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Case Report

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Idiopathic pes calcaneocavus in a young adult: a rare developmental variant with radiographic confirmation

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ABSTRACT

Pes cavus refers to a spectrum of foot deformities characterized by an abnormally elevated medial longitudinal arch, with the calcaneocavus subtype defined by hindfoot dorsiflexion and a high arch being relatively uncommon and most often associated with underlying neuromuscular conditions such as Charcot–Marie–Tooth disease. Idiopathic presentations are rare, and potential developmental contributors remain poorly understood. We report a 19-year-old male with bilateral symptomatic calcaneocavus presenting with progressive forefoot and heel pain, clawing of toes, and functional limitation. Clinical examination revealed high medial arches, dorsiflexed hindfoot, and a rigid deformity as demonstrated by a positive Coleman block test. Weight-bearing radiographs confirmed calcaneocavus alignment, with Meary's angle of >8°, calcaneal inclination of 34°28′, and increased talo–first metatarsal angle. Comprehensive neurological evaluation, including detailed clinical examination and electromyography/nerve conduction studies, was normal. The patient had a notable history of prematurity at 34 weeks of gestation without associated neurological sequelae. Conservative management with custom orthoses and targeted physiotherapy focusing on Achilles tendon stretching and intrinsic muscle strengthening resulted in significant symptomatic improvement at 3-month follow-up, with reduced pain and improved functional capacity. This case represents a rare example of radiographically confirmed idiopathic bilateral calcaneocavus in the absence of neurological or known genetic pathology and introduces prematurity as a potential developmental factor influencing foot morphology. While this association tries to associate prematurity as a factor, the report contributes to the limited literature on non-neuromuscular causes of cavus deformity and highlights the need for further studies to better understand this etiological spectrum.

Keywords: Pes cavus, Pes calcaneocavus, Idiopathic foot deformity

INTRODUCTION

The Pes Cavovarus foot is a complex three-dimensional deformity characterized by an elevated medial longitudinal arch (pes cavus), plantarflexion of the first ray, forefoot pronation, and hindfoot varus. It is most commonly associated with underlying neuromuscular conditions, particularly Charcot–Marie–Tooth disease, cerebral palsy, and other hereditary motor–sensory neuropathies.[1,2] The pathophysiology typically involves muscle imbalance between agonist and antagonist groups, leading to progressive deformity, rigidity, and abnormal load distribution across the foot.[3] Consequently, identification of an underlying neurological etiology remains a central component in the evaluation of patients presenting with cavus deformity.[4]

Clinically, patients with cavovarus foot often present with chronic plantar pain, instability, recurrent ankle sprains, and callosities over pressure points, particularly beneath the first and fifth metatarsal heads. Radiographic assessment plays a crucial role in confirming the diagnosis

and assessing severity, with parameters such as Meary's angle, calcaneal pitch, and talocalcaneal angle providing objective evidence of cavus alignment and hindfoot involvement.[5] Despite the strong association with neurological disorders, a subset of patients presents with so-called idiopathic cavus foot, in which no underlying neuromuscular or systemic cause can be identified.

These cases are relatively uncommon and remain less well understood, posing diagnostic and etiological challenges. Furthermore, potential developmental contributors to such deformities have not been extensively explored in the available literature. In this report, we present a rare case of bilateral rigid cavovarus foot in a neurologically intact young adult with the history of the prematurity. This case highlights the importance of a comprehensive clinical and radiological evaluation, underscores the existence of non-neurological causes of cavus deformity, and introduces a potential developmental perspective that may contribute to the evolving understanding of its etiology.

CASE REPORT

Cavovarus foot is a three-dimensional deformity characterized by a high medial longitudinal arch (pes cavus), a plantarflexed first ray, forefoot pronation, and hindfoot varus. It commonly reflects an underlying neuromuscular imbalance (e.g. Charcot–Marie–Tooth neuropathy, cerebral palsy).

