calculated as the angle between the extrapolated long tibial axis and a line along the calcaneal medial cortex on the most posterior coronal...
image that includes both the tibia and calcaneum, but excludes the sustentaculum tali (Fig. 8). The medial wall is used for the calculation as it has lesser variability due to fewer bony prominences. The normal range is less than 6 degrees (17). Most likely, this angle is underestimated on MRI as it is a non-weight-bearing technique (12).

**Talocalcaneal impingement**

TCI is seen as osseous abutment of the lateral facet of the talus with the calcaneum at the angle of Gissane. Subcortical sclerosis, cystic change and marrow edema are seen in the abutting talus and calcaneum (Fig. 9) with the occasional loose body. Interestingly, talar edema can be seen in asymptomatic individuals with an accessory anterolateral talar facet (18).

**Subfibular impingement**

SFI is seen as osseous abutment between the distal fibula and calcaneum. Occasionally, a calcaneal neofacet may form. Similar to TCI, subcortical sclerosis, cystic change and marrow edema are seen in the abutting distal fibula and calcaneum at the location of impingement (Fig. 10) (19). Marrow edema in relation to a neocalcaneal facet must be distinguished from a fibular stress fracture. In addition to marrow changes, soft tissue entrapment between the calcaneum and distal fibula appears as extensive soft tissue thickening due to fibrosis and fatty atrophy (Fig. 11).

**Peroneal tendon abnormality and lateral adventitial bursitis**

In the endstage of LHI, peroneal tendinosis is seen as increased signal within the enlarged longus and brevis tendons that may progress to subluxation and dislocation secondary to rupture of the peroneal retinaculum in the retromalleolar groove (Fig. 11) (9). The calcaneofibular ligament (CFL) is often trapped and thickened (Fig. 12). Adventitial bursitis may be present in the lateral subcutaneous fat.

**Treatment**

Goals of treatment in LHI syndrome are pain relief and prevention of additional deformities. The first line of treatment is non-operative with conservative management using nonsteroidal antiinflammatory drugs, bracing, and orthotics. Activity modification and physiotherapy are advised. Image-guided injections of local anesthetics and steroids in hindfoot articulations and sural blocks help in confirmation of diagnosis for pre-operative planning. In recalcitrant cases, arthroscopic or open surgery is performed for pain alleviation and deformity management (20). Operative options include subtalar arthrodesis and calcaneal osteotomy (21). Fibular excision, peroneal tendon transposition and sural nerve neurolysis are options for subfibular impingement.

**Conclusion**

In conjunction with clinical examination, diagnostic and interventional radiology techniques are essential to triage patients into conservative and surgical management choices. MRI assessment further directs the orthopedic surgeon towards the appropriate operative algorithm.
Conflict of interest disclosure
The authors declared no conflicts of interest.

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