



Diabetic Complications of the Feet

Discussion paper prepared for

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This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

DIABETIC COMPLICATIONS OF THE FEET

Approximately 7% of North Americans are affected with diabetes. Based on current trends, this percentage will increase further with time. Add the increased longevity of these patients and it is clear that the total population of diabetic patients will continue to grow, as will complications associated with diabetes. During their lifetime, approximately 15% of patients with diabetes will develop a foot ulcer requiring advanced care. It has been estimated that up to 20% of the total health care expenditures on diabetes is related to the management of complications associated with diabetic feet.

Definition of diabetic complications of the feet

A complication refers to a secondary disease or condition developing in the course of a primary disease or condition. Complications of diabetes that affect the lower extremities include:

- Peripheral neuropathy (Impairment of the function of the nerves that provide sensation and control of the muscles in the feet)
- Peripheral arterial disease (Reduced blood supply to feet and legs)
- Restricted joint motion (Most commonly involves the great toe)

Peripheral neuropathy

There are multiple types of peripheral neuropathy associated with diabetes, the most common of which is a distal sensorymotor polyneuropathy. Both limbs are equally affected with symptoms being most marked in the toes and feet. Diabetic neuropathy may be further subdivided into sensory, motor or autonomic neuropathy depending on the specific nerve fibers affected.

Sensory neuropathy: When the small diameter fibers of the nerve are affected, patients may experience severe pain and dysesthesia (abnormal response to stimuli which are not usually painful). Such symptoms may occur in isolation or with concurrent involvement of other parts of the nerve. These patients may complain of severe burning or lancinating pain in the feet with few other symptoms being present.

More often, the large sensory fibers of the nerve are affected resulting in loss of protective sensation to the foot. The onset of this type of neuropathy can be quite insidious such that the patient may not be aware of any abnormality in spite of significant findings on examination. This loss of protective sensation can result in repeated episodes of trauma to the foot going unnoticed.

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Motor neuropathy: When the motor fibers of a nerve are affected, the muscles supplied by the nerve will weaken and atrophy over time. In the case of diabetic polyneuropathy, the small muscles of the feet are primarily affected. In particular, the muscles that straighten the toes are weakened, resulting in a claw toe deformity in which the toes are curled and the balls of the feet become more prominent. See Figure 1 below.

Autonomic neuropathy: Involvement of the autonomic nervous system can result in reduced sweating of the feet. The result is dry thin skin which is prone to cracking. In addition to the skin being less pressure tolerant, cracks in the skin can serve as portals for bacteria to enter the foot and cause more significant infections.

Peripheral arterial disease (PAD)

Diabetes is a known risk factor for the development of PAD. The major vessels in the legs (large vessel disease) and/or the distal arterioles and capillaries (small vessel disease) may be affected in patients with diabetes. Large vessel disease results in stenosis or narrowing of the arteries and is often amenable to bypass surgery and/or angioplasty. Small vessel disease on the other hand usually involves occlusion or blockage of the arterioles and capillaries and is more difficult to treat, as it is not amenable to surgical interventions.

Restricted range of joint movement

Limited joint mobility most commonly affects the small joint of the hands in patients with diabetes, but can also affect movement of the toes. This is most significant for the big toe. Hallux rigidus results in the toe having reduced flexibility when pushing off, resulting in added pressure being applied to the first metatarsophalangeal joint (ball of the foot by the big toe) when walking. Thus a stiff big toe makes the foot more prone to skin breakdown and ulceration.

Clinical presentation of diabetic foot complications

Complications associated with diabetic feet may occur in isolation but more frequently in combination, resulting in variable presentation of symptoms from patient to patient.

Foot and leg pain

Pain in the feet may be related to the presence of neuropathy and or vascular impairment. Neuropathic pain is frequently described as burning or vice-like and reflects involvement of the small diameter nerve fibers affected by peripheral neuropathy.

