Lateral heel pain

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Part 2

Diagnosis

Baxter's neuropathy/inferior calcaneal nerve compression, with calcaneal enthesophyte.

Discussion

Baxter's nerve originates from the lateral plantar nerve near the trifurcation of the posterior tibial nerve at the level of the medial malleolus [1–5]. It may also infrequently arise directly from the tibial nerve or at the common origin of the medial calcaneal nerve [5]. It then travels between the abductor hallucis and quadratus plantae muscles before turning laterally to pass anterior to the medial calcaneal tuberosity. From here, it courses between the quadratus plantae and flexor digitorum brevis muscle before innervating the abductor digiti minimi muscle [3, 4].

Two key sites have been identified for Baxter's nerve entrapments. The nerve is often compressed between the quadratus plantae and abductor hallucis muscle as it turns laterally. It is also commonly compressed as it passes anteriorly to the medial calcaneal tuberosity [1–3]. Compression occurs from either a hypertrophied abductor hallucis muscle, as often seen in long-distance runners, by thickened plantar fascia, inferior calcaneal enthesophytes, or as a result of

altered foot biomechanics and internal derangements leading to nerve stretching from the hypermobile foot [2, 3, 6, 7].

Diagnosis is achieved through the use of MRI, which is the preferred imaging modality [8, 9]. T1-weighted spin-echo or fast spin-echo sequences can characterize the nerve's anatomy along with its surrounding structures, while T2-weighted spin-echo or fast spin-echo sequences allow for better identification of edema within the nerve and its surrounding structures [8, 9].

As Baxter's nerve provides some motor innervations to the flexor digitorum brevis, quadratus plantae and abductor digiti minimi muscles, MRI changes of a denervated muscle can occur as early as 4 days after a traumatic nerve injury [9]. Neurogenic edema may be seen in the subacute phase while muscular atrophy with fatty infiltration may be seen in the chronic phase [9]. One of the hallmark features of Baxter's neuropathy is fatty infiltration and atrophy of the abductor digiti minimi muscle as seen in Fig. 1 of the case presentation [2–4, 6, 7]. One may also find abductor hallucis muscle hypertrophy, a calcaneal enthesophyte (Fig. 2 of the case presentation), surrounding soft tissue edema (Fig. 1 and Fig. 3 of the case presentation), and potential confounding signs of plantar fasciitis on MRI [7].

One study suggests that Baxter's neuropathy may account for an upward of 20 % of heel pain [3]. As Baxter's nerve provides sensory innervation to the long plantar ligament and calcaneal periosteum, patients with Baxter's neuropathies typically present with medial heel pain that is often difficult to distinguish from plantar fasciitis [2, 3, 6, 7]. Other symptoms may include paresthesia along the lateral third of the sole of the foot and the characteristic abductor digiti minimi muscle weakness [2, 3]. The differential diagnosis includes plantar fasciitis, Achilles tendinosis, painful heel pad syndrome, bone tumors, heel spur fracture, calcaneal stress fracture, and bursitis among others [3, 5].

Overall, conservative treatment is often sufficient for Baxter's neuropathy and is nearly identical to the treatment

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of plantar fasciitis. Treatment is primarily focused on reducing the aggravating factors, immobilization, use of anti-inflammatories, orthotics and footwear, and potentially physiotherapy [1, 3, 8]. Surgery is rarely warranted and is reserved only for refractory cases [3].

Conflicts of interest None.

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