Assessment and management of cavus foot deformity

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Abstract

The cavus foot, or pes cavus, is a deformity of the foot characterised by a high longitudinal arch. It is classically associated with neurological conditions and muscle imbalance. The deformity varies in severity and can affect any or all of the hindfoot, midfoot or forefoot. Careful examination should be performed and underlying neurological conditions sought. We outline an *á la carte* approach to surgical management, in which deformity of the ankle and hindfoot is addressed before moving on to assessment and correction of the mid- and forefoot. Flexible deformities can be corrected with ligament rebalancing and osteotomies whereas fixed deformity frequently requires arthrodesis. Dynamic muscle equilibrium should always be re-established using appropriate tendon transfers.

Keywords cavovarus; cavus; deformity correction surgery; varus

Introduction

The cavus foot, or pes cavus, is a deformity of the foot characterised by a high longitudinal arch. It is classically associated with neurological conditions and varies in severity, depending on the underlying cause. The deformity can affect any or all of the hindfoot, midfoot or forefoot. This paper outlines and approach to the clinical evaluation and management of the cavus foot.

Clinical presentation

Cavus feet may present at any point on a spectrum of severity and in order to detect the most subtle or early stages of deformity, careful history taking and physical examination are essential. Family history of foot deformity or neurological conditions should be sought. The presence of pain or progression of deformity should prompt further investigation.

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Hindfoot assessment

Mild cases of cavovarus deformity can be identified clinically by the 'peak-a-boo' sign, whereby the medial aspect of the heel pad can be seen when inspecting the standing patient from the front. Hindfoot varus is best visualised by inspection from behind the patient (Figures 1 and 2). This not only allows easier appreciation of the alignment of the hindfoot relative to the axis of the limb, but also eliminates the possibility of a false positive 'peek-a-boo' sign secondary to external rotation of the limb in the presence of metatarsus adductus.

Hindfoot varus puts the lateral ankle ligament complex under increased stress and frequent injury may lead to lateral ligament complex incompetence. Frequently, young and active patients who present with repeated ankle sprains have subtle cavovarus deformity. Peroneal tendon tears or tendinopathy may also be present.¹ When reconstructing the lateral ligaments of the ankle or peroneal tendons in the presence of pes cavus, we recommend a correction of the cavovarus deformity at the same sitting.

Early identification of cavovarus, whilst the deformity is still flexible, allows correction with soft-tissue rebalancing procedures, rather than osteotomies or fusions. Furthermore, early detection and treatment of varus ankle position may prevent secondary ankle osteoarthritis.

In cavus foot deformities, the talo-calcaneal angle is reduced. This results in the cuboid being positioned inferior to the navicular rather than lateral to it. During the gait cycle, the Chopart joint is defunctioned and the foot remains locked, causing less effective dissipation of stress.² Many of the clinical symptoms arise from this lack of function in the Chopart joint. Patients may present with Achilles tendinopathy and often have reduced range in their triceps surae. The Silfverskiöld test differentiates isolated gastrocnemius contracture from the Achilles tension. Patients may have tibial stress syndrome or stress fractures, varus knee mal-alignment or iliotibial band syndrome. Plantar fasciitis is common, as is midfoot pain or degeneration of the midtarsal joints.

Mid and forefoot

There is invariably an increased medial longitudinal arch obvious on inspection with the plantarflexed position of the first ray. The Coleman block test ascertains whether the hindfoot deformity is fixed or flexible. If, on performing the Coleman block test, the hindfoot returns to a neutral or valgus position, this suggests that the hindfoot deformity is flexible and is likely driven by excessive plantar flexion of the first ray.

In the forefoot, problems may arise from the tripod effect of the cavus foot. The distribution of force is concentrated upon the first and fifth metatarsal heads and callosities may be found here. Additionally, sesamoid stress syndromes may be present along with metatarsalgia. Over time, the toes may become clawed, especially in the presence of a neurological cause or extrinsic weakness. If the lumbricals are overpowered by extensor digitorum and extensor hallucis longus, the toes will progress through flexible to fixed clawing.³



Figure 1 Hindfoot varus, secondary to mal-united ankle fracture.

Patients often favour walking on the outer border of the foot predisposing to a painful lateral column or 5th metatarsal stress fractures (Figure 3²). Again, caution is advised against surgically managing a 5th metatarsal stress fracture in isolation in the presence of cavus deformity, as failure of fixation or non-union is probable. We recommend correction of the cavus at the same time.

Aetiology

Cavus foot deformity occurs due to muscle dysfunction and imbalance between antagonist muscle groups. The term 'subtle



Figure 2 Severe bilateral cavovarus, secondary to Charcot Marie Tooth disease.



Figure 3 Large callosity, demonstrating significant overload under the lateral midfoot, which predisposes to 5th metatarsal stress fractures.

cavus foot' was coined by Manoli et al. in 2005² and has gained popularity in the literature to describe a less severe deformity, in the absence of a clear underlying neurological course.⁴ This has been used interchangeably with idiopathic cavus foot where no recognisable cause has been identified. Otherwise, the deformity is unilateral or bilateral and classified as neuromuscular, congenital or traumatic.³ Familial neurological causes are well recognized and more variants of Charcot-Marie-Tooth (CMT) disease otherwise known as Hereditary Sensorimotor Neuropathy (HSMN) are being identified with the passage of time and research. CMT/HSMN is one of the most common inherited neurological diseases⁵ and accounts for approximately half of the detectable lesions resulting in cavus foot deformity. Other neurological causes include Freidreich's ataxia (Figure 4), polio, spina bifida or any form of dysraphism, cerebral palsy or spinal tumour. A full neurological work-up is often necessary.³ An urgent spinal or neurosurgical referral is required if signs such as: rapid progression, unilateral disease, hyperreflexia, Hoffman's sign or clonus are noted, as these may identify a reversible spinal cord lesion.³

Congenital causes include the idiopathic or subtle cavus foot, clubfoot or arthrogryposis. Under treated clubfoot or recalcitrant variants and arthrogryposis result in feet that are difficult to improve non-operatively. 'Idiopathic' cavus may simply be our lack of knowledge of all underlying causes, or an extreme of the normal spectrum of foot character.

Trauma such as burns, compartment syndrome and crush injuries are common attributing factors to cavus foot deformities.

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