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Vascular or ischemic pain may be of two types. Intermittent claudication refers to cramping type pain that comes on in a specific muscle group with activity. This pain occurs when blood flow is not sufficient to meet the metabolic demand of the active muscle. The location of the pain varies with the specific artery affected, but in diabetics the calves are most frequently involved. Often patients will be able to specify a specific distance they can walk before the pain comes on. It is characteristically relieved with rest and more severe when walking on inclines or hills. If an individual is quite sedentary, they may never walk sufficiently to experience intermittent claudication. Such patients can have advanced PAD even though they have few symptoms.

Rest pain refers to pain due to impaired blood flow into the foot to the point that the basic metabolic demands of tissues in the foot are not being met. The pain is brought on or made worst by elevating the leg and thus is often at its worst at night. Rest pain reflects the presence of advanced PAD.

Foot deformity

A motor neuropathy involving the foot will result in the small muscles of the foot becoming progressively weaker. When these muscles weaken, the toes tend to pull into a clawed position such that the tops of the toes are raised and the metatarsal heads (or balls of the feet) become more prominent. These deformities of the foot are associated with abnormally high pressures being applied to the tops of the toes (when wearing shoes) and to the balls of the feet. Repeated low level trauma to these high-pressure areas (such as may occur after a prolonged period of walking or standing, especially if in non-cushioned shoes) may go unnoticed and result in cumulative damage that subsequently results in a diabetic foot ulcer.

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Figure 1: Claw toe deformity

Note the prominence of the balls of the feet and pressure on the knuckles of the toes from the shoe

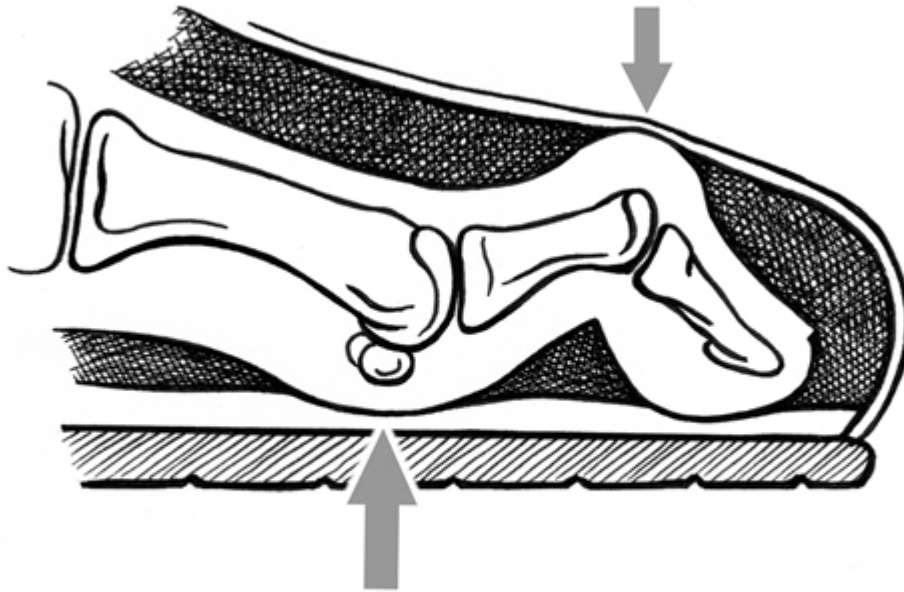


Figure 2: Claw toe deformity:

The initial lesion at the tip of the toe occurred from rubbing on a shoe. The lesion (circled) occurred from rubbing on bed sheets



(Photo: M. Marks)

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In more extreme cases, diabetic patients with advanced neuropathy may develop Charcot joint deformities, also referred to as Charcot arthropathy or Charcot foot deformity. In these situations there is destruction of the normal joint architecture resulting in collapse and mal-alignment of the foot. Although any of the joints of the foot can be affected, one of the most common deformities associated with a Charcot foot is collapse of the mid-foot. The result is a prominence of the sole of the foot, known as a "rocker bottom sole."

