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Transient morton's toe: An uncommon presentation of a common clinical condition

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Abstract
Herpes zoster (Shingles) is a result of reactivation of the Varicella-zoster virus (VZV) in dorsal root ganglia (DRG) after a long latency period following a primary infection (chickenpox). A Morton's toe or neuroma is an enlarged common plantar nerve, usually in the third web space. Our patient, a 52 year old male, presented with an initial clinical and radiological picture of Morton's toe. He however went on to develop characteristic vesicular rash of Herpes zoster over L5 dermatome within 48 hours of onset of forefoot pain signifying a temporal association which responded dramatically to antiviral medication. The clinical features mimicking Morton's toe were probably due to the already compromised/ scarred inter digital space secondary to an old injury. This peculiar case, depicting a clinical presentation of Herpes zoster in the prodromal phase, has not been reported in literature till date. Knowledge of such an association would help clinicians in anticipating Herpes zoster as one of the differential diagnoses in a case of Morton's toe and manage it accordingly. The rapid and substantial recovery in the patient's forefoot symptomatology along with skin lesion following the initiation of antiviral therapy highlights the fact that the clinically apparent Morton's toe was a manifestation of underlying Herpes zoster rather than a true inter digital neuroma.

Keywords: herpes zoster, morton's neuroma, uncommon presentation

Introduction
Varicella-Zoster virus (VZV) causes two distinct clinical entities: Varicella (chickenpox) and Herpes zoster (Shingles). Herpes zoster is characterized by a unilateral vesicular eruption within a dermatome, often associated with severe pain [1, 2]. Herpes zoster (Shingles) is a result of reactivation of the Varicella-zoster virus (VZV) in DRG after a long latency period following primary infection (chickenpox) [3]. The onset of disease is heralded by pain within a dermatome, which may precede skin lesions by 48–72 hours; an erythematous maculopapular rash that rapidly evolves into vesicular lesions. These lesions may remain few in number and continue to form for about 3–5 days with the total duration of disease being 7–10 days; however, it may take as long as 2–4 weeks for the skin to return to normal. The prodromal symptoms which occur prior to the onset of skin eruptions include pain, flu like symptoms, lassitude, stressed feel and depression [4]. Post-infective complications or residual symptoms include post-herpetic neuralgia, segmental zoster paresis, post-herpetic foot drop, peripheral neuropathy, zoster ophthalmicus, Ramsay Hunt syndrome etc. The continuum of pain from onset to resolution is known as *zoster-associated pain*. Although *zoster-associated pain* has a spectrum of presentation, there have not been any reported cases of Herpes zoster presenting in the form of a Morton's toe/Morton's metatarsalgia in the prodromal phase till date. We herein report a rare case of Herpes zoster in a middle-aged healthy male with initial clinical presentation mimicking Morton's toe-a characteristic painful debilitating condition of the forefoot.

Materials and Methods
A 52 year old male patient from East Africa presented to the outpatient department with a chief complaint of pain in his right forefoot of one day duration. The pain was acute in onset, localized to the 3rd web space and cramping in character. The pain was severe enough to disturb his night sleep and aggravated with standing/ walking and was partially relieved with

rest. The patient subjectively complained of numbness in adjoining areas of 2nd and 3rd toes. There was no history of recent trauma or fever and the patient was not a known diabetic or hypertensive. The patient had a past history of fracture involving the forefoot, 20 years ago which healed with conservative management and was asymptomatic thereafter. He gave no history of smoking/tobacco chewing or long term medications or any similar complaints in the past. General physical examination of the patient was unremarkable and local examination of the right foot revealed a localized tenderness over the 3rd web space, more so, on the plantar aspect of foot without any local signs of swelling, erythema, warmth or deformity. Mulder's sign was positive with replication of pain on compression of the forefoot between the finger and thumb so as to compress the transverse arch of the foot; however no palpable or audible click was appreciated. There were no objective signs of sensory loss or motor weakness distally and the capillary refill time was less than 2 seconds.

On laboratory evaluation, ESR and CRP were normal. The patient had normal haematological and biochemical parameters with blood sugars levels being within normal. Radiographs of the forefoot, dorso-plantar and oblique views showed an old fracture of head of proximal phalanx of 3rd toe with post traumatic arthrodesis of the proximal interphalangeal joint of 3rd toe. Ultrasonography of forefoot showed relative thickening of the interdigital nerve of the 3rd web space with perineural edema. Being a peripheral hospital with limited resources, MRI and nerve conduction studies could not be done. Based on the above findings, a clinical diagnosis of Morton's toe was made and he was managed on an outpatient basis with analgesics and a metatarsal pad. Two days later the same patient again reported to the outpatient clinic with painful skin rash over his right leg. Clinically, he had vesicular skin eruptions distributed along the anterolateral aspect of right leg (L5 dermatome), typical of Herpes zoster with no motor or sensory deficits. Viral markers for Human Immuno-deficiency Virus were tested and found to be negative. The patient was started on oral Acyclovir 800mg five times daily for 5 days along with topical calamine lotion and analgesics. The lesions healed to form scabs within 7 days of antiviral therapy with remarkable decrease in the pain involving the skin lesions as well as the forefoot pain. On follow up visit at 2 weeks post onset of symptoms, the patient was asymptomatic with no pain either in forefoot or the healed skin lesions and there were no subjective or objective sensory/ motor deficits in his limbs. He did not require any further intervention for his forefoot pain.

