THE DIABETIC FOOT
CURRENT PROBLEMS AND THEIR MANAGEMENT

Ashok Kumar Das

Introduction

Diabetic foot disease is an important problem confronting the diabetologists, internists and surgeons[1]. The advent of insulin overcame the acute problems of ketoacidosis and infection, but could not prevent the vascular and neurological complications. Foot is the most vulnerable part in a diabetic. It is exposed to frequent trauma and requires a sensitive sensory protection, which is often lacking in a diabetic. The foot, being farthest away from the central nervous system and hemodynamically disadvantageously placed, becomes the common site of complicated lesions. Diabetics with foot complications occupy more hospital beds in UK than by those with all other complications taken together[2]. In USA upto 70% of all non-traumatic amputations occur in diabetics[3]. Twenty five percent of Joslin Clinic admissions are for patients with foot problems. Almost half of these patients need amputations and more than half of the amputated patients die.[4]

Therefore, it has been rightly said that "nowhere else in the body do we see so clearly the ravages and magnitude of diabetic vascular complications and neuropathy as in the diabetic foot"[5].

The extent of diabetic foot problem in the Indian context is not entirely apparent. The incidence of neuropathy is quite high even though peripheral vascular disease is reported to be low. However, bare foot walking, infections and lack of health education compound the diabetic foot problems in India.

AETIOLOGICAL CONSIDERATION :

Although the aetiopathogenesis of diabetic foot disease is multifactorial, three main factors, namely neuropathy, ischaemia and infection lead to tissue necrosis and ulcer formation[5]. The other factors which may directly or through above three ways contribute to genesis of diabetic foot ulcer include foot biomechanics and weight bearing, peripheral vessel calcifications, trauma, (possibly) diabetic autonomic neuropathy and microangiopathy and diabetic skeletal disease. The three main factors would be discussed.

Associate Professor, Department of Medicine, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry 605006.
Report of the experiences gained during a Commonwealth Fellowship in Diabetes (1986-87) at Diabetic Department, King's College Hospital, London, United Kingdom.

Neuropathy in Diabetic Foot

Multiple factors such as blood glucose concentration, blood lipids, structure of myelin sheath and its permeability, axonal flow and micro and macroangiopathy of the peripheral nerves contribute to the production of diabetic neuropathy[7].

Blood Flow in Diabetic Neuropathic Foot

Recent studies have shown that the blood flow is increased in diabetic foot. The latter is due to the arterio-venous shunting and dilated and stiff peripheral arteries[8].

Edmonds and Watking have shown by means of doppler studies that there is increased velocity of forward flow in the peripheral arteries of the neuropathic limb[9]. The pulsatility index which is inversely proportional to the quantity of the blood flow, is markedly reduced in diabetic foot. The normal doppler flow pattern is triphasic : a forward flow in systole followed by a reverse flow and a further short forward flow in diastole. In diabetics there is an increased forward flow with the absence of reverse flow. By means of venous occlusion plethysmography, big toe and mid foot blood flow was shown to be five times greater in diabetic neuropathy than in normals. Ward et al have shown that the skin temperature is increased in the neuropathic limb[10]. This has been further corroborated by Ucrcher who reported a mean skin temperature of 33.5°C in neuropathic foot compared to 25.8°C in controls.[11] This raised skin temperature produces warm neuropathic foot (in contra-distinction to cold ischaemic foot). Diabetic autonomic denervation is the most important single factor responsible for the increased blood flow in diabetic foot.

Skeletal Changes in Diabetic Foot

Due to increased blood flow to the lower limb there in enhanced blood supply to the bones of the diabetic foot. Radio-isotopic studies have shown that the uptake of technetium methylene diphosphonate is increased both initially and at four hours[12]. Blood flow is maximum in diabetic charcot's arthropathy.

Measurement of Arteriovenous Shunting in Diabetic Neuropathic Foot

J.D. Ward first observed the prominent turgid veins over lower part of calf and dorsum of foot in recumbency in a diabetic foot.[10] Using radioactive labelled human albumin microspheres, arteriovenous shunt volume has been measured to be 8.48% in diabetic neuropathy as against 5% in controls.

