MANAGEMENT OF DIABETIC FOOT - A PROSPECTIVE STUDY

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ABSTRACT

BACKGROUND
Ulcer is break of continuity of epithelium. The covering could be skin or mucous membrane. Diabetic ulcer develops due to neurovascularopathy. It causes microangiopathy. It starts on undersurface of toes. It is painless ulcer and edges are indurated and inflamed. Diabetic foot is the commonest complication of diabetes, commoner after 40 years of age. Type 2 NIDDM patients are 85% prone to develop diabetic foot ulcers.

MATERIALS AND METHODS
70 patients were studied retrospectively between January 2014 and January 2016, admitted in our unit of tertiary care hospital. Majority of ulcers were diabetic.

RESULTS
Proper examination, control of blood sugar and local care of ulcer with dressing. Treatment is control of diabetes and local care of ulcer. Surgical treatment was debridement of ulcer.

CONCLUSION
Multi-team approach for primary management of composite wounds should be understood. Emphasis should be for proper examination, local care of foot and proper footwear.

KEYWORDS
Ulcer, diabetes, neuropathy, microangiopathy.


BACKGROUND
Diabetic foot is one of the commonest complication of diabetes. The number of diabetic patients are expected to double between 2000 and 2030. It is common after 40 years of age, but less common in Caucasians. Ulcer is a break in continuity of epithelium. Type 2 NIDDM patients are 85% prone to develop diabetic ulcers. Diabetic ulcers are type of metabolic ulcers that develop due to neuropathy. Rollo in 1797 described improvement of officer placed on meat diet. The term mellitus distinguishing it from diabetes insipidus was attributed to Rollo. The relation of diabetes with ulcer was described by John Rollo.1,2 Complications of diabetes are leg, foot lesions 58%, coronary artery disease 30%, stroke 6%, ophthalmic 3%. Predisposing factors of diabetic ulcers are uncontrolled diabetes, vascular disease, neuropathy and infection.

MATERIALS AND METHODS
We studied 70 patients of ulcer foot from January 2014 to January 2016, admitted in our unit of tertiary care hospital. Majority of foot ulcers were diabetic; 52 patients were diabetic ulcers, 7 patients were vascular, 11 patients were bacterial, 4 patients were other causes.

DISCUSSION
Ulcer deformity, discoloration, blisters, skin, fissures were examined. Clinical examination should assess patient’s musculoskeletal system, posture, gait, temperature, neurological system, pulses and ankle brachial index. Investigations required are blood sugar estimation, protein profile, glycosylated haemoglobin, culture sensitivity, x-ray foot, MRI scanning. Treatment were regular dressing, debridement, control of blood sugar. Patients were put on insulin after sliding scale. Majority of patients healed with proper dressing and debridement. Patient’s bone was exposed, those requiring flap coverage were referred to plastic surgeon.

RESULTS
Treatment of diabetic ulcer was regular dressing, debridement, control of blood sugar. Patients were put on insulin after sliding scale. Majority of patients were healed with proper dressing and debridement. Seven patients whose bone was exposed requiring flap coverage were referred to plastic surgeon. Vascular ulcers were referred to vascular surgeon for further management.

