Linking risk factors: the role of history in predicting outcome

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ARTICLE POINTS

1 Determining a patient's position along the risk spectrum can provide useful information about future healing or complications.

2 Risk assessment should include a consideration of several factors, including family history and history of micro- or macrovascular disease.

3 Patient history will provide a high index of suspicion for vascular disease prior to examination.

5 Risk factors associated with diabetes are dynamic. Therefore, constant evaluation and treatment are important.

KEY WORDS
- **Risk factors**
- **Examination**
- **Patient history**
- **Peripheral vascular disease**

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Introduction

When a patient with diabetes presents after a minor traumatic event such as an abrasion, foot wear irritation, or inter-space infection, can you predict the outcome based on history and exam? It is important to know where the patient is along the risk spectrum of peripheral vascular disease and what effect this will have on their healing potential. Early identification of complications can also expedite treatment. This article reviews risk factors for peripheral vascular disease and illustrates their importance through a case presentation.

There are several questions the clinician must ask to assess risk in patients with vascular disease, such as 'how long has the patient had diabetes' and 'how well has it been controlled'? Vascular complications are directly linked to the duration of diabetes and glycaemic control. For every 30 mg/dl (1.7 mmol/L) elevation in fasting blood glucose, there is an equivalent increase of one percentage point in HbA_{1c}. Each 1% increase in HbA_{1c} is in turn associated with a 28% increased risk of peripheral vascular disease (PVD; Adler, 2002; Lee, 1999). Risk assessment should include a consideration of the following areas: macrovascular disease, microvascular disease, functional/microvascular disease, metabolic syndrome, and family history. Patients who have positive responses in several of these categories will tend to have more severe disease and therefore more complications.

1. Macrovascular disease

Is there a history of coronary artery disease, stroke, or intermittent claudication or rest pain (Adler, 2002; Boyko, 1997)?

2. Microvascular disease

Is there history of retinopathy, neuropathy, or nephropathy?

3. Functional/microvascular disease

Does the patient have gastroparesis or impotence? The autonomic nervous system controls blood flow, which is impaired due to lack of nitric oxide and resultant endothelial dysfunction. Endothelial damage with capillary leakage leads to the problem wound (Pham, 1998). Patients with autonomic neuropathy pulses may have severe functional microvascular disease (Tooke, 1986).

4. Metabolic syndrome

Is there a history of impaired glucose tolerance or insulin resistance? Does the patient have dyslipidaemia? Does the patient have obesity (central adiposity), hypertension, or a smoking habit? A 10 mmHg increase in systolic blood pressure is associated with a 25% increase in the risk of PVD (Adler, 2002; Kallio, 2003).

5. Family history

Is there a family history of diabetes? If so, did family members have end-stage complications of diabetes, including renal failure and amputations? Patients with positive family history of complications in our experience tend to have similar outcomes. This is likely related to the psychological and social aspects of patients managing their disease. Patients' education level, perception and environment can affect their outcome (Reiber et al, 1992; Armstrong et al, 1997).

Vascular exam

The patient's history will provide a high index of suspicion for vascular disease prior

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1 Ulceration should be evaluated in a systematic fashion. There are key findings within the wound that will indicate if the patient has PVD.

2 Clinicians must establish if there is a relationship between the location of the ulcer and its aetiology.

3 The size of the ulcer may help to determine how long it has been present.

4 Significant amounts of hyperkeratoic tissue around the rim of the ulcer and undermining the ulcer indicate improper offloading. This phenomenon is called the 'edge effect'.

to the examination. Symptoms of vascular disease are cold feet or muscle cramps with activity or rest. Patients with intermittent claudication complain of cramping or pain in the calf, thigh or foot with activity. However, intermittent claudication may be masked by neuropathy. This pain is reproducible usually after about 10–15 minutes of activity. Night cramps indicate less arterial flow without the assistance of gravity.

The lower extremity vascular exam should include palpation of the femoral, popliteal, dorsalis pedis and posterior tibial pulses in both extremities. Capillary fill time should be assessed with the foot at heart level. Diminished or absent pedal pulses, femoral bruits and venous fill time greater than 20 seconds has been associated with increased likelihood of PVD (Boyko, 1997). Patients without pedal pulses should be assessed by Doppler ultrasound.