Figure 3:
Charcot foot deformity with collapse of the mid-foot on x-ray



(Photo: M. Marks)

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Figure 4:

Charcot foot deformity with a "Rocker bottom sole" - Note the raised area (circled).



(Photo: M. Marks)

Charcot arthropathy can develop seemingly spontaneously with no history of known trauma. It is also quite common that these extreme changes in the architecture of the foot will occur following a relatively minor trauma. During the acute phase the foot is usually warm and swollen. Patients may or may not experience much pain and may still walk on the foot in spite of unrecognized fractures or underlying tissue destruction. When the foot eventually heals, it usually has reduced mobility and marked bony mal-alignment. Both of these features result in elevated areas of pressure in the foot when walking, predisposing the overlying tissues to ulceration. Approximately 0.2% of people with diabetes will develop an obvious Charcot foot deformity on visual inspection, but up to 3% of patients with diabetes will have joint changes on x-ray.

Foot ulcers

The complication that receives the most attention related to a diabetic foot is the foot ulcer. Such ulcers are the precursor for 85% of lower extremity amputations required by patients with diabetes. Approximately 10 - 15% of diabetic foot ulcers are primarily related to PAD. Often a blood vessel will be acutely blocked, stopping blood flow to a focal area of the foot. The result may be localized gangrene in the foot such as that seen in Figure 5.

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Figure 5:
Dry gangrene (black area) due to PAD



(Photo: M. Marks)

While foot ulcers can be related to impaired blood flow, more often neuropathy, trauma and infection are equally important factors in the development and healing of diabetic foot ulcers. In fact, PAD is thought to be more of a factor in delayed wound healing once a diabetic foot ulcer has developed than the primary reason for most ulcers developing.

It is important to note that minor trauma is a frequent precursor to serious diabetic foot ulcers. Injuries that an otherwise healthy person would readily heal frequently progress in patients with diabetes. This is in part related to the fact that such injuries are not noticed when protective sensation is lacking; the initial lesion is often neglected by those affected as it is not painful. Combined with impaired blood flow and limited ability to mount a normal inflammatory/healing response, these seemingly minor injuries result in much more serious conditions than one would normally expect. Thus a torn toenail, a blister on the foot, or a superficial burn are much more significant in a diabetic with impaired sensation and blood flow than in the normal population. Some estimate that 60-85% of foot ulcers are started with a minor trauma, most commonly associated with ill-fitting shoes. Other sources of trauma include accidental cuts when cutting toe nails, burns from hot water used to soak feet, and acute mechanical trauma (stubbing a toe; dropping an object on the foot).

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Figure 6:

Diabetic foot ulcer resulting from prolonged walking in a hard-soled shoe



(Photo: M. Marks)

Figure 7:

Diabetic foot ulcer with gangrene (black area) resulting from rubbing on a shoe



(Photo: M. Marks)