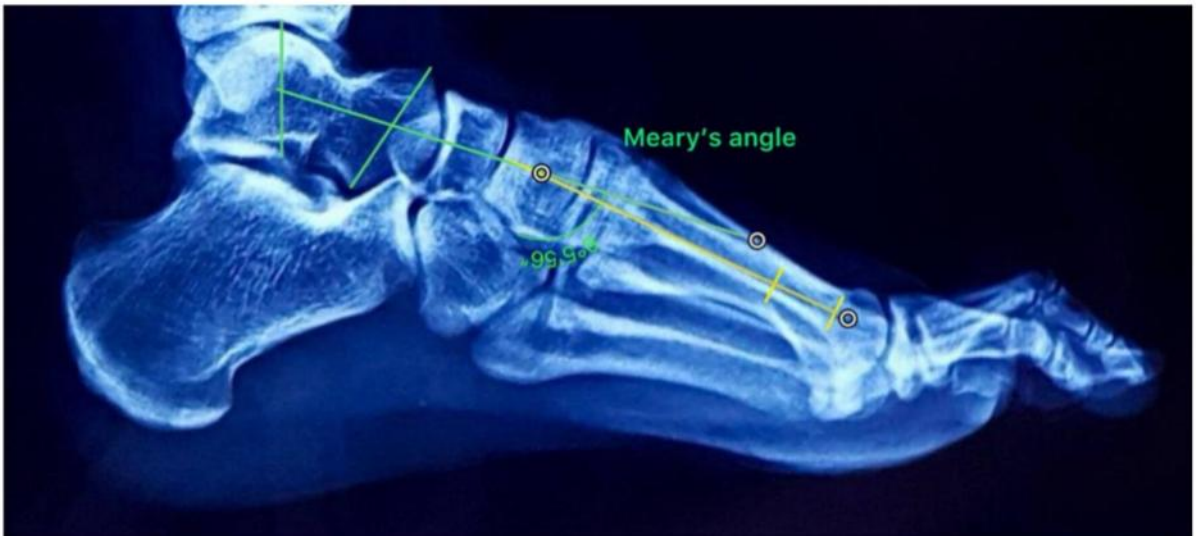

Figure 1: Weight-bearing lateral foot radiograph demonstrating a high medial arch and planter flexed first metatarsal. Meary's (talo-first metatarsal) angle on lateral view was +8º (normal~0º, cavus if >5).

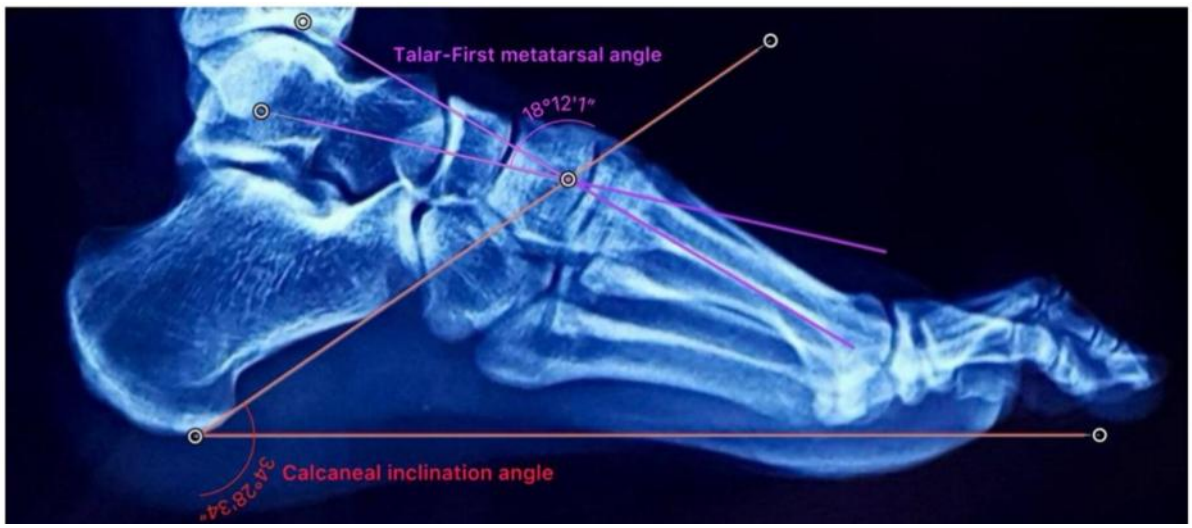

Figure 2: Weight-bearing lateral radiograph of a cavovarus foot (right), with lines drawn along the talus and first metatarsal. Calcaneal pitch (inclination of the calcaneus) was 34º (elevated: normal~20-30º, supporting a calcaneocavus component). The talar declination angle was decreased (consistent with plantarflexed forefoot). The calcaneal-first metatarsal (Hibb) angle was significantly increased, indicating a supinated hindfoot. On the lateral film the first metatarsal was plantarflexed (declination~18º).

