MANAGEMENT OF DIABETIC FOOT COMPLICATIONS OTHER THAN ULCERATION

1. Diabetic Distal Symmetric Polyneuropathy (DPN)

Etiology
DPN is a chronic sensory and motor neuropathy involving the distal extremities, usually commonly the lower limbs. It is the most common type of nerve damage seen in people with diabetes. DPN is associated with the following risk factors: duration of diabetes, poor glycemic control, high blood pressure, and hyperlipidemia.

Patients with DPN may experience symptoms such as neuropathic pain (burning, stabbing or aching pain, paresthesias, hyperasthesias) or numb feet. Neuropathic pain tends to be worse at night. Up to 50% of people with DPN may be asymptomatic; some of these may present with a painless foot ulcer.11

Diagnosis
DPN can be diagnosed only after a complete clinical examination of the limbs, including sensory and motor nerve testing. Other causes of peripheral neuropathy (such as vitamin B12 deficiency, hypothyroidism, uremia and chronic inflammatory demyelinating polyneuropathy) should be excluded before making a diagnosis of DPN.

Management
Includes:

- Foot care and education - all patients with DPN are at increased risk of foot ulceration.
- Stable and optimal blood glucose control – observational studies suggest that neuropathic symptoms improve with less fluctuation in blood glucose levels.11
- Pharmacologic therapy for neuropathic pain – A number of medications have proved in randomized trials to be useful in the treatment of painful diabetic neuropathy. These medications include: Tricyclic antidepressants (amitriptyline, nortriptyline, imipramine); Anticonvulsants (gabapentin, pregabalin, sodium valproate, lamotrigine); SSRI antidepressants; Opioids (oxycodone, morphine). All these drugs have side effects that may limit their therapeutic usefulness. Generally, it is advisable to start with a low dose and gradually titrate the dose to symptom relief in order to lessen the severity of side effects. Combinations of some drugs may be used in cases of severe neuropathic pain.12
- Non-pharmacologic therapies – for patients with severe neuropathic pain, multidisciplinary interventions (such as podiatric, physical, behavioral and psychosocial therapies) may assist with long-term management.
2. **Peripheral Arterial Disease (PAD) in Diabetes**

**Etiology**

Diabetes mellitus is associated with proatherogenic changes due to increases in vascular inflammation, derangements in the cellular components of the blood vessels (vascular endothelial cells and vascular smooth muscle cells), and alterations in platelet function and hemostatic factors. These vascular abnormalities lead to the formation of atherosclerotic plaques, resulting in narrowing or occlusion of large and small arteries. Poor glycemic control, smoking, hypertension and hyperlipidemia are associated with more accelerated atherosclerosis. In people with diabetes, PAD tends to involve the blood vessels below the knee, resulting in poor oxygen delivery to the tissues of the foot (ischemia). This will contribute to skin ulceration, poor wound healing and development of gangrene.

Damage to the sympathetic nerves that control the blood flow to the foot is termed autonomic neuropathy. Autonomic neuropathy results in high intraluminal flow rates, a tendency to vasodilation and resultant arteriovenous shunting. Arteriovenous shunt formation in the lower limb of diabetics is associated with reduced nutritive skin blood flow and with inflammatory changes in the skin.

Diabetes and smoking are the strongest risk factors for PAD. Other factors are hypertension, hyperlipidemia and advanced age.

**Diagnosis**

*a) Clinical Evaluation*

**History** - depending on disease severity, individuals with PAD present with a spectrum of symptoms ranging from no pain, intermittent claudication (leg muscle pain or fatigue on walking), rest pain in the leg or foot, to nonhealing foot ulcers and gangrene.

**Physical examination** – decreased or absent arterial pulses in the distal leg and foot, and signs of chronic vascular insufficiency (cool, dry skin; absence of hair growth; dystrophic nails; dependent rubor; pallor on elevation of the foot).

*b) Diagnostic Testing*

“Noninvasive” arterial studies are performed to evaluate perfusion of the lower limbs:

- **Ankle-Brachial Index (ABI)** – is a reproducible and reasonably accurate noninvasive measurement for the detection of PAD and disease severity. It is the ratio of the systolic blood pressure in the ankles (dorsalis pedis and posterior tibial arteries) and the systolic blood pressure in the arms (brachial artery). The measurements are taken with a blood pressure cuff and a hand-held Doppler. ABI is considered normal if it is in the range of 0.91 – 1.30. A lower blood pressure in the ankle, resulting in an ABI of <0.9 is indicative of PAD. Severe arterial obstruction usually has an ABI <0.4.

