Limb Conservation in Severe Diabetic Foot Infection
- A New Technique
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ABSTRACT

Over the years, inspite of improved methods of controlling diabetic foot infections, the incidence of amputations is on the rise. In an attempt to salvage limbs with moderate to severe infections of the foot diabetics, a new surgical technique has been evolved which takes into account the anatomy of the layers of the foot and the mode of spread along facial planes. A detailed study of limbs amputated for uncontrolled diabetic foot infections was carried out by means of plain radiographs, angiography and dissection of these limbs. The factors leading to ischaemia and gangrene and the perpetuation of infection were delineated. Based on these findings, it was found that infections of the foot have a tendency to track along tissue planes to sites distant from the site of origin. It was therefore inferred that current conservative surgical techniques consisting mainly of localised drainage and debridement may not eradicate infection resulting in amputation being resorted to in a significant number of cases. A new technique has been developed by the author involving decompression of infected planes by incising the plantar fascia. This method has resulted in the salvage of apparently irretrievable limbs and the overall amputation rate in moderate to severe diabetic foot infections was as low as 5%

INTRODUCTION

The major predisposing factors for foot infections in diabetics are angiopathy and neuropathy. In addition to this, a number of social and environmental factors like barefoot walking, improper footwear (“Hawaii footwear”) wet floors in kitchens and bathrooms resulting in constant wetting of the foot contribute to foot infections. The anesthesia caused by the neuropathy results in the patients being oblivious to the ensuing damage to the foot from rodent bites (Fig. 1), ants, cockroaches, trauma from mechanical and thermal injury. Ascending and fulminant infection results in amputation of a significant number of these limbs. Amputation of these limbs are usually done as a life saving measure. A careful study of these amputated limbs has resulted in a good understanding of the pathogenesis of this condition and has helped to evolve a logical surgical technique to salvage these limbs. Even though some limbs may have to be sacrificed, a viable functional and stable foot with an occasional loss of one or two digits can be obtained in a vast majority of these cases.

MATERIALS AND METHODS

The study comprised of 512 patients attending private surgical clinics and the Voluntary Health Services Hospital from 1980 to 1994. The patients were from different socio-economic strata in the age group of 35-70 years. Sex distribution was equal. The predominant presenting features were-trophic ulcer, web space infection with evidence of deep infection in the form of fullness of the sole with or without redness and fluctuation, swollen ankle and ascending infection in the posterior compartment of leg. Some of these patients presented with the above mentioned features and/or gangrene and others reported with progression of infection or gangrene. In a significant number of patients earlier surgical intervention had met with disappointing results. These patients were promptly subjected to an operative decompression of the fascial compartments of the foot- a technique developed by the author.

The general condition, cardiac and diabetic status were assessed prior to the procedure which was performed under general anesthesia, intravenous ketamine or sedation using a combination of Diazepam and pentazocine given intravenously.

The incision (Fig.2) is made over the site of maximum fluctuation in the involved toe or web space and deepened to the second layer of sole cutting the planter fascia along the line of flexor tendons till either pus or oedema fluid is seen. Care
is taken to avoid injury to the plantar arteries in this area. The incision should stop at the medial border of foot and should not be extended up to the medial malleolus for fear of jeopardising the blood supply to the skin over the calcaneum, since the arterial supply comes from the perforator vessels through the medial aspect.

Having made an adequate incision in length and depth, pus is let out and dead tissue including the sloughed tendon and muscles are debrided. Doubtfully viable tissues are not sacrificed but taken care of during reviews. The wound is cleaned with diluted hydrogen peroxide and loosely packed with gauze impregnated with Povidone Iodine or Eusol. When there is evidence of infection around the ankle, then through a separate incision around the ankle, then through a separate incision behind the medial malleolus the flexor canal in which the posterior tibial artery is found is laid open, thus decompressing it, which contains the posterior tibial artery (Fig. 3).

If the patient presents with infection which has already extended to the leg, then the posterior compartment is laid open until Flexor Digitorum Longus muscle is exposed on which the subsoleal plexus of vein bathing in pus is seen.

If a toe disarticulation, the cause is traced to a retracted tendon through which pus has tracked to this layer (Figs. 4, 5, 6, 7).

Oedema of the dorsum of the foot is a frequent association in diabetic foot infections and does not necessarily indicate spread of infection to this region. Hence there is no indication for an incision over the dorsum. An adequate decompression in the sole invariably results in resolution of the dorsal oedema. In rare instances, we have found a dorsal abscess needing separate drainage.

The wound is inspected, either daily or on alternate days, depending on the soakage of dressing. Desloughing and debridement carried out, cleaned with diluted hydrogen peroxide and dressed with any one of the following – Povidone Iodine, Eusol, Metranidazole or any antiseptic powder. We do not advocate local insulin for dressing as insulin is inactivated in the presence of pus. Simultaneously, appropriate antibiotic coverage along with metranidazole is provided. Severe infections may need prolonged antibiotic therapy each drug being
exhibited for about 10-14 days depending upon the clinical response and antibiotic sensitivity. Good diabetic management is essential with plain insulin given in divided doses. Pentoxyl, 800 – 1200 mgm in divided doses daily also is prescribed for patients in whom there is diminished arterial flow.

Once the infection was fully controlled, secondary suturing hastened early recovery in a number of cases. Patients who had significant loss of skin and in whom the wound was granulating well; split skin grafting was resorted to successfully (Fig. 6).

Initially, the procedure was done in non-atherosclerotic limbs with satisfying results and the benefit was later extended to atherosclerotic limbs also, though the percentage of feet saved was relatively less in the latter group.