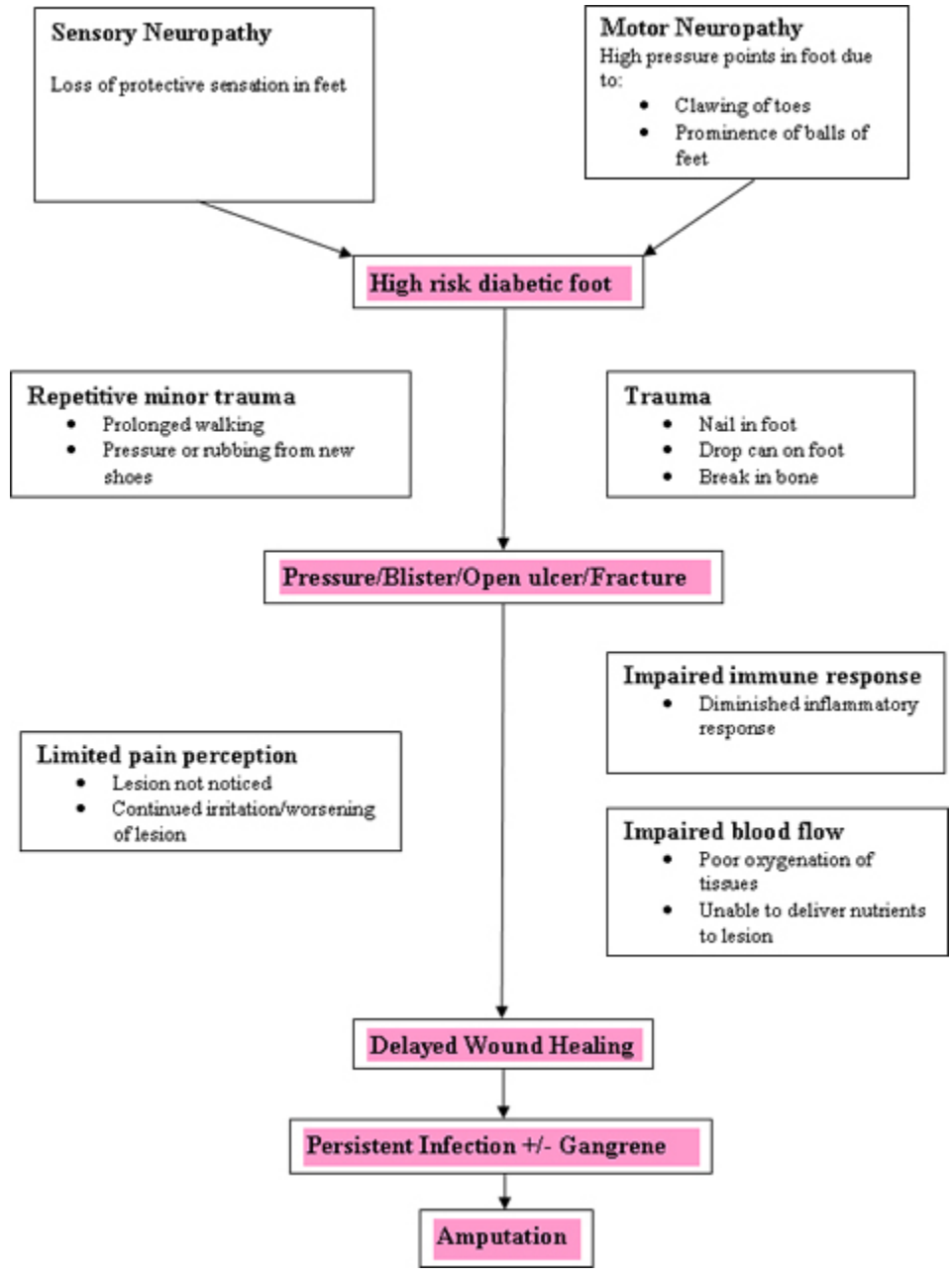
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Infection is often associated with diabetic foot ulcers but is rarely the cause of such ulcers. The usual portal of entry for an infectious process is a disruption in the integrity of the skin - such as with a puncture wound, a blister or an ulcer. Even when an infectious process is well established, the usual signs and symptoms may be diminished due to the presence of PAD and neuropathy. Thus, infections may be at an advanced stage before they become recognized. This makes the infection more difficult to treat and poses a higher risk of it extending to involve the underlying bone, increasing the risk of amputation.

The onset and progression of most diabetic foot ulcers can be thought of a cascade of events involving each of the factors outlined above (Refer to Figure 8). No matter what the etiology, an open ulcer on the foot predisposes the diabetic foot to infection, gangrene, and increased risk of amputation.

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Figure 8:
Cascade of Events Resulting in Diabetic Foot Ulcers



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The usual course of vascular/neurological foot problems in persons with diabetes.

The pathologic changes associated with diabetic neuropathy and PAD tend to progress over time. The actual rate of progression varies with glycemic control, duration of diabetes, genetic predisposition, gender, and lifestyle factors (smoking, diet and exercise). Type I diabetics tend to have a faster rate of progression than Type II diabetics.

