

From the diabetic foot ulcer and beyond: how do foot infections spread in patients with diabetes?

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A diabetic foot infection is usually the result of a pre-existing foot ulceration and is the leading cause of lower extremity amputation in patients with diabetes. It is widely accepted that diabetic foot infections may be challenging to treat for several reasons. The devastating effects of hyperglycemia on host defense, ischemia, multi-drug resistant bacteria and spreading of infection through the foot may complicate the course of diabetic foot infections. Understanding the ways in which infections spread through the diabetic foot is a pivotal factor in order to decide the best approach for the patient's treatment. The ways in which infections spread can be explained by the anatomical division of the foot into compartments, the tendons included in the compartments, the initial location of the point of entry of the infection and the type of infection that the patient has. The aim of this paper is to further comment on the existed and proposed anatomical principles of the spread of infection through the foot in patients with diabetes.

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Diabetic foot infections in the presence of peripheral neuropathy and/or vascular compromise can have devastating results in the patient's overall treatment. A diabetic foot ulceration as a consequence of neuropathy, peripheral arterial disease or both is frequently the point of entry of the bacteria into the foot. Penetrating trauma can also inoculate the infection into the foot and puncture wounds have a high risk of leading to osteomyelitis (1). The deleterious effect of hyperglycemia on neutrophils (2), has also been recognized as immunopathy, and may have a role in the severity of the infection. The treatment of diabetic foot infections is complex and should be based on a stepwise approach (3, 4). The initial evaluation of the type of infection (5) and its severity are key points in order to decide the best management for each patient to achieve limb salvage. In addition to culture-directed antibiotic therapy, most infections require surgical inter-

vention, ranging from minor surgical debridement to major resection, amputation or revascularization (6). Some of the best outcomes in treating patients within these complex pathologies are achieved when teams including and not limited to experienced health care professionals, physicians and surgeons perform aggressive revascularization to treat these challenging patients (3, 6–8).

The surgeon who treats patients with diabetes and foot infections should have a thorough knowledge of the compartmental anatomy of the foot and also be able to identify the ways in which infections can spread through the diabetic foot (5, 9). In many cases, staged reconstructive surgical procedures may be necessary in order to save the infected diabetic foot (10) and revisional surgeries should be based on the same anatomical principles. The purpose of our perspective article is to analyze the ways in which infections can spread through the diabetic foot.

Anatomical principles

It is very important to understand that the foot is divided into rigid compartments. This has two implications for the surgeon:

1. Compartmental pressure may increase as a consequence of the infection and tissue damage may be more extensive than expected. Ischemic necrosis will add to the damage promoted by bacteria and host defenses. The compartment that is affected by the infection should be exposed in an efficient and expedited way in order to diminish the compartmental pressure.
2. The surgeon needs to be aware and have a high index of suspicion regarding the initial entry point of infection.

Figure 1 demonstrates a clinical view of the plantar aspect of the foot in a diabetic patient with necrosis of the fourth toe and the division of the foot into three plantar compartments. The floor of the compartments is the rigid