Pallor on elevation and redness on dependant positioning should also be assessed. Pallor of the skin associated with microvascular dysfunction may be subtle. Sparse hair distribution is another subtle finding, patients will relate less hair growing on their legs to ageing. Skin temperature should be evaluated from the most proximal level of the leg to the feet.

Subcutaneous atrophy to the distal tuft of the toe is also indicative of severe vascular disease. The clinician may feel a sensation similar to a 'baked potato' on palpation. This is indicative of poor arterial flow, which places the patient at increased risk of infection and amputation. Focal areas of gangrene may represent a microembolic phenomenon (Benvegna, 1990; Sachs, 1982). Patients with venous stasis can also have arterial disease and may present with significant induration as well as swelling.

Ulceration should be evaluated in a systematic fashion. There are key findings within the wound that will indicate if the patient has PVD. These findings also correlate with the potential for a problem with healing.

Key questions for the clinician include:

1. Where is the ulceration located?
Clinicians must establish if there is a relationship between the location of the ulcer and its aetiology. For example, a severe digital contracture and tight foot wear can result in a digital ulceration at the dorsal inter-phalangeal joint.

2. How large is the ulcer?
This may help determine how long the ulcer has been present.

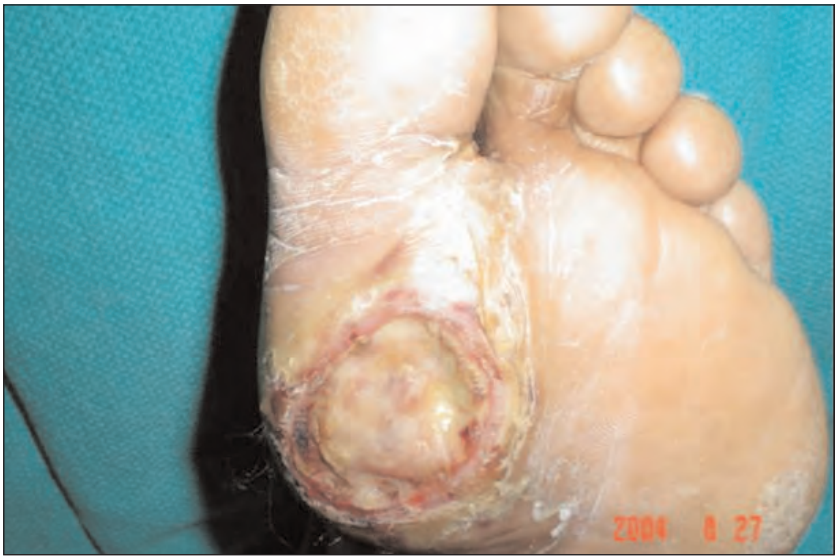
3. What do the wound margins look like?
Significant amounts of hyperkeratoic tissue around the rim of the ulcer and undermining the ulcer indicate improper offloading. This phenomenon is called the 'edge effect' (Armstrong, 1998).

4. What does the base look like?
Common descriptive terms are granular, fibrotic (yellow dense connective tissue) or necrotic tissue in the wound base. This tissue will need to be thoroughly debrided. It is also important to note if there is drainage from the wound, i.e. purulence or serous. The presence of foul odour may indicate Gram-negative or anaerobic infection.

5. How deep is the ulceration? Which underlying structures are involved?
The depth of the ulceration is an indication of how long the lesion has been present. Long-standing ulcerations will progress from colonisation to contamination and infection. Determining which structures are involved will help with infection eradication and incision planning if debridement is needed.

6. Is the ulcer infected? Are there systemic signs of infections?
Patients with diabetes may be unable to mount an adequate response to infection (Armstrong et al, 1996). Clinicians must rely on the five cardinal signs of infection: redness, heat, swelling, pain, and loss of function. In the previous section, the duration of the ulceration and the potential deleterious effects on underlying structures was discussed.