Ulcer is discontinuity of skin or mucous membrane which occurs anywhere in body. Ulcers consists of floor, edge, margins and base. Types of ulcer could be traumatic, inflammatory, vascular and neoplastic. Ulcers are described for location, floor, red granulation tissue for healing ulcer, necrotic for spreading, pale for tubercular. Edges are sloping for traumatic, punched for gummatous ulcers, undermined for tubercular ulcers, raised for basal cell carcinoma, everted for squamous cell carcinoma. Palpation of edges, bleeding, surrounding area, lymph nodes, peripheral vessels, sensation, joint function, varicose veins and central nervous system.
Investigations are blood picture, chest x-ray, duplex scan, angiography, biopsy of ulcer. Treatment of ulcer is antibiotics after obtaining culture and sensitivity, dressing by betadine, hydrogen peroxide and eusol. Granulation tissue should be excised. Treatment of chronic ulcer is ultraviolet rays, amniotic for epithelisation, chordon for formation of granulation tissue and skin grafting. Debridling solutions are hydrogen peroxide releases nascent oxygen destroys anaerobic bacteria. Eusol is hypochlorite solution for debridement. Hydrophilic colloid are adhesive absorbs fluid, necrotic tissue autolysis, impermeable to fluids and bacteria. Alginate are polymer absorbs exudates. Platelet derived growth factor, endothelial growth acts through tyrosine kinase receptor, stimulates growth of cells and angiogenesis stimulating repair. Traumatic ulcers occur on areas of bony prominences, painful ulcers and heal with dressing, antibiotics. Venous ulcers are superficial, painless ulcers cause periositis tibia. Arterial ulcer is painful in younger patients. Neuropathic ulcer develops from diabetic neuropathy, nerve injuries and pressure points. It develops infected discharge, osteomyelitis. Treatment is immobilisation of foot by plaster, posterior slab. Trophic ulcer is due to malnutrition, trauma. Infection is bacteria fusiformis and Borrelia venticii develops pustule, bursts and forms ulcer. The edges are undermined, slough, seropurulent discharge. Treatment is broad-spectrum antibiotics and dressing. Thrombotic ulcers are due to deep vein thrombosis. Clinical symptoms are pain, deep ulcer, pigmented. Dorsiflexion of foot causing calf pain called Moses’ sign. Treatment is rest, antibiotics, crepe bandage. Bazar ulcer is due to ischaemia of lower limb managed with antibiotics and sympathectomy for obese young females. Diabetic ulcers manifests due to neuropathy. It is distal, stocking type of distribution. Nerve damage is due to formation of sorbitol from sugar. Sorbitol causes demyelination of fibres, decreased tendon reflexes. Pain, temperature sensations are decreased. Clawing of toes results in neuropathy of muscles of foot. There is absence of sweating, resistance of infection.

The phagocytic activity of leucocytes is reduced and if patient develops ketacidosis granulocyte and chemotaxis is reduced making them susceptible to infections. Diabetic angiopathy results from accelerated atherosclerosis, produces thickening of basement membrane. Neuropathy with secondary infection forms diabetic ulcer. Ulcer starts with minor trauma and develops cellulitis. Anaerobic infection produces multiple abscesses producing ischaemia, gangrene, osteomyelitis and sepsicaemia. Investigations are haemoglobin, blood sugar estimations, culture sensitivity, x-ray, ECG, blood urea, serum creatinine and duplex scanning. Treatment of diabetic ulcer is control of diabetes, infection and local treatment of ulcer and surgery for non-healing ulcer. Control of diabetes is by putting patient on sliding scale and insulin or oral hypoglycaemics. Control of infection is by antibiotics after culture sensitivity. Local treatment of diabetic ulcer is debridement, dressing and skin grafting. Revascularisation of diabetic ulcer is balloon angioplasty, bypass surgery from popliteal to tibial or pedal artery using saphenous vein. Patient education is necessary for rubber shoes, not walking barefoot, keeping foot dry, cleaning of foot and proper shoes for oxygenation of toes. Spreading ulcer is debrided and treated with skin grafting and flaps. Abscesses are treated with drainage; gangrene is treated with disarticulations, excision; cellulitis is treated with fasciectomy. Ischaemic limb is treated with revascularisation and bypass surgery.

Combination of growth factors leads to predictable success. PDGF is useful for diabetic and pressure sores. Epidermal growth factors for venous ulcers, fibroblast growth factors for pressure ulcers and granulocyte macrophage stimulating factor for diabetic ulcers. Artificial adjuncts is Alloderm acellular human matrix, Integra bovine collagen and chondroitin sulphate for silastic membrane.

Dermagraft is polyglactin mesh for diabetic ulcer. Stem cells and gene therapy are being tried. Use of adeno virus and plasmids for wound is being tried. Dressings are biocompatible alginate mesh, non-absorbent dressing and Sorbsan or Granulix. Collagen based dressing, a natural polymer is used for healing of ulcers. Healthy surface is grafted with skin graft, consists of epidermis and part of dermis for clean wounds. Wound coverage is putting muscle flaps. Muscle flaps are myocutaneous flaps supplied by myocutaneous perforators supply, skin and subcutaneous tissues were used where bone is exposed. Free flaps are transferred from any part of the body to recipient vessel. Tissue expanders for management of wounds is to expand with tissue expander for reconstruction of created defect. The advantages are minimal morbidity, quality and texture of adjacent skin are excellent.