However, the ABI is less reliable in confirming the diagnosis of PAD in some individuals with diabetes and in the elderly, who tend to have calcified, poorly
compressible blood vessels, resulting in ABI values being inaccurate and often elevated at >1.30.15

- **Toe blood pressure** – systolic blood pressure in the toes may be a more accurate measure of the severity of PAD in individuals with calcified arteries in the lower leg, because vessel calcification does not extend to the digital arteries. There is growing evidence that toe blood pressures may have a role in predicting foot ulceration risk and predicting successful wound healing.15 Toe blood pressure >40mmHg is favorable.

In an individual with confirmed PAD, further tests to assess the location and severity of arterial occlusion may be performed. These involve vascular laboratory measurements and angiography. In Saskatchewan, referral to a Vascular Surgeon is required for these tests; angiography is performed only when revascularization procedures are being considered.

**Management of PAD**
The presence of PAD in a person with diabetes indicates high risk for other cardiovascular disease (CVD), including coronary artery disease and stroke. Therefore, medical management of PAD includes all the measures routinely recommended for CVD risk reduction, as follows: smoking cessation; optimal glycemic control; treatment of hypertension; use of an anti-platelet agent (e.g. Aspirin); use of lipid-lowering drugs (e.g. Statins); exercise. Smoking cessation and a supervised exercise (walking) program are the two most important interventions in the treatment of symptomatic PAD. Treatment with lipid-lowering drugs has been shown to reduce the severity of symptoms of intermittent claudication.15,16

All individuals with diabetes and PAD should receive regular preventive foot care to minimize the risk of developing foot ulceration and amputation.

Individuals with severe PAD or symptoms suggestive of critical limb ischemia (rest pain in leg or foot, progressive or non-healing ulceration, or gangrene) require referral to a Vascular Surgeon. The urgency of this referral depends on the individual’s presenting clinical symptoms, medical history and comorbidities.

3. **Diabetes Related Dermatology**

With diabetes comes the increased occurrence of skin changes and pathology. Autonomic neuropathy can effect innervation of the sweat glands, leading to dry skin. This condition may seem minor, but, in conjunction with the triad of diabetic peripheral arterial disease, neuropathy and injury, this may lead to skin ulceration and can be limb threatening. The health care practitioner should be aware that presentations such as small skin cracks, thick nails and callus formation can be precursors to diabetic foot wounds. Such conditions need to be monitored and treated by the appropriate practitioner to prevent them from progressing to ulceration. Self-treatment of these skin conditions by the individual with diabetes is a common cause of new diabetic foot ulcers.
Management
Early detection and treatment, along with patient education on self-management is the key to prevent progression of minor dermatologic pathology to ulceration. Self-management of hyperkeratosis and anhydrosis would involve regular use of pumice stone or foot file (non sharp instruments) and skin emollients or lotions.

Podiatric or medical management would involve precise scalpel debridement of hyperkeratoses, including reduction of callus and enucleation of helomata. Methods to reduce pressure on these sites can be devised (e.g. accommodative footwear, deflective padding, insoles, and/or orthoses). There may also be a need for specialty hypertrophic nail debridement. Conservative or surgical removal of involuted or ingrowing toenail edges may also be required.

4. Deformities in the Diabetic Foot

Etiology
Distal muscle atrophy is commonly associated with loss of motor nerve function. The outcome of weakening intrinsic foot muscles is overall muscle imbalance, which produces changes in foot structure and gait. The resulting deformity and limited range of motion contribute to increased mechanical stress (compression and shear forces) on corresponding areas of the foot.