7. Is the ulceration ischaemic?
Ischaemic wounds will not have a granular base. These wounds typically have a pale-coloured base with fibrotic/necrotic tissue



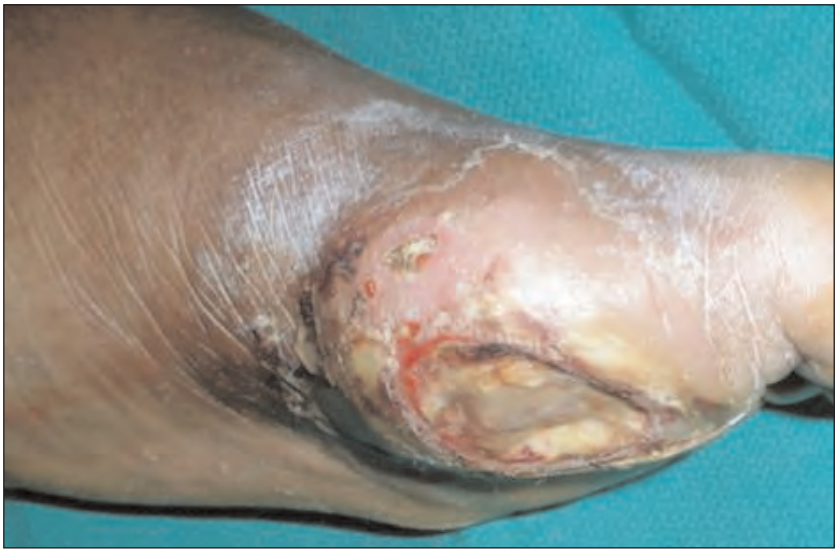


Figure 1 (top and above). Infected ischaemic ulceration at the plantar first metatarsal phalangeal joint with dense yellow-grey fibrotic tissue in the wound base.

that indicates a lack of blood flow.

Case example

Chief complaint

A 36-year-old African American male with diabetes presented to the emergency room with ulceration beneath the first metatarsal head on his left foot (see *Figure 1*).

History of present illness

The ulceration had been present for five weeks. The lesion began after walking with his family at an amusement park. He denied previous self or professional treatment for his feet. He related having a fever for three days.

Past medical history

He has had uncontrolled diabetes for 14 years. He related that he seldom checks his blood glucose and when he does it is often greater than 300 mg/dl (16.7 mmol/L).

Social history

He denied tobacco, alcohol or drug usage.

Family history

His mother and father both had diabetes. His father had renal failure and a below-the-knee amputation, and died after a massive myocardial infarction (MI).

Review of systems

The patient has neuropathy, retinopathy, impotence and gastroparesis.

Exam

The patient is an obese male with central adiposity. On vascular examination he had non-palpable popliteal, dorsalis pedis and posterior tibial pulses on the left. All other pulses were palpable. There was pitting oedema to the left foot with induration at the ankle. His left foot was pale. His capillary fill time was delayed at the left hallux; all other sites were normal. His protective sensation was evaluated with a Semmes Weinstein monofilament and was absent in both feet.

The ulceration beneath the first metatarsal phalangeal joint was 3x3 cm, full thickness with a dense grey-yellow denatured fibro/fatty tissue. There was serous drainage with no odour.

His temperature was 101.1 °F (38.4 °C). Pertinent laboratory values included: white blood cell count, 12.1 cmm; blood urea nitrogen, 13 mg/dl (4.6 mmol/L); creatinine, 1.3 mg/dl (115 μmol/L); blood glucose, 496 mg/dl (27.6 mmol/L); HbA_{1c}, 16.1 %.

This case presentation is quite a common one in our institution. We must quickly decide on treatment and be able to explain to the patient his likely potential outcome by correlating his risk factors.

Where is this patient along the risk spectrum of vascular disease?

1. Macrovascular

He has not had a coronary event or stroke. However, he has had uncontrolled diabetes for 14 years.

Figure 2. Open wound post-operative day three with non-healing yellow fibro/fatty tissue in wound base.