However, a subset of patients has "idiopathic" cavus feet with no identifiable neurologic or systemic disease.[6] Patients typically present with chronic plantar foot pain (often under the first and fifth metatarsal heads), instability, and callosities at pressure points. We present a case of bilateral rigid cavovarus foot in a healthy 19-year-old male, emphasizing the clinical evaluation, radiographic findings, and relevant literature.[7,8]

A 19-year-old male presented with a 3-year history of gradually progressive, bilateral forefoot and heel pain aggravated by prolonged walking and stair climbing. He reported difficulty in sports and cosmetic concern about high arches and toe clawing. There was no history of trauma, neuropathic symptoms (e.g. burning dysesthesias), or systemic illness. The patient's perinatal history was notable for premature birth at 34 weeks cause being premature rupture of membranes and an 8-week neonatal ICU course for respiratory (CPAP) and feeding support; no central nervous system injury (e.g. intraventricular hemorrhage, periventricular leukomalacia) was documented. Developmental milestones were normal. There was no family history of neuropathy or foot deformity.

On examination, both feet had markedly elevated medial longitudinal arches and hindfoot varus with the heels tilted inward (ankle–hindfoot dorsiflexion). The lesser toes were clawed, and tender callosities were present under the first and fifth metatarsal heads (consistent with lateral overloading). The Coleman block test was performed (patient stands on a 2.5-cm block beneath the heel and lateral forefoot, allowing the first metatarsal to hang free). The hindfoot remained in varus when the block was used, indicating a rigid deformity (lack of correction of varus suggests hindfoot-driven cavus rather than flexible forefoot-driven cavus).[9]

Ankle dorsiflexion was mildly restricted bilaterally due to gastrocnemius–soleus tightness, but the deformity itself was in part hindfoot-driven. Subtalar and midfoot motion were reduced and appeared structurally fixed (not painful). Neurologic examination of the lower limbs was normal: muscle bulk and tone were normal; strength was 5/5 in all muscle groups (including tibialis anterior, peroneals, gastrocnemius); sensation (light touch, vibration) was intact; reflexes (patellar, Achilles) were symmetric and brisk, with no upper motor neuron signs. Gait analysis showed forefoot loading (due to high arch) but no evidence of foot drop or steppage. Other systems were unremarkable.[10]

Weight-bearing and non-weight-bearing foot radiographs (anteroposterior and lateral views) showed the following cavus deformity parameters: Meary's (talo–first metatarsal) angle on lateral view was +8° (normal ~0°, cavus if >5°). The calcaneal pitch (inclination of the calcaneus) was 34° (elevated; normal ~20-30°, supporting a calcaneocavus component). The talar declination angle was decreased (consistent with plantarflexed forefoot). The calcaneal–first metatarsal (Hibb) angle was significantly increased, indicating a supinated hindfoot. On the lateral film the first metatarsal was plantarflexed (declination~18°).[4,11] The AP view showed a narrowed talocalcaneal angle (parallel talus and calcaneus), consistent with hindfoot varus rigidity. No fractures, tarsal coalitions, or dysplasia were identified. (Figure 1 and Figure 2 illustrate a similar cavovarus radiographic pattern).

DISCUSSION

Pes calcaneocavus is classically understood as a manifestation of neuromuscular imbalance, wherein relative overactivity of intrinsic foot muscles and weakness of antagonistic groups result in hindfoot dorsiflexion and forefoot plantarflexion, producing a rigid high-arched foot. Multiple studies have consistently emphasized that the majority of cavus deformities are secondary to neurological etiologies, most notably Charcot–Marie–Tooth disease and other hereditary motor–sensory neuropathies. In a landmark review, Mann et al highlighted that bilateral cavus deformity in young individuals should be presumed neurological until proven otherwise.[12] Similarly, Burns et al demonstrated that even subtle cavus deformities may harbor underlying neuropathy detectable on electrophysiological testing.[6] In contrast to these established observations, the present case is notable for the complete absence of neurological involvement. The patient exhibited normal tone, power, reflexes, and intact sensation, with no upper motor neuron signs, and electrophysiological studies of all limbs (EMG/NCS) were unremarkable. This diverges from the prevailing literature, where idiopathic cavus is considered relatively uncommon. The findings in this case therefore align more closely with the smaller subset of reports describing structurally driven or idiopathic cavus deformities without detectable neuromuscular pathology.[2]