Stiffening of Arterial Wall

The medial wall calcification in the peripheral vessels in the lower limb raises ankle brachial systolic pressure and shortens transit times of pulse wave.

Edmonds and Watkins have shown calcification in the arteries of foot and ankle in 16 out of 20 patients with severe diabetic peripheral neuropathy aged between 22 and 50 years. In Charcot's diabetic neuroarthropathy vascular calcification is found in about 90%[14].

Diabetic autonomic neuropathy (sympathetic failure) is the main mechanism of increased production of blood flow[15].

Effect of enhanced blood flow in the Neuropathic Foot

Ward et al have shown that rapid increase in flow of blood bypasses small vessels and the capillary nutrient circulation and results in a relative distal ischaemia[10].

The other effects of increased blood flow in a neuropathic diabetic foot are: 1. neuropathic oedema 2. painful diabetic neuropathy.

Neuropathic Oedema

Neuropathic oedema was first described in 1893. Rundles and Martin recognised this as a manifestation of diabetic neuropathy after about 50 years[16]. The enhanced blood flow, vasodilatation and arteriovenous shunting, all arising out of sympathetic denervation lead to abnormal venous pooling and oedema. Edmonds et al postulated use of sympathomimetic agents for stimulating vaso-constriction to reduce this oedema and ephedrine used by them was effective in its treatment[14].

Painful Neuropathy in Diabetic Foot[17]

Watkins et al have demonstrated high peripheral blood flow and increased warmth in the acutely painful diabetic neuropathic foot. Cutting down the blood flow by means of a sphygmomanometric cuff reduced the blood flow and the pain, and this opens new vistas (in the form of therapeutic reduction of blood flow) as a possible treatment of diabetic neuropathy.

Diabetic Charcot's Neuro-Arthropathic foot and increased Blood Flow

Rarefaction and demineralisation of bone secondary to enhanced blood flow has been documented in the experimental animal. Insensitive feet, coupled with demineralisation of bones cannot withstand the mechanical stress and result in fracture leading to gross deformity of Charcot's neuroarthropathy.

Ischaemia

Ischaemia is the second major factor in the causation of diabetic foot syndromes. Atherosclerosis of the large vessels of the leg in a diabetic is often multisegmental, distal and bilateral. It has been shown that there is a predilection of atherosclerosis for the vessels below the knee in diabetics.

Small Vessel Disease in Diabetic Foot

Until recently the role of microvascular disease in the genesis of diabetic foot lesion has not been clearly elucidated. Parving and Resmusen have demonstrated functional abnormality in the form of leakage of albumin from the capillaries to the interstitium; however an occlusive microvascular disease in the diabetic foot has not been clearly demonstrated.[18]

Trauma and Infection

The tissue necrosis usually sets in following minor trauma which gets complicated by infection. The presence of neuropathy makes the feet insensitive and the diabetic patient is often not aware of even a severe mechanical trauma and gross infection. However in the predominantly neuropathic feet owing to an excellent blood flow the major infection can be contained to some extent; but the ischaemic foot has no communication in and between the plantar and dorsal arterial arches, and infection advances rapidly.

CLASSIFICATION OF DIABETIC FOOT ULCER

From the practical point of view the diabetic foot can be divided into two major types : (1) Ischaemic diabetic foot and (2) Non Ischaemic neuropathic diabetic foot[5].

In the ischaemic foot the occlusive vascular disease is the main factor, whereas in the nonischaemic neuropathic foot there is increased blood flow, and peripheral neuropathy predominates.

The major differences between the two types are depicted below :

	Non-ischaemic neuropathic foot	*Ischaemic foot*
Nature of foot	Warm Numb Dry	Cold Sensitive
Pain	Painless	Painful
Foot pulses	Palpable	Absent
Site of ulcer	Plantar surface Pressure points	Sides of digits Rest pain
Complications	Neuropathic ulcer Neuropathic charcot's joint Neuropathic oedema	Claudication Ulceration/Necrosis Gangrene

Practical Value of this Classification[19]

The clinical distinction of diabetic foot ulcers into ischaemic and non-ischaemic neuropathic group has practical value in rationalising its management.