Pre-diabetes also increases ones risk of complications associated with diabetes, especially heart disease and stroke. Pre-diabetes is diagnosed when one has an elevation of fasting blood sugar levels or an impaired glucose tolerance test, but yet remains below the level required to diagnose diabetes. While these individuals have an increased likelihood of developing diabetes they can delay or prevent onset of diabetes with weight loss and exercise.

Interestingly, at any point in time, one-third to one-half of individuals with diabetes is undiagnosed. Symptoms experienced may be relatively mild and go unrecognized by those affected - for months to years. However, these same patients may gradually develop significant complications associated with diabetes. Occasionally diabetes is first diagnosed when an individual presents with advanced findings of neuropathy and vascular disease severe enough to require an amputation. In these cases it is assumed that the diabetes was present but undiagnosed for an extended period. In such cases, a minor foot trauma may again be the initiating factor in causing a non-healing foot ulcer, ultimately leading to the diagnosis of diabetes.

As well, occasionally an individual will have been diagnosed with diabetes but refused treatment or was lost to follow-up with a physician. Again, in spite of limited overt symptoms, these individuals are especially prone to develop medical complications associated with diabetes.

The earliest clinical finding associated with neuropathy is usually loss of sensation in the toes, with gradual progression up the leg. Patients are not always aware of having loss sensation in the feet due to the gradual onset of the condition. Typically sensory abnormalities will predate the onset of motor findings.

Peripheral vascular disease also tends to be progressive. However, up to 50% of patients with PAD (Ankle-Brachial Index of 0.8 or less - as defined on page 11) will be asymptomatic. Of those with symptoms, 70% will have classic claudication (cramping leg pain brought on with exercise) and 2-4% will have critical ischemia in the lower extremities. After 5 years, 80% of patients with non-critical limb pain due to vascular disease will remain stable, 15-20% will have worsening of their claudication and 1-2 % will progress to develop critical limb ischemia.

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How does one determine if foot conditions are related to diabetes?

Peripheral neuropathy is present in up to 50% of patients with Type II diabetes and is present in over 80% of diabetic patients who develop a foot ulcer. However, diabetic neuropathy is a diagnosis of exclusion. Up to 10% of patients with diabetes with new onset peripheral neuropathy will have other causes for their neuropathy. A detailed history and physical examination, along with screening blood work is usually sufficient to exclude other causes of neuropathy in these situations. For example, Lyme disease, heavy metal poisoning, and cancer are also potential causes of a polyneuropathy that might be related to one's work environment.

Similarly, while peripheral vascular disease is commonly associated with diabetes, it also occurs in patients with other predisposing factors. One should not assume that all diabetics have polyneuropathy or PAD. A traumatic injury to a foot with subsequent delayed healing may be related to the extent of the injury or secondary infection, rather than complications associated with diabetes. In such cases, a documented normal sensory and vascular examination would be sufficient to rule out a pre-existing polyneuropathy and PAD. Alternatively, in situations where sensory and vascular findings are already evident at the time of injury, the trauma may aggravate a preexisting condition or initiate a cascade of events that lead to more serious problems.

Diagnostic tests: Which are appropriate and how are they interpreted?

Peripheral neuropathy

Nerve conduction studies are the gold standard to assess for peripheral neuropathy. However, as these tests are costly and have only limited availability they are rarely done in clinical practice to make the diagnosis of a peripheral neuropathy associated with diabetes. Rather, basic screening tests, completed as part of a physical examination, are more commonly used.

Vibration sense: A person's ability to sense vibration can be assessed with a 128-Hz tuning fork. The patient is asked to identify when they feel a vibration from the tuning fork and when it disappears on dampening. If the examiner is able to feel the vibration in their hand when the patient can no longer feel vibration in their toes, the test is abnormal.