Radiographic parameters in this patient were consistent with previously described diagnostic criteria for cavus deformity. Increased Meary's angle, elevated calcaneal pitch, decreased talar declination, and reduced talocalcaneal angle have all been well documented as objective indicators of cavus alignment and hindfoot rigidity. These findings correlate strongly with clinical features such as clawing of toes, plantar callosities, and forefoot overload, as described in earlier radiographic analyses of cavus feet.[4,11] The rigidity demonstrated by a non-correctable Coleman block test further supports the structural nature of the deformity, distinguishing it from flexible variants often seen in early or neurologically driven cases. With respect to management, existing evidence supports an initial conservative approach in symptomatic cavus feet. A Cochrane collaboration review has shown that custom foot orthoses can significantly improve pain and functional outcomes in patients with pes cavus. Surgical intervention is typically reserved for progressive, rigid, or functionally limiting deformities, particularly in cases with an identifiable neuromuscular

etiology. The favorable early response to conservative treatment in this patient is consistent with prior reports suggesting that idiopathic cavus deformities may be effectively managed non-operatively when detected before severe structural progression.

A unique and noteworthy aspect of this case is the history of prematurity (34 weeks' gestation), which introduces a potential developmental consideration not well expressed in current literature. While prematurity has been associated with altered muscle tone, delayed motor patterning, and prolonged neonatal positioning, its relationship with long-term foot architecture, specifically the development of pes calcaneocavus remains unclear. Unlike prior studies that focus predominantly on genetic or neurological causes, this case raises the possibility that subtle developmental factors may contribute to structural foot deformities even in the absence of overt neurological injury. To date, no robust studies have established a direct association between prematurity and idiopathic calcaneocavus, making this observation hypothesis-generating.

Conservative management is supported by existing systematic review evidence, including a Cochrane analysis showing that custom foot orthoses improve pain and function in symptomatic cavus feet. Surgery is generally reserved for progressive, rigid or disabling deformities, particularly in neuromuscular cases. Our patient's favorable response aligns with published outcomes for idiopathic cavus managed non-operatively.

This case therefore contributes to the existing body of knowledge in several ways. First, it reinforces the importance of thorough neurological evaluation in cavus deformities while also demonstrating that true idiopathic cases do exist. Second, it highlights the role of radiographic parameters in confirming diagnosis and assessing rigidity. Third, and most importantly, it introduces prematurity as a potential factor in the pathogenesis of structural foot deformities. This may prompt future longitudinal and observational studies to investigate musculoskeletal outcomes in preterm infants, particularly in relation to foot biomechanics and arch development.

Limitations of this report include its single-case design, which limits generalizability. Genetic testing was not performed; however, the absence of clinical and electrophysiological evidence of neuropathy makes a hereditary cause unlikely. Additionally, the short duration of follow-up precludes conclusions regarding long-term progression or response to treatment.

The patient's history of 34-week prematurity introduces a speculative but intriguing developmental angle. Premature infants may experience altered muscle tone, prolonged NICU positioning and atypical early loading patterns, any of which could influence foot architecture. Although this patient had normal neurology and no neonatal CNS injury, prematurity may contribute subtly to musculoskeletal maturation. Existing literature does not describe a clear association between prematurity and idiopathic calcaneocavus, making this a noteworthy hypothesis for future observational studies.

CONCLUSION

This case highlights a rare presentation of idiopathic pes calcaneocavus in a young adult with completely normal neurological and electrophysiological evaluation, challenging the widely accepted association of cavus deformity with underlying neuromuscular pathology, particularly Charcot–Marie–Tooth disease. The favourable response to conservative management further supports existing evidence that non-operative treatment can be effective in structurally driven cases when identified early. Importantly, the coexistence of prematurity without overt neurological sequelae introduces a novel and underexplored developmental perspective in the pathogenesis of cavus deformity. Electromyography and nerve conduction studies were normal. Routine laboratory studies were unremarkable. Genetic testing was not pursued due to absence of clinical or electrophysiologic indicators of hereditary neuropathy. The patient was managed conservatively with custom-moulded orthoses providing medial arch support and lateral forefoot posting.

Physiotherapy emphasized Achilles stretching, intrinsic foot muscle strengthening and gait retraining. Footwear modification with cushioned, rocker-bottom shoes with heel modifications were recommended. At a 3-month review, the patient reported substantial pain reduction, increased walking distance and improved comfort during daily activities. Callosities had softened and gait appeared more plantigrade. Continued orthosis uses and exercises were advised, with periodic reassessment. By documenting this unique combination of findings, the present report expands current understanding of the etiological spectrum of pes calcaneocavus, underscores the need to consider non-neurological and developmental contributors, and provides a basis for future research exploring long-term musculoskeletal outcomes in preterm individuals.

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