The non-ischaemic neuropathic ulcer tends to heal quickly with rest and antibiotics. Chiropody is the mainstay of treatment. The recurrence of ulcer is prevented by redistribution of weight bearing forces by moulded insoles. In the ischaemic foot ulcer, medical management is successful in three-fourth of cases; the remainder need arterial reconstruction or angioplasty.

LABORATORY ASSESSMENT OF DIABETIC FOOT

Assessment of peripheral neuropathy and evaluation of peripheral arterial status are the two important investigations in a diabetic foot. Recently there has been an upsurge of interest in using these investigations in diabetic foot care and quantify the deficit accurately[20].

Assessment of Diabetic Peripheral Neuropathy[21]

Accurate sensory testing in diabetic neuropathy is of paramount importance in the diagnosis, objective quantification and monitoring natural evolution or effects of therapy. These involve testing for (1) Vibration perception Threshold (VPT), (2) Thermal Discrimination Threshold (TDT).

Vibration Perception Threshold

Vibration perception threshold (VPT) is assessed by a Biothesiometer, which has a rubber tractor that vibrates at 100 Hz when operating on 50 Hz mains. There is a linear scale which depicts the applied voltage. The VPT is measured on the plantar aspect of the distal phalanx of the big toe.

The operation of the instrument is simple. The subject is seated comfortably and is first made to feel what vibration sense is, by testing over a bony site. The amplitude of the vibration is gradually increased from zero, until the subject just appreciates it. The VPT is taken as the mean of three recordings. The values can be expressed in the arbitrary scale of volts or as amplitude of vibration which is proportional to the square of the applied voltage. However the former is simple and gives adequate quantification. The accepted range of the normal subjects and their relationship to age and sex has been described in the literature[22].

Thermal Discrimination Threshold (TDT)

Thermal discrimination threshold can be quantitatively assessed by using 1. Marstock stimulator 2. Automated thermal threshold tester[23,24,25].

Marstock Stimulator *(Somedic, Stockhom, Sweden)*

It works on the peltier principle. The metal element (or the probe) which can be either heated or cooled is held in position without pressure. The subject is given a reversing switch in the hand and is asked to press as soon as the thermode is felt to be warm or cool. The temperature of the probe is measured by a

thermocouple and recorded on a chart from which TDT can be readily calculated.

Automated thermal thereshold tester *(Glasgow System)*

The method of examination is a "psychophysical" one and it uses "forced choice technique". The instrument comprises of a thermode assembly consisting of a thermode, a thermode interface unit with a digital thermometer and visual display unit and water bath with a thermostat. Further it has a microprocessor unit and a subject response box communicating with the computer. The microprocessor in conjuction with the computer controls the temperature of thermode and duration of stimulus (hot or cold) over two separate time periods indicated by two lights (1 and 2) in the subject response box. The microprocessor processes the return signals from the patient response box, controls the up and down transformation of the temperature and gives the computerised value of hot threshold and cold threshold.

Das et al[26] conducted a comparative evaluation of automated thermal thereshold tester and the Marstock stimulator in the assessment of diabetic neuropathy.

It was found that delineation of diabetic neuropaths from normal was achieved by either instrument. However in the Marstock stimulator the amount of stimulus applied was large and thus delineation was much better. Many subjects found Marstock easy to perform. In the automated thermal tester a greater degree of concentration and patient cooperation was called for. The Marstock stimulator measures the hot TDT whereas the automated thermal tester measures the hot threshold and cold threshold. They are not the same, but possibly complement each other[26].

Evaluation of Peripheral Arterial Status :

Although angiography and visualisation of the vascular tree is taken as the gold standard for evaluation of peripheral arterial disease, currently, non-invasive vascular laboratory has assumed an important role in the evaluation of peripheral ischaemia and the diabetic foot.[27] The various tests used consist of : 1. Doppler ultrasound (for estimation of ankle brachial ratio). 2. photo-plethysmography 3. transcutaneous oximetry 4. laser-doppler flowmetry 5. television microscopy.