Pressure sensation: Thin monofilaments can be used to assess pressure sensation in the foot. Inability to detect the pressure applied by a 5.07 monofilament indicates loss of protective sensation in the foot and the presence of a sensory neuropathy. Loss of fine touch in the foot, detected by stroking the foot with a tissue, indicates even more advanced neuropathic change.

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Documentation of these findings even at the time of an injury would indicate that the neuropathic disease process had been present for a significant period, thus predating the actual injury.

PAD

Contrast angiography is the gold standard for the diagnosis of peripheral arterial disease. Dye is injected into the vascular system to assess for blockages or partial occlusions of the vessels in the legs. Magnetic resonance angiography may also be used to assess blood flow in the legs. These studies would typically only be undertaken if surgical intervention is being considered to restore blood flow in the legs.

Clinical exam findings may include absence of pulses in the lower limbs, reduced hair growth, and discoloration of the feet. In advanced disease, patients may have increased redness of the feet when they hang down (dependent rubor). While there are screening tests to assess blood flow in the legs, these are not typically done in a physician's office. More commonly they will be completed in a vascular studies lab and may include the following:

Ankle Brachial Index is calculated by measuring systolic blood pressure at the ankle and the arm. Normally the ratio for Ankle:Arm is 1.1 - 1.2. Patients with claudication will typically have a ratio of 0.4 - 0.9 while a ratio of 0.4 or less is typically associated with rest pain and/or death of tissues (gangrene). Caution is needed in interpreting the results of these studies for diabetics as the ratio can be artificially elevated if the patient also has hardening of the arteries (and they often do).

Segmental doppler pressures and plethysmography are also used to assess peripheral blood flow. A 20 mmHg drop in pressure when comparing legs or comparing two adjacent segments in the same leg is considered a significant indicator of impaired blood flow. A blunted pressure pulse contour obtained with plethysmography can also indicate the presence of significant restriction of blood flow.

Foot deformity

Plain x-rays of the feet will detect bony destruction associated with Charcot arthropathy and chronic infection of the bone (osteomyelitis). As these two conditions can look very similar on x-ray, MRI may be helpful in the acute stages to distinguish between an infection and Charcot arthropathy. There are occasions when both conditions occur in unison. For example a patient may develop an ulcer due to the bony changes associated with Charcot arthropathy. If the ulcer progresses to expose the underlying bone and causes it to become infected it may

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be impossible to determine which changes in the foot were due to the initial Charcot deformity and which are due to a superimposed osteomyelitis. In this situation, pre-injury investigations may provide a baseline indication of bony involvement. A bone or deep tissue biopsy that is negative for infection would also exclude the diagnosis of osteomyelitis and support the diagnosis of Charcot arthropathy.

Differential diagnosis

All of the complications associated with diabetic feet can be caused by other conditions that should be excluded before concluding that diabetes is the primary cause. Although diabetes is the leading cause of peripheral polyneuropathy in the developed world, there are multiple causes of polyneuropathy that may occur concomitantly with diabetes. Other considerations in the differential diagnosis for diabetic polyneuropathy, depending on the history and physical findings of the patient, include the following:

- Metabolic: Uremia, Hypothyroidism, B12 deficiency
- Toxic: Alcohol, heavy metals - lead and mercury, hydrocarbons, drugs
- Infectious/Inflammatory: Sarcoidosis, Leprosy, Lyme disease, SLE
- Other: Leukemia, amyloidosis, paraneoplastic syndrome, and hereditary neuropathies

PAD is associated with diabetes but is not caused by diabetes per se. Rather patients with diabetes appear to be predisposed to develop PAD due to arteriosclerosis. Four primary risk factors for the development of peripheral arterial atherosclerosis include: Diabetes (often poorly controlled), hyperlipidemia (high cholesterol levels), cigarette smoking, and hypertension (high blood pressure). These are the same risk factors for the development of arteriosclerosis associated with coronary artery disease and cerebral vascular disease. The incidence of PAD is also known to increase with age. Of those over 70 years of age 14.5 % will have PAD, while only 0.9% of the general population between the ages of 40 and 49 years will have PAD.