Diagnosis
Toe deformities can be easily recognized by the professional. A flexible deformity that involves extension contracture at the metatarsophalangeal joint with flexion contracture at the proximal interphalangeal joint is commonly referred to as a “claw toe”. While a fixed deformity that involves extension contracture of the metatarsophalangeal joint and flexion contracture of the interphalangeal joint is commonly referred to as a “hammer toe.” Claw and hammer toes can be a sign of distal muscle atrophy and neuropathy. Claw toes and hammer toes increase pressure on the metatarsal heads, dorsal interphalangeal joints and apices of the toes. Increased ground reaction forces may lead to callus formation and ulceration at the metatarsal heads and digital apices. Hallux rigidus (complete loss of dorsiflexion at the first metatarsophalangeal joint), or hallux limitus (partial loss of dorsiflexion at the first metatarsophalangeal joint) also predisposes to ulceration, since the toe-off phase of gait requires 45 degrees of metatarsophalangeal joint dorsiflexion. Limitation in dorsiflexion of the hallux can lead to increased ground reaction forces at the joint with callus formation and increased risk of ulceration. It is important to note that any pedal deformity has the potential to increase local tissue pressures and can be the underlying cause of tissue breakdown. Bunions (hallux abducto valgus deformities) for example, place the medial aspect of the first metatarsophalangeal joint at risk. This is due to an increased forefoot width, which causes excessive pressure within the toebox of standard fitting shoes. The identification and management of such deformities is critical in the prevention of ulceration.
Management
Treatments for such deformities involve conservative offloading with padding, the use of custom orthoses and specialty shoes. More aggressive management would involve orthopedic surgery, which should comprise of joint fixation, arthroplasty or digital amputation. The goal of any treatment plan is to reduce the mechanical stress at the problematic area, thus reducing risk of skin ulceration.

5. Charcot Foot (Neuropathic Osteoarthropathy, Diabetic Neuroarthropathy)

Definition
Charcot Foot is a progressive condition characterized by joint dislocation, pathological fractures and severe destruction of pedal architecture. This may result in debilitating deformity or amputation. The condition is associated with severe peripheral neuropathy and the most common etiology is diabetes mellitus. Eichenholz’s classification divides osteopathy into developmental, coalescent and reconstructive stages.

Etiology
The etiology of Charcot foot most likely is a combination of both the neuro-vascular and neuro-traumatic theories. It is generally accepted that trauma superimposed on a severely neuropathic extremity can precipitate the development of an acute Charcot foot. With the development of autonomic neuropathy, there is an increased blood flow to the foot, resulting in osteopenia and a relative weakness of the bone. The presence of sensory neuropathy renders the patient unaware of the precipitating trauma and often profound bone destruction that occurs during ambulation. A vicious cycle ensues whereby the patient continues to walk on the injured foot, thereby allowing further bone and joint damage to occur.18

Did You Know?
In a person with diabetes, a warm swollen red foot, without a portal of entry for skin infection should be considered an Acute Charcot Arthropathy until investigations have proven otherwise.

Diagnosis of Acute Charcot Arthropathy

a) Clinical Examination
The following characteristics, in the presence of intact skin, are often diagnostic of acute Charcot arthropathy:
- Profound unilateral swelling
- Increased skin temperature
- Erythema
- Joint effusion
- Bone resorption (seen on x-ray) in an insensate foot
In more than 75% of cases, the patient will present with some degree of pain in an otherwise insensate foot. In more than 75% of cases, the diagnosis is complicated by the fact that patients first present with concomitant ulceration, which raises the possibility of cellulitis and osteomyelitis.

b) Investigation

- X-Rays - plain radiographs are invaluable in ascertaining the presence of osteoarthropathy. In most cases, no further imaging studies will be required to make a correct diagnosis. However, with a concomitant wound, it may be difficult to differentiate between acute Charcot arthropathy and osteomyelitis based solely on plain radiographs.

- Laboratory and other imaging studies may be required including: white blood cell count (WBC); erythrocyte sedimentation rate (ESR); a bone biopsy; nuclear medicine scans; magnetic resonance imaging (MRI).

Did You Know?
The key to preventing further deformity, while waiting for further investigations and specialist referral, is non-weight bearing of the affected limb. Ideally an urgent referral should be made to a podiatrist or orthopedic surgeon.

c) Management of Acute Charcot Arthropathy

- Immobilization and reduction of stress are the mainstays of treatment for acute Charcot arthropathy. Use offloading modalities, such as wheelchair, crutches, walker, casts, braces/splints, surgical shoe with insert.

- Following a period of offloading, a reduction in skin temperature and edema indicates the stage of quiescence, at which point, the patient progresses into the post-acute phase of treatment.

- Progression to protected weight bearing is permitted, usually with the aid of some type of assisting device.

- Through the use of appropriately applied total contact casts or other offloading modalities, most patients may safely ambulate while bony consolidation of fractures progresses.

- The mean time of rest and immobilization (casting followed by removable cast walker) prior to return to permanent footwear is approximately 4-6 months.

Reconstructive surgery may be considered if a deformity or instability exists that cannot effectively be controlled or accommodated by prescription footwear or bracing. If the arthropathy is identified in its early stages and non-weight-bearing is instituted, surgery is usually unnecessary.