Diabetic foot ulcers is dreadful microvascular complication causing neuropathy, peripheral artery disease and foot deformity. Evaluation of bone infection is by plain radiographs and magnetic resonance imaging. Management is required on focussing and prevention, learning, aggressive intervention and optimal use of foot wear. Effective management of diabetic foot ulcers is total contact casting, debridement, silver dressing, oedema management by compression therapy, negative pressure wound therapy, offloading boots, patient education and biological dressing.

Foot ulcers are a major complication of diabetes. Growth factors derived from blood platelets, endothelium and macrophages could be important treatment for these wound s; 28 randomised trials for 2365 patients, the result was based on platelet derived wound healing formula and recombinant human platelet derived growth factor. Growth factors could increase the likelihood for complete healing of foot ulcers.

Transcutaneous oxygen pressure was used to measure microcirculation. Good microcirculation and better wound healing. Weight bearing exercises programme designed to distal and proximal lower extremities to reduce peak forefoot pressure of patients of diabetic neuropathy. Diabetic foot ulceration is a serious medical condition and causes are neuropathy and high plantar pressure. Heel and midfoot bears minimal weight of body and high pressure is under forefoot. Treatment is by reducing peak forefoot pressure and spreading plantar pressure by modification of gait pattern.

Weight bearing exercises programme designed to distal and proximal lower extremities to reduce peak forefoot pressure of patients of diabetic neuropathy. Recent studies is advocating treatment of diabetic ulcers by anmiotic derived tissue graft coupled with contact casting proved encouraging. Diabetic mellitus is chronic disorder of glucose metabolism with multisystem involvement. Diabetes accelerates the atherosclerotic process and major risk for peripheral artery disease. Diabetic foot complications are because of ischaemia, neuropathy and infection predominantly small vessel involvement.

Soaking feet in water in diabetic ulcer is harmful. Neuropathy affects function of sweat glands and thickening of capillary basement membrane causes neutrophil migration and dampening tissue response to infection.
Diabetic patients present with claudication and rest pain. Ankle brachial pressure index is tested to determine ischaemic limb. Severe ischaemic limb is less than 0.44 - 0.55 and ankle brachial pressure of 55 mmHg.

Toe pressure less than 30 mmHg requires revascularisation, essential to promote tissue healing. Tissue healing is likely to occur with conservative measures of TcPO2 greater than 50 mmHg and revascularisation is warranted for TcPO2 less than 30 mmHg. CT scan, MRI, subtraction angiography and bone biopsy are required for diagnosis. Primary prevention is risk factor of antiplatelet therapy, smoking, hypercholesterolemia and hypertension. Treatment of neuropathic ulcer is restricted weight bearing. Topical dressing, weight offloading, protective footwear, orthosis, contact cast, crutches and management of ischaemia. Ischaemic ulcer requires antibiotics after culture and sensitivity. Hyperbaric oxygen therapy has been used for wound healing and limb salvage.

Revascularisation with autologous veins and saphenic vein grafts are tried. Revascularisation of 1000 vascularisation showed patency of 78.2% after 5 years.

Advances of endovascular procedures showed improved results of below knee intervention. Meta-analysis of angioplasty compared to crural bypass reported similar limb salvage and secondary interventions were higher for angioplasty.12

CONCLUSION
Diabetic foot is one of the commonest complication of diabetes. Emphasis should be on proper examination, because only 12 - 20% of patients are examined properly. Management should focus on diagnosis by x-ray, pus culture, MRI, meticulous dressing, debridement and control of blood sugar. Most diabetic patients are controlled with insulin in 2 - 3 weeks. Reducing peak plantar pressures by weight bearing exercises and proper footwear forms basis of management of diabetic ulcer and neuropathy. Non-weight bearing is essential. Weight relief shoes prevents foot trauma. Annual foot screening of diabetic patients will reduce incidence of foot related trauma to insensitive foot. Annual examination of feet, patient education, self-care, quality of footwear to reduce areas of foot pressure prevents ulcers, improves gait, stabilises foot and causes relief of pain.

Multidisciplinary approach is required with emphasis on patient education, reducing the incidence of diabetic foot complications.13

REFERENCES