

A Case of Malum Perforans Pedis Complicated by Chronic Osteomyelitis

Malum perforans pedis (MPP) is a chronic trophic ulceration of the sole, resulting from chronic denervating diseases accompanied with constant trauma at the pressure bearing areas. Rarely, MPP is caused by spinal cord injury complicated by chronic osteomyelitis.

A 40-year-old woman presented with a deep punched out ulceration on the left heel with an eighteen-year history of recurrent serosanguineous followed by purulent exudate and a ten-year history of deep abscesses, draining sinuses, and subsequent tumefaction of the sides of the heel. Two years prior to the development of the asymptomatic ulceration, a lower lumbal vertebral fracture with bilateral paresthesia in S-1 and S-2 distribution occurred, while motor functions were unimpaired.

Physical examination revealed a large triangular shaped punched out deep ulcer on the undersurface of the left heel (Figure 1). The ulcer had yellowish hyperkeratotic margin, flesh colored moist base, and distortion and tumefaction of the sides of the heel (Figure 2). The skin was tender and fluctuant over some points, with a few sinuses of purulent discharge. *Pseudomonas aeruginosa* was isolated in the culture of the exudate from the ulcerated lesion and the draining sinuses. Radiographic images showed erosion of the undersurface of the calcaneus (Figure



Figure 1. Deep perforated ulcer of the heel with surrounding callosity.

3). The rest of the physical examination and routine blood and urinalyses were unremarkable.

Based on the history, physical examination, and radiographic findings, diagnosis of MPP complicated by chronic osteomyelitis was made. The culture of the debrided tissue grew *Staphylococcus* and *Pseudomonas aeruginosa*; appropriate antibiotics were administered for six weeks. The orthopedic surgeon decided on a sural neurofasciocutaneous flap to cover the wound.

MPP or neurotrophic or perforating ulcer of the foot is a chronic trophic ulcer in chronic denervating diseases such as tabes dorsalis, arteriosclerosis, leprosy, diabetes mellitus, ankylosing spondylitis, spinal canal stenosis, spondylolisthesis, radiation injury of the lumbosacral plexus, intrathecal tumor, and Bureau-Barrière syndrome accompanied with constant trauma particularly at the pressure bearing areas (1-7). Tabes dorsalis and arteriosclerosis affect the posterolateral tracts of the spinal cord, resulting in loss of pain sensation of the acral areas. Syringomyelia disrupts the nerve pathways of the lateral tracts.

In our patient, MPP developed as a result of spinal cord injury in the S1 and S2 distribution with the loss of pain sensation.



Figure 2. Grossly deformed sides of the heel due to tumefaction and recurrent deep abscesses and sinuses.



Figure 3. Radiograph of the heel showing markedly eroded undersurface of the calcaneus.

MPP typically begins as an area of well-demarcated hyperkeratosis at the ball of the foot or the undersurface of heel (1). The lesion softens and turns into a malodorous, moist area exuding a purulent discharge with an indolent, necrotic painless deep ulcer. Neuropathy, due to the underlying disease process, renders the ulcer painless and the patient continues to walk, resulting in callus formation around the ulcer. The ulcer typically does not heal with conventional therapies employed for chronic wounds and is often complicated with secondary bacterial infection. In our case, osteomyelitis of the tarsal bones had occurred. Differential diagnosis of MPP includes diabetic foot and ulcers caused by suppurating corns or calluses.

The underlying cause should be discovered by carefully analyzing the patient history and a thorough physical examination. The treatment of the underlying disorder should be combined with general measures to relieve pressure on the ulcer, such as padding and keeping pressure off the foot as much as possible. Total contact casting involving reducing plantar pressures, debridement of the surrounding callus, and platelet-derived growth factor applied once daily has been shown to promote healing combined with protection of the foot if the claw-like deformities have already developed (8,9,10).

In all cases, local and systemic antibiotics are needed to treat secondary bacterial infection along with an extensive debridement if complicated by osteomyelitis.

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Shahbaz A. Janjua¹, Zrinjka Pastar²

¹*Ayza Skin & Research Center, Lalamusa, Pakistan*

²*Ministry of Defense Republic of Croatia, Zagreb, Croatia*

Corresponding author:

Shahbaz A. Janjua, MD
Ayza Skin & Research Center
Lalamusa
Pakistan
shahbaz.janjua@telederm.org

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