# DIABETIC FOOT

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Diabetic foot condition refers to a spectrum of foot conditions brought about by the diabetic disease processes like neuropathy, angiopathy, immunopathy and malnutrition. It includes conditions such as: hypertrophic callositas, frank trophic changes like dry scaly skins with deformed nails; foot deformities like clawing of the toes and rocker bottom foot, as seen in Charcot foot. These different conditions make the foot at risk. And since the feet are subjected to repetitive stresses of weight bearing, this repetitive trauma causes damages to these already "at-risk" feet producing the other end of the DM foot spectrum, namely: non-healing wounds, infections such as felons, abscesses & osteomyelitis to frank wet or dry gangrene. This is also the reason why they are more common than that of the upper extremity coupled with the accepted fact that the hand has a better blood supply than that of the foot. From "at-risk" condition, it only becomes emergent when it poses danger to the viability of the foot itself or its parts or even to the life of the patient himself. Diabetic ulcers have been shown to precede amputation, at any level, in 85% of cases. Management of these cases become difficult because of the diabetic processes together with their often associated co-morbidities like impaired renal function and other cardiac conditions.

## Peripheral Neuropathy (PN)

Pathophysyiology

PN has been associated with higher incidence of amputation in the lower extremity diabetic wounds. While sensory neuropathy is most often implicated in DM hand, in DM foot, the roles of motor & autonomic neuropathies are also equally implicated as well, thus the often used term, Triopathy. Although still poorly understood, the pathophysiology of PN postulated includes: (1) Loss of myelinated & non-myelinated nerve fibers; (2) Ischemic & focal nerve loss due to poor micro-circulation & oxygen delivery; and (3) Slowing of nerve conduction by hyperglycemia itself.

Sensory

Sensory neuropathy presents as varying degrees of numbness and dysesthesias, classically in a "glove & stocking distribution". The involved extremity is thus unable to respond normally to repetitive mechanical stresses resulting in tissue breakdown (ulcer). This becomes a portal of entry for pathogenic bacterias.

Motor

Damage to the innervation of the intrinsic foot muscles leads to imbalance between the flexors & extensors of the toes or foot. This produces anatomic foot deformities e.g. claw toes and permanent bony prominences. These bony prominences become pressure points (abnormal stress risers) and areas for for skin breakdown.

Autonomic

Autonomic neuropathy decreases the sweat and oil gland function of the skin as well as arterio-venous shunting. A state of dry skin is produced which will be prone to spontaneous cracking and peeling which may also result in an ulcer. This, together with the abnormally hard and deformed nails are the trophic changes we see in DM foot.

Charcot foot is a neuropathic condition often associated with Syphillis & Tabes Dorsalis. It may also be seen in DM foot. Motor & autonomic neuropathies are both implicated in this condition. Muscle & joint laxity produces collapse in the foot arches. Autonomic neuropathy on the other hand, through AV shunting phenomenon, causes impairment of vascular smooth muscle function, increasing abnormal increase blood flow to the bones, producing resorption, osteolysis & bone fragmentation. This may lead to a permanent deformity called "rocker-bottom foot" if not detected & managed early on and allowed to progress. This again produces abnormal pressure points and may be a site of ulcer development especially in patients with sensory neuropathy as well.

**Vasculopathy**

Peripheral arterial occlusive disease (PAOD) is a hallmark of Diabetic foot. It involves large vessels, the femoropopliteal and smaller vessels below the knee, the tibials & peroneals. It is often diffuse, bilateral & progressive. It is a major contributory factor in ulcerations in 50% of cases. Hyperglycemia causes endothelial dysfunction and smooth muscle abnormalities in peripheral arteries. Constriction is produced by the decrease in the previously mentioned endothelium-derived vasodilator, Nitric Oxide. Hyperglycemia itself may also increase production of Thromboxane A2 which causes vasoconstriction and a known platelet aggregation agonist. This constrictive phenomenon, in turn, causes a decrease in oxygen and nutrient delivery essential in normal wound healing. In severe cases, cell necrosis & gangrene may result. Other diabetes-mediated contributors to PAOD are smoking, hypertension & hyperlipidemia.

**Immunopathy, Nutrition & Wound Healing**

Diabetics, despite the obvious abundance of sugar in their circulation are considered malnourished due to the imbalance of CHO, Protein & Fat metabolism. Proteins are especially essential in normal wound healing and its relative scarcity may be due to both a decrease in production (abnormal protein metabolism) and a decrease in delivery due to PAOD. To aggravate the situation, poor wound healing may be complicated by the presence of infection which may be caused by a poor immune response, a condition that may be caused by a decrease in serum albumin. Chemotactic property of PMN's are observed to be altered in this condition.

**Emergency Management of Diabetic Foot Infections**

Worldwide, the population of diabetics is increasing due to increase in population growth, aging, urbanization and increasing prevalence of obesity & physical inactivity. The number is expected to double from year 2000 to year 2030. This will impact even underdeveloped and developing countries like the Philippines, which is expected to be #9 worldwide in total

diabetic population by year 2030. Diabetic foot problem therefore can be a potential public health concern. A diabetic has a 10-fold risk of being hospitalized for infections of the bone and soft tissue of the foot. They also have a 30X higher lifetime risk of lower extremity amputation compared to the normal population.

Limb & life-threatening foot infections in diabetics are infections caused by necrotizing fasciitis, any type of circumferential gangrene or any infection with systemic toxicity or metabolic instability. Our treatment goals, in decreasing order of priority, remain the same: to save the patient's life; to save his limb; and to amputate at the most physiologic level. To achieve our goals, the patients should receive optimal pre-op, intra-op and post-op cares by their attending physicians which can only be possible through multi-disciplinary approach. This may involve the Internist (Endocrinologist, Cardiologist etc), Orthopedic Surgeon, Vascular Surgeon, Physiatrist (in cooperation with Physical Therapist & Orthotist), Nurse Educator and Nutritionist as the situation so needs. Optimal care decreases the high mortality & morbidity rate anticipated in the management especially of severe cases. It is dictated by the urgency of the situation and the availability of resources, both human & facility. This is not to mention the availability of financial resources of the patient & his family during this time of crisis.