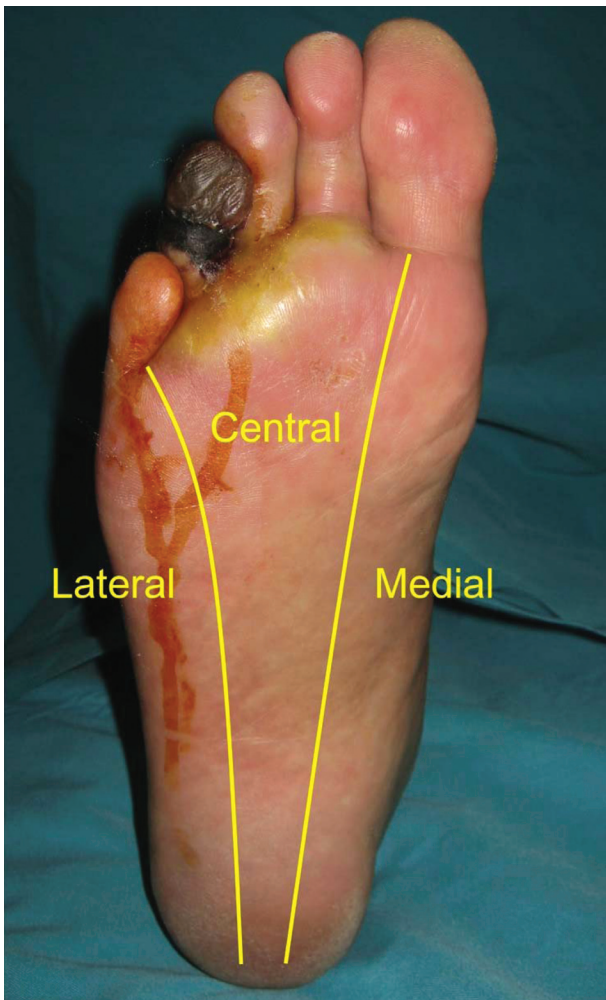


Fig. 1. Plantar compartments of the foot.

plantar aponeurosis which is attached to the calcaneus and extends distally to the toes (Fig. 2, in blue). The plantar aponeurosis is the outermost fascia and represents the anatomical layer located beneath the subcutaneous tissue. The medial and central plantar foot compartments are separated by the medial intermuscular septum, which extends from the medial calcaneal tuberosity to the first metatarsal head. The central and lateral compartments are separated by the lateral intermuscular septum, which extends from the calcaneus to the fifth metatarsal head (Fig. 1, yellow lines). The medial compartment contains the flexor hallucis brevis, abductor hallucis and flexor hallucis longus tendons. The central compartment contains the flexor digitorum brevis, lumbrical muscles, flexor digitorum longus tendons and quadratus plantae muscle. The lateral compartment contains the flexor digiti minimi brevis and abductor digiti minimi muscles. The interosseous compartment is

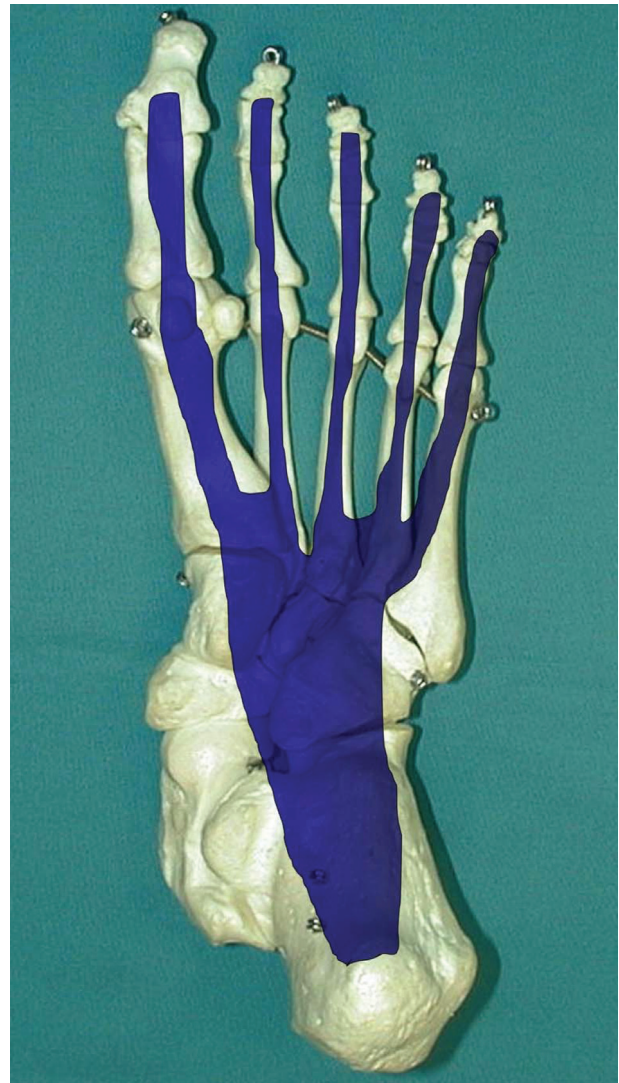


Fig. 2. Plantar aponeurosis.