RESULTS

In this study only 5% limbs had to be amputated and this was done only when a fair trial of conservative management as mentioned above, failed to improve the limbs. These patients either had very poor vascularity of the foot or presented in a moribund condition, or reported after being inadequately managed elsewhere leading to progressive ascending infection with toxemia.

DISCUSSION

A clear understanding of the anatomy of the layers of the foot helps in the management of foot infections (Fig. 8).

The II Layer of the sole of foot containing the flexor tendons and vessels is enclosed inferiorly by the plantar fascia and superiorly by the metatarsals, small muscles and ligaments of the foot. The fascial layer continues distally into the toes through the tunnel of the medial flexor retinaculum into the subsoleal space along the flexor tendons. The tunnel also contains the neurovascular bundle. The arterial arch (plantar arch) is formed at the level of the heads of the metatarsals by the medial and lateral plantar arteries. The digital vessels which arise from the arch pass via the tunnel of the intermetatarsal ligament to the toes.

A brief description of the pathophysiology of diabetic foot infection is important to understand the principles of the procedure of decompression.

The basic problem in diabetic foot is due to the neuropathy and vasculopathy caused by hyperglycemia. Neuropathy, in its late stage, causes near total anesthesia of the foot and its associated problems; likewise, the vascular changes are those of arteriosclerosis, which occur at an earlier age in
diabetics, causing not only changes in the intima but also calcifications in peripheral tissue and local gigantism. The combination of neuropathy, macro and micro angiopathy leads to gangrene of digit or foot. Minor problems like fissure, ulceration, fungal and bacterial infections occur easily in diabetic foot. If timely appropriate treatment is not instituted, the infection in the toe can spread along the tunnel in which the long flexor tendons pass, or along the lumbrical canal to the flexor tendon from the web space. This pus further tracks down to the layer between the plantar fascia, interossei and bone. Pus starts accumulating in this layer which we have called the mid compartment of sole. The boundaries of this compartment are – plantar aponeurosis superficially and metatarsals and interosseous muscle deeper. The plantar aponeurosis slips are inserted to each of five toes, and on either side, the plantar aponeurosis sends a septum from each of its edges to the first to fifth metatarsal bone and proximally upto the calcaneum. Accumulation of pus or oedema in this compartment clinically manifests with either swelling, prominence up to the medial arch of foot or with pain, tenderness, redness, and dorsal oedema due to lymphangitis. This mid compartment contains the long flexor tendons and neurovascular bundle. If the pus or oedema is not let out completely, there is an increase in the mid compartment pressure with accumulation of more pus or oedema fluid in the unyielding mid compartment leading to compression of the arteries or toxic thrombosis of artery. This leads to progressive gangrene of the tissue supplied by these vessels. Left unattended, the infection tracks up into the leg along the sheaths of Flexor Digitorum Longus, Tibialis Posterior and Flexor Hallucis Longus, which is strapped by the flexor retinaculum and medial ligament of the ankle and below by the bony tunnel which contains, in addition to the tendons, the neurovascular bundle coming to the sole of foot. Being a pre-existing potential space in the posterior compartment of leg below soleus muscle, pus tracks along from the sole, goes on to bathe the subsoleal plexus of veins, oedema and pus compress the venous plexus leading to ankle and foot oedema or embolism due to venous thrombosis and toxemia due to absorption of toxic material from the accumulated pus (Figs. 9, 10, 11).

The surgical interventions done for treatment of diabetic foot infections so far has only been to let out the pus when the infection has progressed. The various procedures adopted included disarticulation of digits, ray amputation and midfoot amputation. Yet none of these gave the desired results, because of the faulty and inadequate drainage of pus. In the management of toe and web space infection, it is usual to incise deep or disarticulate the toe. Once this is done, the cut end of flexor tendons hitherto attached to the disarticulated bone, retract into the sole of foot carrying the infection with it to the deeper enclose layers – mid compartment of sole. The person after having undergone the procedure normally lie flat on his back with the toes pointing upwards, allowing pus to track along the flexor...
tendon canal to the mid compartment of sole and accumulate in this closed space. For the pus to drain, the patient has to lie face down with toes pointing downwards. It is a common observation that the adjoining toes becoming gangrenous toe. This is prevented if the digital vessels are also decompressed up to the heads of metatarsal as described latter and not just do a disarticulation of toe or web space incision for infections of toe or web space. Web space or digital infection spread to base of toe causing gangrene of toe due to involvement of digital vessels. Spread of infection to mid compartment causes gangrene of more than one toe due to thrombosis of vessels taking off from the plantar arch, spread to ankle or flexor retinaculum level involves the main plantar artery causing gangrene of sole.

Diabetic foot infections are difficult to treat because of the triad of hyperglycaemia, neuropathy and angiopathy. To this is added a fourth factor of compression and/or thrombosis of the plantar vessels due to the anatomical position of the vessels in the fascial compartments of sole, ankle and leg. This aspect has been brought out by the study of limbs of severe foot infections, which necessitated amputations. The factors causing ascending infection and gangrene at various levels have been brought out and explained in the study. By evolving the decompression procedure of the mid-compartment of sole, ankle and posterior compartment of leg, the author has amply demonstrated the usefulness of the procedure in preventing amputations in about 95% of cases treated. Hence, this study proves the importance of the fascial compartment with trapped infection and their decompression in the treatment of severe diabetic foot infection, as the prime modality of treatment in addition to good diabetic control.

REFERENCES