These noninvasive blood flow evaluations are inexpensive, reliable and reproducible.

Laser doppler flowmetry and television microscopy are two recent advances in the study of peripheral vessel and microcirculation in the diabetic foot[27,28]. But

the prohibitive cost and skin colour are limiting factors in using them in our country. In contradistinction, Doppler ultrasound and transcutaneous oximetry are simple methods of noninvasive evaluation of diabetic foot, which can be applied in the tropics. These two methods are described briefly.

Doppler Ultrasound and Determination of Ankle Brachial Pressure Index

This consists of two piezo-electric crystals mounted in a probe from which sound waves of various frequencies are emitted. The second crystal receives the sound waves reflected from moving particles in the blood vessel producing a voltage change. The latter can be amplified to an audible sound.

Acoustic gel is applied to the probe and it is held at an appropriate angle to the vessel being examined (posterior tibial artery). The pressure index is calculated as the ratio of ankle systolic to brachial systolic pressure. The ankle pressure is derived using a sphygmomanometer cuff (12 cm diameter) applied just above the ankle. The 10 mHz doppler probe is placed over the posterior tibial artery and the cuff is inflated. During deflation a return of acoustic flow signals indicates the level of ankle systolic pressure.

Transcutaneous Oximetry

Oxygen gas can diffuse through the body tissue and can be detected by an electrochemical oxygen sensor applied over the skin surface. The cathode and anode of the oxygen sensor are warmed to a temperature higher than the normal body surface temperature at which vasodilation of the cutaneous vessels takes place. Oxygen diffuses from the arterialised capillary bed to the skin surface and is measured by the Clark type sensor. This test was originally devised for neonatal monitoring. However presently this method is extensively used in assessing the peripheral vessel disease in diabetics and in exercise testing.

Transcutaneous oximetry is significantly lower in diabetics with peripheral vessel disease than in normals. Further it has been suggested that transcutaneous oximetry and regional perfusion index may become a useful prognosticator to indicate optimal amputation level. Holdich et al[29] have estimated transcutaneous oximetry during exercise and observed it to be a simple cheap and useful technique for assessing vascular claudication. A few reports have appeared describing the superiority of transcutaneous oximetry in non-invasive, vascular diagnosis in patients with diabetes[30].

MANAGEMENT OF DIABETIC FOOT

The management of diabetic foot is dependent on team work, comprising of chiropodist, diabetes specialist nurse and educator, diabetologist-physician, surgeon, the radiologist and the shoe fitter.[31] The gratifying role of a specialised diabetic foot clinic in improved survival of the diabetic foot has become

evident from the results of such a clinic at Diabetic Department in King's College Hospital, London[19]. Edmonds and Watkins have reported that over three years this specialised foot clinic and organised approach achieved a high rate of ulcer healing and reduced the number of major amputations. The essential areas of management are special shoes, intensive chiropody (the pivot of the clinic), precise antibiotic therapy, percutaneous angioplasty and appropriate surgery. Edmonds et al reported a healing of 86% in neuropathic ulcer and 72% in ischaemic ulcers[20]. The important aspects of management of diabetic foot are discussed in either classes separately.

Management of Neuropathic foot :- (A) Diabetic neuropathic ulcer :- The three cardinal principles of management are[5] : 1. appropriate and timely removal of callus 2. control of infection 3. reduction of weight bearing forces 4. adequate patient education

Removal of Callus :

Delbridge and Lequesne et al[32] have shown that the formation of callus is central to the development of neuropathic ulcer in a diabetic. The latter arises out of constant friction and pressure over the insensitive foot and ill-fitting foot wear. Charcot's deformity, rocker-bottom deformity clawed toes and hammer toes are the most vulnerable deformities prone to callus formation. The subject is asked to come for regular chiropody when the callus is removed.[33] Excess keratin is passed away so that the underlying lesion can be drained and ulcers allowed to re-epitheliaze.