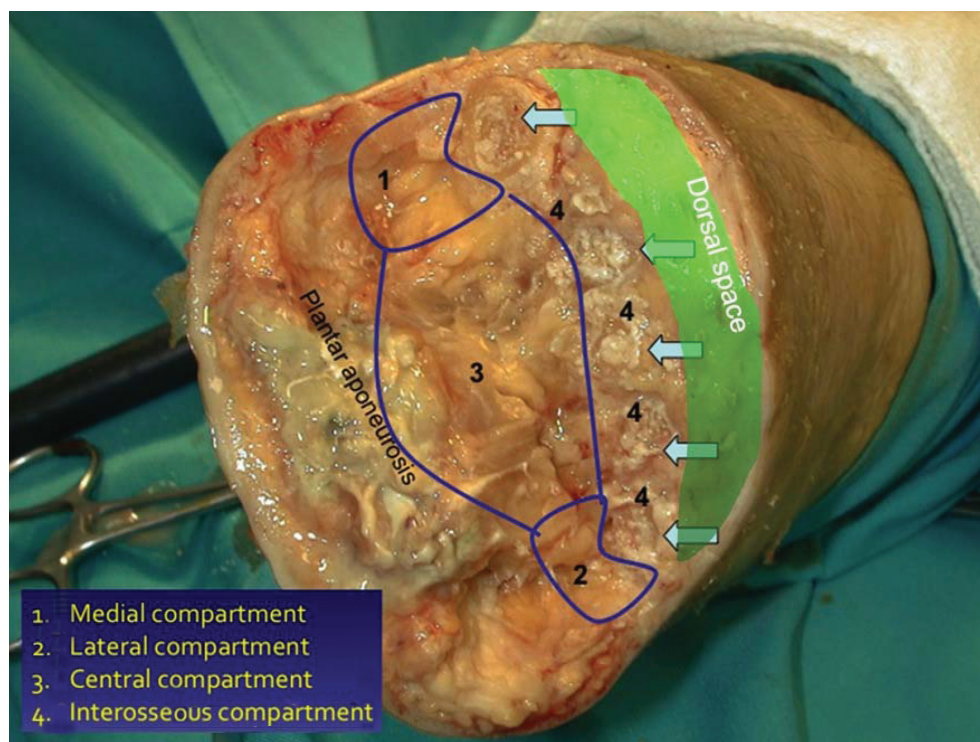


Fig. 3. Cross-section of the compartments of the foot.

located between the metatarsal bones and contains the interossei muscles. Figure 3 demonstrates a cross-section of the compartments of the foot (note that the blue arrows show the metatarsal bones). Currently, there is an on-going debate as to the exact anatomical location and number of foot compartments (11). It has been argued that the central compartment has two sub-compartments: superficial and deep or 'calcaneal' section (12) – though this subdivision was not recognized by other authors (13).

The dorsal compartment (14, 15) is also very important in some types of diabetic foot infections. The dorsal space has a thin layer of subcutaneous tissue and the tendons

contained in this space can be easily exposed in the presence of an ulceration. The tendons of the great toe are the extensor hallucis longus and extensor hallucis brevis. From the second through the fifth toes, the tendons are the extensor digitorum longus and extensor digitorum brevis.

It has recently been reported that a transversal forefoot space through which infections can spread may also be found (5). This space is located between the superficial transverse metatarsal ligament (deeper) and the superficial layer of the digital band of the aponeurosis. It contains fibrous septa forming loculations occupied by



Fig. 4. Transverse forefoot compartment. (A) Changes in color (green arrows) suggest involvement of the compartment. (B) Compartment surgically resected.

smooth areolar tissue. Figure 4 demonstrates a common example of a diabetic foot infection through this transverse forefoot space. This patient was admitted to our hospital and was initially presented with critical ischemia, peripheral edema, severe pain, leukocytosis and necrosis of the fourth toe. Changes in skin color in the plantar forefoot space (Fig. 4A, green arrows) and pain when palpated the affected area were detected during the initial physical examination. Emergent partial fourth ray (metatarsal and toe) amputation with exploration and surgical debridement of the transverse space, which was filled with purulent drainage, were performed (Fig. 4B).