Other potential causes of PAD include:

- Limb trauma resulting in an acute arterial injury
- Hypercoagulable state
- Vasculitis - including thromboangitis obliterans (Buerger's disease)
- Arterial fibrodysplasia
- Arterial dissection
- Occluded limb aneurysms
- Radiation fibrosis

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How frequently does diabetes lead to toe/foot amputation in the usual course?

The reported incidence of foot and toe amputations is variable worldwide. Reporting structures, health care infrastructure, the experience of surgical teams and definitions of what constitutes a minor versus a major amputation all influence the results. In North America more than 60% of lower extremity amputations are associated with diabetes. Of this patient group, 85% will have had a complicated foot ulcer that resulted in an amputation. Approximately 15% of diabetics will develop a foot ulcer during the course of their disease; 7-20% of these ulcers will lead to a lower extremity amputation.

Amputation rates for all diabetics range from 5 - 8 per 1,000. In other words slightly less than 1% of all diabetics will require a lower extremity amputation. The rates for men are twice as high as for women with diabetes. On average the following rates of amputation have been noted for patients with diabetes:

- Toe - 2.6 per 1000
- Below knee amputation - 1.6 per 1000
- Foot - 0.8 per 1000
- Above knee amputation - 0.8 per 1000

Based on the combined estimates presented above, the following can be used as a guide in assessing the risk of a diabetic patient requiring an amputation. If one were to follow 1000 patients with diabetes you can expect that approximately:

- 150 will develop at least one foot ulcer
- 16 will have at least one amputation at the levels indicated below:
 - 8 toe amputations
 - 4 below knee amputations
 - 2 foot amputations
 - 2 above knee amputations

For any patient with diabetes, the best predictors of future limb amputation include:

- History of a previous foot ulcer
- Presence of neuropathy resulting in loss of protective sensation
- Presence of impaired blood flow into the foot
- Poor glycemic control (persistently high blood sugars or poorly controlled diabetes).

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Controversy in the medical community about diabetic foot disease

Patients with diabetes have onset of neurological and vascular complications at variable rates. The initial stages of development of neuropathy and PAD can be overlooked by both the patient and the physician as symptoms are frequently absent. Screening for these complications is the only way to know when they have their initial onset. By the time frank symptoms develop the process will have usually been progressing over a period of years.

Charcot arthropathy is perhaps the least understood complication of diabetes associated with the foot. There is not full agreement as to why the foot is predisposed to Charcot arthropathy. Most patients have advanced sensory polyneuropathy when they develop a Charcot foot deformity and are frequently unaware of the extent of injury they may have had to the foot. Yet some patients do report pain associated with Charcot arthropathy. Osteopenia, or a thinness of the bone, associated with this condition may be related to altered blood flow in the limb due to autonomic neuropathy. However evidence of an autonomic process is not always present at the time of diagnosis of a Charcot arthropathy.

An acute Charcot arthropathy can be easily mistaken for a cellulitis or inflammation of the skin, especially in the early stages of onset. The distinction of a Charcot arthropathy from osteomyelitis, or a bony infection in a diabetic foot also be challenging. Both conditions can eventually result in gross destruction of the affected bones. Bone scans can present with similar findings and even an MRI may not be always able to distinguish between joint destruction due to a chronic infection versus a Charcot arthropathy. However, a grossly destroyed joint with no sign of surrounding soft tissue infection on MRI is unlikely to be due to osteomyelitis. White blood cell labeled scans can help in identifying infections associated with osteomyelitis, but even then must be interpreted in light of the clinical presentation.

Relationship between injuries to the foot, or ill-fitting foot wear (blisters) and the diabetic foot. Can these types of trauma lead to aggravation/acceleration of the usual course of vascular/neurological foot problems?

