Congenital aplasia of the heel pad

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DESCRIPTION

Congenital aplasia of the heel pad is a descriptive term for a rare anomaly of the foot noted at birth or within the first few years of life. The normal heel pad is a specialised structure that lies between the os calcis and plantar skin consisting of microchambers and macrochambers of adipose tissue divided by intricate fibroelastic septations.

The os calcis is the first tarsal bone to ossify between 20 and 24 weeks after birth, and maturation of the heel pad continues through adolescence. Thickness of the heel pad averages 18 mm, ranging from 14.4 mm to 24.5 mm. The foot is an organ of support and locomotion, with the heel pad acting as a shock absorber designed to alleviate compression and shear forces on the os calcis which it envelopes. On heel strike, 2.5 times the body weight goes through the heel pad during each step in the gait cycle. Absence of heel pad exposes the sole of the heel to undue pressure from os calcis and can lead to the development of callosities and intracallosal keratoma formation.

We present some interesting clinical photographs and plain radiographs of a 14-year-old boy who attended clinic with a painful limp and cosmetic deformity of his left foot since birth (figures 1 and 2). There was no history of high energy injury to the foot. Parents did not know of any other family members with similar features. The patient walked with a slight antalgic gait with minimal limb length discrepancy. His left heel had multiple callosities and was clearly smaller than the normal right foot. The os calcis could be felt immediately underneath the thickened skin and there was an absence of a normal soft heel pad. The hind foot was in varus with pes planus deformity. The ankle, subtalar and midfoot joint range of movements were equal to the right side. It was felt that the deformity of his foot was secondary to his adapted gait. Neurological examination was normal, with no sensory or motor deficit.

Foot and axial radiographs of both calcanei show a smaller size of the left os calcis which was underdeveloped with an altered shape. The soft tissue heel shadow was absent (figures 3 and 4).

We believe such a presentation has not been reported before. This anomaly can be confused with congenital vertical talus or congenital talo equino varus (clubfoot) but these have classical features and thus can be ruled out with clinical examination and appropriate radiography.

Figure 1 A: Standing clinical photographs of the patient with aplastic left heel. B: in supine position showing aplastic left heel.

Figure 2 Clinical photograph of both heels with the patient in prone position.

Figure 3 Plain radiograph of the os calcanei showing an absent left heel pad.

Featured goals of management were considered, which included prevention of direct pressure and injury to the sole of the heel with improvement in gait pattern. A careful consideration of surgical treatment with ‘autologous reconstruction flap surgery’ could be achieved with a vascularised myofasciocutaneous flap. This was discussed with the family, including complications such as painful surgical scar, viability of flap and loss of proprioception in the reconstructed heel. They declined surgery. Non-operative management using a custom-made orthosis with well-padded cushion heel ‘filler’ inserts to improve shock absorption and medial arch support was advised. This is being modified at regular intervals as growth of the foot progresses.

Learning points

► Congenital aplasia of the heel pad may be easily confused with other common congenital foot abnormalities such as congenital talo equino varus (clubfoot) or congenital vertical talus.
► It can be an unusual and rare cause of limp in adolescence.
► This complex condition is difficult to treat and footwear modifications may alleviate some of the symptoms.

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