How do infections spread in the diabetic foot?

Infections spread through the foot along the tendons and their sheaths. Tendons are included in the compartments and they are poorly vascularized structures. For this reason, infections arising in the toes can compromise the entire foot structures. Involvement of the flexor tendons, which are included in the central compartment, are demonstrated in Fig. 5 (blue arrows). Tendons involved in infections are broad, thickened, edematous and purulent on some occasions. Instrumental exploration of the central compartment should be carried out, as demonstrated in Fig. 6. On other occasions, the fascia and tendons become frankly necrotic (Fig. 7) and emergent surgery is necessary for a successful limb salvage outcome. In such cases, necrotizing soft tissue infections, necrotizing fasciitis and/or necrotizing tenosynovitis need to be diagnosed and treated in an expedited manner.

Physical examination of the infected diabetic foot is crucial in order to identify the point of bacterial entry which is frequently attributed to the presence of an ulcer. The location of the ulcer, whether it is plantar, dorsal, medial, lateral or interdigital, must be defined and examined in detail. In addition, clinicians should also have a high index of suspicion in tendinous infectious

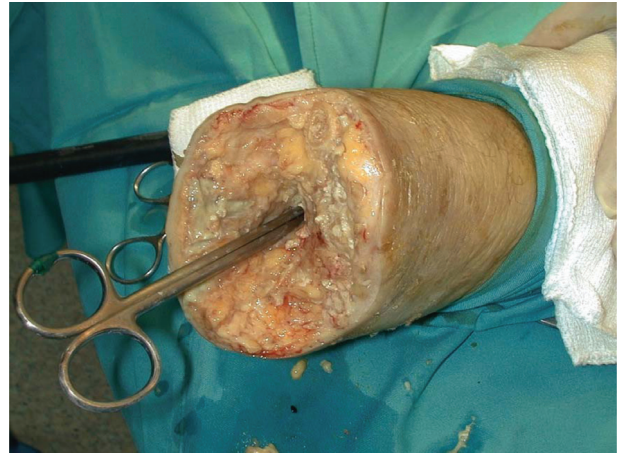


Fig. 6. Instrumental exploration of the central compartment.

process when the tendon is located in close approximation to the infected ulcer. For example, the flexor hallucis longus tendon is located between the sesamoid bones underneath the first metatarsal head and in the presence of sesamoid osteomyelitis, inflammatory changes to the tendon is a frequent result. This case scenario is demonstrated in Fig. 8 with a diabetic foot infection underneath the plantar aspect of the first metatarsal head and a history of sesamoid osteomyelitis.

Suppurative inflammation and drainage through the ulcer while palpating at a location distant from the ulcer is consistent with a spreading infection, and the clinician should always take into consideration the pathway of the affected tendons. The patient demonstrated in Fig. 9 had an ulcer in the proximal interphalangeal joint of the second toe. She had undergone a drainage procedure at another facility and sought consultation in our department due to the infection spreading and severity. The red arrow shows the point of entry of the infection. When the dorsum of the foot was palpated in the area of the green arrow, purulent drainage was increased. In such

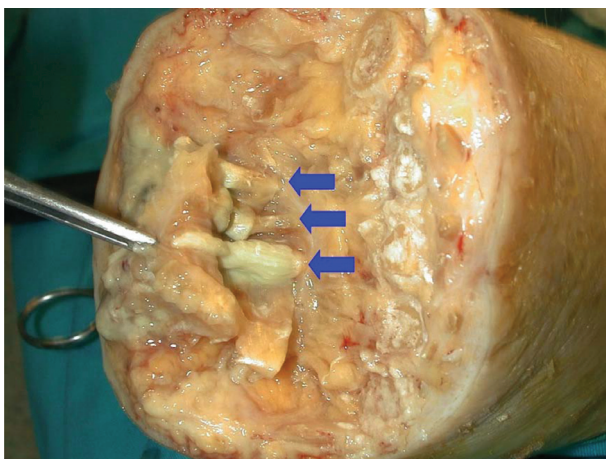


Fig. 5. Flexor tendon involvement (blue arrows) in the central compartment.



