

Diabetic foot infection: Dispelling the myths



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The development of diabetic foot infection is a highly significant staging post on the road to amputation. Although amputation may result from severe ischaemia or gross deformity of Charcot's osteoarthropathy, this is rare, and infection is usually the final common pathway to amputation.

However, efficient management of diabetic foot infections is hindered by lack of understanding. I have expressed such misunderstanding in 10 myths of diabetic foot infection to encourage debate and provoke discussion with an overall aim to improve diabetic foot care.

Myth 1: Diabetic foot infections always present with the classical signs of local infection

WRONG! Erythema and pain may be absent. The reason for this is that neuropathy leads to a diminished axon reflex and failure of vasodilatation. Furthermore, ischaemia also leads to absence of erythema in the ischaemic limb. Immunopathy results in limited abscess formation.

Myth 2: Diabetic foot infections always present with the classical signs of systemic infection

WRONG! Often there is no leucocytosis and fever. Leucocytosis is a poor indicator of acute osteomyelitis of the foot in diabetes mellitus: 54% of patients with acute osteomyelitis had normal white blood cell counts in a study by Armstrong et al (1996).

Myth 3: Diabetic foot infections rarely present with gangrene

WRONG! Infection proceeds rapidly to wet gangrene. This is a characteristic feature of the diabetic foot. Wet gangrene is caused by infection complicating a digital, metatarsal or heel ulcer, and leads to a septic vasculitis of the digital and small arteries of the foot. The walls of these

arteries are infiltrated by polymorphs, leading to occlusion of the lumen by septic thrombus.

Myth 4: Diabetic foot infections are caused only by Gram-positive bacteria or anaerobes

WRONG! Gram-negative bacteria also contribute. The microbiology of the diabetic foot is unique. Infection can be caused by Gram-positive aerobic, Gram-negative aerobic and anaerobic bacteria, singly or in combination.

Myth 5: Diabetic foot infections do not always need investigation with wound swabs and tissue samples

WRONG! Rational antibiotic therapy is dependent on identification of the infecting bacteria. Ulcer swabs and tissue samples indicate the presence of bacteria that may progress from colonisation to active infection and need targeted antibiotic therapy. Deep swabs or tissue should be taken from the ulcer after initial debridement and if the patient undergoes operative debridement then deep tissue should also be sent for analysis.

Myth 6: In the initial empirical treatment, antibiotics should cover only Gram-positives and anaerobes

WRONG! Cover Gram-negatives as well. At initial presentation, it is important to prescribe a wide spectrum of antibiotics because it is impossible to predict the organisms from the clinical appearance. As there is a poor immune response of the diabetes patient to sepsis, even bacteria regarded as skin commensals may cause severe tissue damage. This includes Gram-negative organisms such as *Citrobacter*, *Serratia* and *Pseudomonas*. When Gram-negative bacteria are isolated from an ulcer swab they should not be regarded

automatically as insignificant, especially in the diabetic neuroischaemic foot.

Myth 7: In follow-up treatment, broad-spectrum antibiotics should be given

WRONG! Target therapy. When specific organisms are isolated from reliable specimens, it is advisable to focus therapy with appropriate advice from the microbiologist or infectious disease physician.

Myth 8: Diabetic foot infections can be treated with antibiotics alone and rarely need surgery

WRONG! The diabetic foot often needs the great surgical macrophage, i.e. the surgeon! The definite indications for urgent surgical intervention are:

- Large area of infected sloughy tissue
- Localised fluctuance and expression of pus
- Crepitus with gas in the soft tissues on X-ray
- Purplish discolouration of the skin, indicating subcutaneous necrosis.

Myth 9: Osteomyelitis must be always treated with surgery

WRONG! Modern antibiotics with good bony penetration may cure osteomyelitis. Parenteral therapy has in the past been given for four to six weeks, followed by oral therapy for six weeks. It may be possible to limit the parenteral therapy to two weeks and follow this with appropriate oral antibiotics. However, if the ulcer persists after three months' treatment, with continued probing to bone which is fragmented on X-ray, resection of the underlying bone may be carried out. This may entail toe amputation or removal of the metatarsal head.

Myth 10: Amputations from diabetic foot infections are inevitable

WRONG! Early aggressive, targeted antibiotic treatment can save toes and limbs. It is important to have a practical approach which can diagnose infections early, treat them rapidly and aggressively, and, by this means, amputations are not inevitable. ■

Armstrong DG, Lavery LA, Sariaya M, Ashry H (1996) Leukocytosis is a poor indicator of acute osteomyelitis of the foot in diabetes mellitus. *Journal of Foot and Ankle Surgery* **35**(4):280–83

Further reading

International Working Group on the Diabetic Foot (2003) Supplement to the International Consensus on the Diabetic Foot: *Consensus on Diagnosing and Treating the Infected Diabetic Foot*. International Working Group on the Diabetic Foot. See www.iwgdf.org/consensus/infected_df.htm (accessed 22.02.05)