Control of infection

After removing the callus a bacterial swab may be taken from the floor of the ulcer and appropriate antibiotic instituted. If there is gangrene in a digit, the "ray amputation" may be performed. In this the toe and a part of metatarsal are removed when the ulcer heals. A timely ray amputation may save the whole limb.

Distribution and reduction of the weight bearing force:

The most important aspect of promoting healing is to remove weight bearing force from the site of ulcer and ensure a redistribution of the shearing forces[34]. This can be achieved by the use of

1. special foots *2*. polyethylene foam insoles (Plastozole) 3. micro cell rubber insole (Tovey's insole)[35]. 4. "Cork cradle" shoe. 5. special windows cut out in the shoes to accommodate the deformed foot. 6. total contact plast cast with minimum padding[35]. These special moulded soles are usually accommodated in extra depth shoes.

Patient Education

The value of individual patient education and motivation far adequate self foot care cannot be over emphasised.

Management of diabetic neuraorthropathy (Charcot's joints) :

If an early diagnosis is made before gross disorganisation of the joint, immobilisation may help. Special insoles and shoes may be fitted to accommodate the deformity and prevent ulceration.

Management of painful diabetic neuropathic foot

Severely painful neuropathic foot in a diabetic may be a real management problem. At King's College, the protocal for management starts with pain killers and dilantin and carbamazepin, measures to diminish blood flow, and ganglion and neural blocking agents. By and large, the management is far from satisfactory. However, the hyperglycaemic painful neuropathy patients demonstrate significant subjective improvement from an insulin infusion pump and tight control of blood glucose.

Management of diabetic ischaemic foot

This can be described under :

1. medical management 2. surgical management 3. percutaneous transluminal angioplasty of stenosed and occluded lower extremity arteries.

Medical Management :- It is indicated when ulcer is small, recent and also in those patients unfit for reconstructive surgery. These ischaemic ulcers are painful, and pain relievers are often indicated. The rest of the medical management consists as in the neuropathic ulcer comprising of chiropody, antibiotics and special foot wear and a strong advise for giving up smoking.

Surgical management

This comprises of arterial reconstruction, sympathectomy and amputation.

Reconstructive surgery

Bypass surgery consists of grafting or bypassing the obstruction. Dacron grafts do not produce that good results in legs[37]. Indications for reconstructive surgery are intractable rest pain and claudication, nonhealing ulcers even after good medical management.

With an absent or weak femoral artery pulsation aortioiliac disease is suspected and a transfemormal or translumbar arteriography is performed. It can demonstrate the blood flow in iliac and femoral arteries. When the femoral pulse is good, the demonstration of superficial femoral, popliteal and distal tibial and peroneal arteries is better achieved by femoral arteriography.

The classical arteriographic finding is one of stenotic and occlusive lesions of the branches of popliteal artery. In the presence of a block in the femoral artery, a reversed saphenous vein graft is carried out. Even femoral to distal tibial or peroneal artery bypass is possible by using reversed saphenous vein graft. When saphenous vein graft is not available, modified umblilical veins or synthetic polytetrafluroethylene grafts are substituted.[37,38]

Lumbar Sympathectomy : For relief of ischaemic rest pain this has an important role to play. Cotton et al.[39] observed significant relief of pain even in the absence of any improvement in haemodynamic blood flow measurements.

Percutaneous transluminal angioplasty : This is a fast developing procedure using intraluminal balloons to dilate stenosis in the obstructed arteries. Most lower extremity vessel dilatations are presently done with a double lumen catheter. The inner lumen allows direct percuteneous insertion of the dilating catheter over a guide-wire. The balloon is inflated by injecting contrast agents through the outer lumen.[40]

This procedure is performed under local anaesthesia and has been used in the iliac, femoral and popliteal arterial stenoses. Angioplasty is particularly useful in high risk patients where surgery is contraindicated. The lesions most amenable for angioplasty include stenoses less 4 cm long and occlusions shorter than 10 cm. Iliac artery angioplasties have better success rate than the leg arteries (87%-100% patency after 2 years).[41] In the femoral artery, patency rates after angioplasty vary from 72 to 84% after 2 years. The popliteal artery patency after angioplasty is about 50% after 2 years.[5]

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