Fig. 7. Necrosis of the plantar aponeurosis and flexor tendons.



Fig. 8. Flexor hallucis longus involvement in a case of sesamoid osteomyelitis.

cases, the clinician should always suspect extensor tendon involvement with eventual resection and extensive debridement as it was performed for this patient (Fig. 10).

It is also suggested that infection is most commonly spread from a higher to a lower pressure areas of the diabetic foot. For example, an infected plantar neuropathic ulceration upon weight bearing status of the foot can spread the infection to the dorsal structures of the affected foot. However, it is less common that infections arising on the dorsal aspect of the foot to spread plantarly since the dorsum of the foot is not usually experiencing a high pressure area.

In cases where plantar neuropathic forefoot ulcerations are complicated by osteomyelitis of the involved metatarsal heads, the joint capsule is violated and purulence may commonly drain to the dorsum of the foot. Figure 11 demonstrates a plantar neuropathic ulceration underneath the third metatarsal head with purulent drainage and osteomyelitis of the affected



Fig. 9. Spread of the infection along the extensor tendons.



Fig. 10. Surgical debridement of the dorsum of the foot.

metatarsophalangeal joint in radiographic findings (Fig. 12, encircled). Suppurative inflammation and fluctuance was evident on the dorsum of the foot and purulence was drained through a dorsal incision (Fig. 13).

Diabetic foot infections can also spread from the plantar to the dorsal aspect of the foot through the interosseous compartment. In such cases, severe infection and necrosis to the plantar structures of the foot due to the high pressure achieved in the central compartment is seen. When pressure into the affected compartment is higher than the associated capillary pressure, necrosis usually appears. Deep infection of the diabetic foot produces neutrophilic vasculitis of the digital arteries, subsequent thrombosis and necrosis of the involved toe. Figure 14 demonstrates a plantar neuropathic ulceration underneath the third metatarsal head with necrosis of the third toe and a high pressure in the central compartment. The dorsal aspect of the foot is demonstrated in Fig. 15. An open transmetatarsal amputation and extensive debridement of the central compartment



Fig. 11. Plantar ulcer with suppuration.



Fig. 12. Osteomyelitis of the third metatarsophalangeal joint (encircled).



Fig. 15. Pre-necrotizing changes and suppuration of the dorsum of the foot.



Fig. 13. Purulent material drained through a dorsal approach.

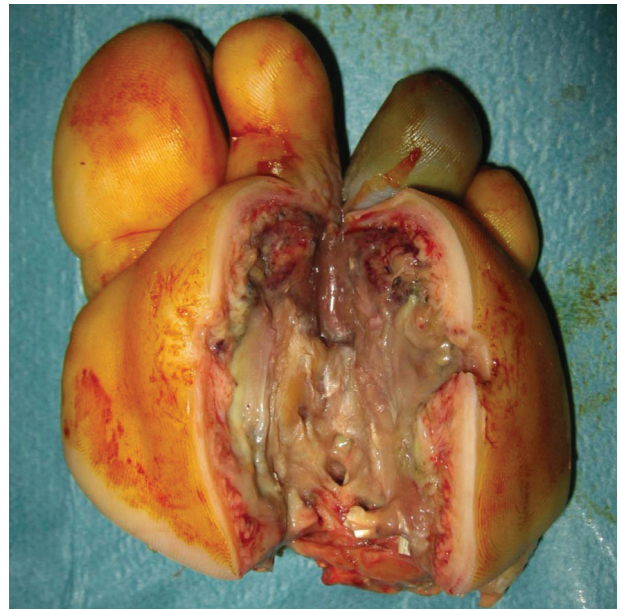


Fig. 16. Specimen showing severe destruction of the interosseous compartment.



Fig. 14. Neuropathic plantar ulceration with necrosis of the third toe.

were required to control the severity of the infection. Severe destruction of the interosseous compartment is seen in Fig. 16.

Conclusion

The pathways of which diabetic foot infections can spread are explained by the anatomical division of the foot into compartments, the tendons included in the compartment, the initial location of the entry point of infection and the type of infection that the patient has. Clinicians should be alert to these considerations when the patient is first presented with a diabetic foot infection.

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