**Pre-operative Care**

Younger patients with less severe cases may only need the basic lab exams like; plain radiographs of the chest & involved extremity; CBC & FBS. Patients are routinely hydrated, glycemic control initiated & broad spectrum antibiotics covering mixed infections promptly started. Swabs are not routinely done pre-op as superficial swabs are not reliable. In less severe cases, such as "enclosed" abscess in patients with less metabolic and systemic constitutional signs & symptoms, antibiotics for G(+) organisms like Staph or Strep may only be what is needed as the start-up empiric antibiotic of choice.

Older patients & those with more constitutional signs & symptoms may need more pre-op preparation. Mortality rates in these cases may go as high as 60-70% in some studies. In addition to the enumerated exams, Internists may require the following: ECG & 2-D Echo, serum electrolytes and even blood cultures. Blood products are given pre-op if necessary with caution against pulmonary congestion and a replacement target hemoglobin value of 10 or 11 may be safer.

**Intra-operative Care**

The goal of surgery ideally is the complete eradication of infection and removal of devitalized tissues. This is only possible realistically with amputation way above the infected area. As Orthopedic Surgeons who are concerned with the eventual post-op function of the patient, a compromise between biologic level & physiologic level of amputation must be considered & therefore complete eradication might not be at all possible. This is usually seen with most debridements especially if patients in serious condition are operated on urgently. In this instance, as much infected and devitalized tissues are removed, as the intra-op condition of the patient may permit. A second stage debridement is then planned at a later

date when the condition of the patient is more optimal and a lengthier more meticulous surgery can be carried out.

**Anesthesia & Prepping**

In most instances, surgery is carried out under regional anesthesia. However in compromised patients, the Anesthesiologist might be more comfortable with General Anesthesia. The lower extremity is prepped & draped up to just below the inguinal area in case the surgical procedure have to be carried out more proximally. Tourniquet is usually not necessary and might even obscure judgment as to the viability of the tissues.

**Debridement**

Pus & all necrotic tissues are removed until healthy bleeding tissues are seen. Towards the end of the procedure, it is good practice to milk the tissues around the surgical wound to see if the infection have dissected further & therefore a need to extend the exposure & debridement. At this point, specimen is collected for gram stain and culture. Copious irrigation with saline-antiseptic or saline-antibiotic solution is routinely done after adequate hemostasis. Wounds are generally left unsutured & dressed with saline-antibiotic (usually Gentamycin) mixture-soaked gauze & topped off with dry gauze then wrapped with elastic bandage. If available, vacuum-assisted wound coverage may be done. This type of wound coverage procedure has been shown to be a very good alternative as it is known to hasten production of granulation tissue.

**Amputation**

*Biologic* level of amputation is at the level visually devoid of infection and the surrounding soft tissues are healthy enough for eventual wound healing, whether by primary or secondary intention. The 4 C's of muscle tissue should be observed. These are: 1. Good *Capacity to bleed*, 2*. Contractile* muscles, 3. Muscle *Color* is beefy red or pinkish and 4. Muscle *Consistency* is healthy and not friable. Bahebeck et al recommends 2 – 5 cm margin above visible necrosis and below the most distal palpable pulse. Considerations regarding final functional outcome or *physiologic* level of amputation should be given equal importance. If at all feasible, an amputation at a level which is weight bearing, at the foot or ankle is preferable over one that would require a prosthesis, as in below or above knee amputation. If more than one ray needs to be removed, an outright transmetatarsal amputation should be considered. Removal of 2 rays will make the weight bearing surface area of the foot too small making it susceptible to pressure sore formation. Besides, balance can also be a problem in this instance.

Stump closure should be done if there are no visible signs of infection and the surrounding tissues are healthy. If both are not present, or when in doubt, stumps are better left unsutured. A 2$^{nd}$ stage stump closure may be done at a later date when conditions are more optimal or the wound may be allowed to heal by secondary intention. Vacuum-assisted wound dressing method may also be used. Specimen for gram stain & culture may be

collected intra-operatively if the operative site shows signs of gross infection. Otherwise collection of the specimen may be done post-operatively from the amputated part.

**Post-operative Care**

Empirical antibiotic(s) is continued post-operatively while awaiting for the culture result. It is then promptly replaced with the appropriate culture specific antibiotic(s) and continued for 10 – 14 days. Wounds are inspected 1 – 2 days post-op together with removal of the drain.

Unsutured stumps and post-op open wounds can be managed in several acceptable methods. Stumps may be closed primarily in 3 – 5 days when the wound is clean as evidenced by beginning granulation. Wounds that cannot be closed are cleaned with peroxide solution & dressed with gauze soaked with Dakins' solution daily or alternatively with vacuum-assisted method every 3 – 5 days and allowed to granulate. This may then be covered with split thickness skin graft. Using the wet-to-dry dressing by Dakins' solution, majority of the open wounds, irregardless of size, may eventually heal completely by secondary intention in 1 – 5 mos. This is a personal observation in more than 90% of patients who declined to undergo skin grafting. They were discharged from the hospital after four conditions were met: 1. Blood sugar is controlled; 2. Wound is clean & viable; 3. Appropriate culture specific antibiotics is identified ( given for a total of 2 weeks) and 4. They can manage to have their wounds cared at home, in a similar routine being done in the hospital.