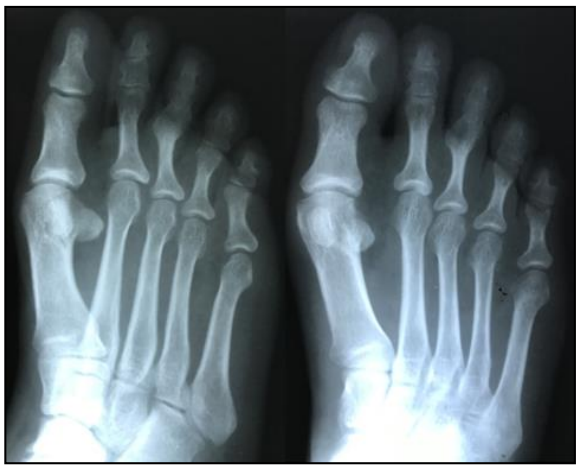

Fig 1: X-ray Right fore foot – Dorsoplantar and oblique views

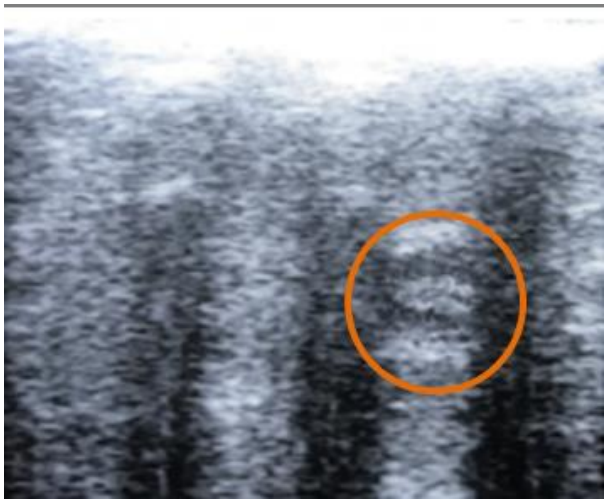

Fig 2: Ultrasonography of intermetatarsal space – right foot

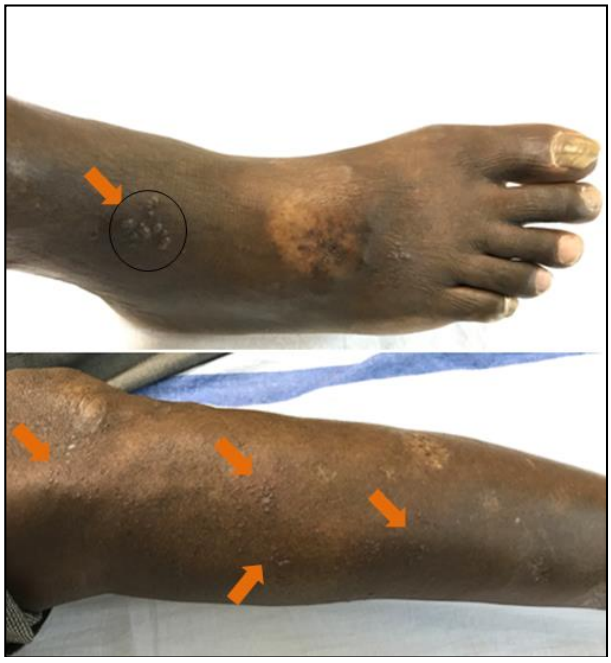

Fig 3: Vesicular lesions over anterolateral aspect of right leg (L5 dermatome)

Discussion

Herpes zoster occurs at all ages, but its incidence is highest among individuals in the sixth decade of life and beyond [5]. Pathogenesis of Herpes zoster involves local reactivation of latent VZV in DRG secondary to either a modification in the pathogenicity of the virus or a weakening in the host's immune defences, most notably those mediated by T cells [6, 7]. The reactivated virus travels antegrade along the peripheral process of the pseudo motor neuron of DRG to reach the targets of innervation including the skin to cause the characteristic lesion. Histopathological examination of representative neural tissue during active Herpes zoster demonstrates haemorrhage, oedema, and lymphocytic infiltration [5].

The medial plantar nerve (L4, L5) innervates adjacent surfaces of the medial three and half toes through common plantar digital nerves. A Morton's toe or neuroma is an enlarged common plantar nerve, usually in the third interspace. In this region of the foot, the lateral plantar nerve often unites with the medial plantar nerve and as the two nerves join, the resulting nerve is typically larger in diameter than those of the other toes [8]. Our patient had a slightly

thickened common plantar nerve with perineural edema on ultrasound which corroborates with the pathological changes of neural tissue affected by Herpes zoster. However, although the medial plantar nerve receives fibres from the L5 nerve root, we did not find any herpetic lesion in the skin supplied by this nerve. The skin lesions were predominantly distributed over the anterolateral aspect of leg (L5 dermatome). This preferential distribution of skin lesions may be explained by the fact that the common digital nerve is a branch of medial plantar nerve which is a mixed nerve with fibres from both L4 and L5 as compared to the L5 predominant sensory distribution along anterolateral aspect of leg. The clinical features mimicking Morton's toe were probably due to the already compromised/ scarred interdigital space secondary to old injury in the region. The possibility of a subclinical interdigital neuroma which got clinically apparent with the onset of Herpes zoster cannot be totally ruled out.

The rapid and substantial recovery in the patient's forefoot symptomatology along with skin lesion following the initiation of antiviral therapy highlighted the fact that the clinically apparent Morton's toe was a manifestation of underlying Herpes zoster than a true interdigital neuroma. This case is peculiar since such a clinical presentation of Herpes zoster in the prodromal phase has not been reported in literature till date. Knowledge of such an association would help clinicians in anticipating Herpes zoster as one of the differential diagnoses in a case of Morton's toe and manage it accordingly.

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