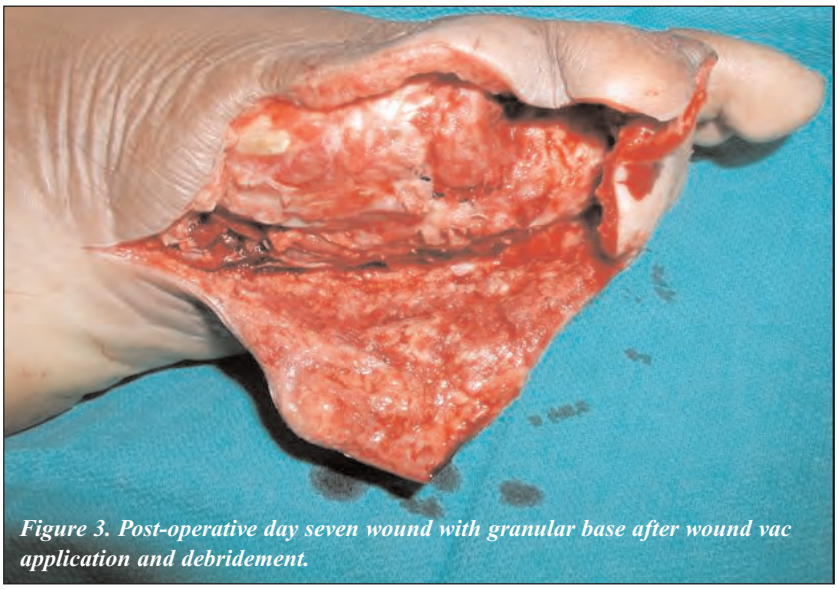


Figure 3. Post-operative day seven wound with granular base after wound vac application and debridement.

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1 Based on the clinical picture, the patient had an infected ischaemic wound with moderate to severe PVD.

2 The patient has functional microvascular disease evidenced by his gastroparesis and impotence and therefore will have endothelial dysfunction.

2. Microvascular

The patient relates neuropathy both subjectively and on exam. He has retinopathy.

3. Functional/microvascular

He had gastroparesis and impotence in a review of his systems.

4. Metabolic syndrome

The patient is obese with central adiposity.

5. History/diabetes control

He has had diabetes for 14 years that has been uncontrolled, as evidenced by HbA_{1c} of 16.1%.

6. Family history

There is positive family history of diabetes in both parents. His father had end-stage complication including amputation, renal failure, and MI.

Based on the clinical picture, the patient had an infected ischaemic wound with moderate-to-severe PVD. The aetiology of this wound, as per Cavanagh (1994), includes linking the risk factors: structure, function, footwear, and lifestyle (activity level and psychosocial).

Aetiopathogenesis in this patient was a combination of his increased activity, hallux limitus (limited joint mobility), neuropathy, and denial regarding his disease. Despite only being 36 years old, he was at risk of limb loss. The infection is likely to be due to the chronicity of the wound with colonisation, and contamination progressing to infection. The ulceration is ischaemic, as evidenced by the lack of pedal pulses, pallor and the dense grey-yellow denatured fibro/fatty tissue in the wound base.

His history is the primary predictor for ischaemia, with a 28% increase in PVD seen with elevated HbA_{1c} (Adler, 2002). He has functional microvascular disease evidenced by his gastroparesis and impotence and, therefore, will have endothelial dysfunction.

In our experience, this is one the most consistent findings in patients who have a problem wound. Patients with problem wounds will have chronic serous drainage and dense fibrotic tissue. Ischaemic ulcerations on the plantar aspect of the foot typically suggest that there is a lesion within the posterior circulation. This patient had a vascular evaluation, which included non-invasive studies and an arteriogram. As suspected, the posterior tibial artery was occluded.

The patient underwent a femoral to dorsalis pedis bypass graft. During his hospital stay he remained on intravenous antibiotics. He also had a partial first ray amputation due to significant infection along the flexor hallucis longus tendon and lack of soft tissue coverage (*Figure 2*).

The patient continued to undergo wound care, which included serial debridement and application of a wound vac (*Figure 3*). The wound was closed primarily (*Figure 4*);

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1 The wound was closed primarily; however, it dehisced on post-op day three.

2 The patient's family history for diabetic complications indicates that he is genetically predisposed to the same severe disease and fate.

3 Risk factors associated with diabetes are dynamic and not static – therefore, constant evaluation and treatment is important.

Figure 4. Wound closed with dorsal flap to cover the plantar defect.

however, it dehisced on post-op day three after the closure (*Figure 5*). He is currently healing his lesion secondarily. His family history for diabetic complications indicates that he is genetically predisposed to the same severe disease and fate. Risk factors associated with diabetes are dynamic and not static – therefore, constant evaluation and treatment is important. ■

Figure 5. Dehisced wound after bypass with functional microvascular disease.

NOTE: Debridement of ulcers in such high-risk patients as described should only be undertaken by a podiatrist or surgeon who has the requisite experience and is a member of, or has direct access to, the multidisciplinary foot care team.

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