Minor injuries to the foot, including the development of blisters from ill-fitting footwear can lead to aggravation of a diabetic foot. The injury does not change the underlying neural or vascular pathology. However, minor injuries to the foot increase the risk of an open foot ulcer. Any open ulcer on a diabetic foot leaves the foot at risk for infection, gangrene and the need for subsequent amputation (See Figure 8).

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Case: Recurrent blisters. Worker sustains a blister from his work boots; the blister heals but then recurs and becomes infected, eventually leading to amputation.

The occurrence of a blister does not cause subsequent blisters nor predispose the foot to new blisters. Rather the occurrence of a blister in a diabetic foot is a sign of propensity to develop subsequent blisters and ulcers. In these situations, patients will typically have evidence of sensory loss in the foot due to polyneuropathy. The primary issue is the neuropathy resulting in loss of sensation and inability to detect normally painful tissue irritation in the feet. The rate of recurrence of blisters/ulcers after an initial break in the skin in a diabetic foot ranges from 28% - 34% at 1 year, increases to approximately 50% after 3 years and further increases to 85% at 5 years. Thus subsequent ulcers are expected in the usual course of a diabetic foot.

It must be stated however, that trauma is usually involved in initiating the process of ulcer development in the predisposed diabetic foot (affected with sensory loss and/or vascular impairment). Depending on the extent of the trauma sustained, it may be viewed as making the underlying condition apparent (a blister develops after prolonged walking on a hard surface) versus aggravating an underlying condition (a heavy weight is dropped directly onto the foot).

Case: Infection in a pre-existing blister due to polluted material leaking into boots. Infection is treated but the problem recurs, becomes gangrenous and eventually requires amputation.

The extent of the infection, size of the resulting ulceration and duration of treatment will all influence subsequent outcomes for this patient. Assuming a spread of infection beyond the immediate vicinity of the blister it is likely that the underlying tissues were scarred and left in a more fragile state than they had been prior to the infection. Marked inflammation and swelling of the tissues may also aggravate small vessel disease by causing a reactive vasculitis and further impairing blood flow to the affected area. Such changes could result in increased risk of subsequent ulceration and skin breakdown. This would especially be the situation if the polluted material were caustic or highly infectious for example. As well, if the underlying bone were infected it is more difficult to eradicate all infectious pathogens - the patient may go on to develop chronic osteomyelitis, again increasing risk for open ulcers on the foot and recurrent infections. Thus the polluted material leaking into the boots may aggravate the usual course of the diabetic foot problem.

On the other hand, if the resulting infection were limited in nature and tissues healed completely, then the polluted material may not have caused any additional pathology over and above that associated with the natural disease process. In this situation, any recurrence of an ulcer is more likely to be associated with the natural progression of the diabetic foot than the initial infection.

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Case: An initial foot blister heals in a diabetic worker; the worker develops a second blister and then requires an amputation. Is the amputation related to the first blister, the second or the usual course of the diabetic foot?

If a diabetic worker has a foot ulcer or blister that completely heals it will not be the cause of a subsequent amputation. However, with a deep ulcer the skin may close on the surface while the underlying ulcer remains. In such a situation the ulcer may have appeared to heal but will subsequently re-open in the same area with more extensive tissue loss under the skin than is immediately apparent on visual inspection. Such ulcers are often described as having extensive undermining.

Assuming a simple blister or superficial ulcer that fully healed, the occurrence of this initial lesion signals that the worker is at high risk for subsequent lesions due to complications associated with diabetes. The second lesion is likely to have its onset following a trauma to the foot, which may or may not be work related. If the second blister results in an infection or gangrene in the foot it can lead to amputation (in a predisposed foot affected with neuropathy and PAD). In deciding if this process is more related to the natural course of diabetes versus an acute injury one might ask if a non-diabetic worker would be expected to have a similar outcome with the same injury. Frequently the answer will be no. For example most workers who happen to develop a blister due to wearing new shoes would not expect the lesion to progress to the point of amputation. Rather, it is the underlying disease process that results in this progression.

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