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Diyabetik Ayakta Anatomik Değişiklikler

Anatomical Changes in Diabetic foot

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ÖZ

Amaç: Diyabetik hastalarda amputasyon gerektiren gangren gelişme olasılığı diğerlerine göre 15 kat fazladır. Travmaya bağlı olmayan uzuv amputasyonlarının çoğu diyabet komplikasyonlarına bağlıdır. Bu çalışmanın amacı, diyabetik ayak ülseri olan hastalarda ortaya çıkan çeşitli anatomik değişikliklerin değerlendirilmesidir.

Materyal ve Metot: BHDC Hindistan Genel Cerrahi Bölümü Başkanı ve profesörü gözetiminde Delhi' de bulunan bir 3. Basamak hastanenin diyabet kliniğine başvuru yapan 70 hasta üzerinde ayak lezyonlarının bulunduğu bölge, büyüklüğü, genel özellikleri ve etiyolojisi ile yara yeri sürüntülerinin kültür ve duyarlılık özellikleri konusunda bir prospektif çalışma yürütüldü.

Bulgular: En sık bulgular arasında deri ve tırnak değişiklikleri (47 hasta, %67,1) yer almaktaydı ve nasırlar, sertlikler, kuruluklar, fissürler ve hipertrofiler şeklinde görülmekteydi. Yirmi hastada (%28,5 ülserler) ve üç hastada gangren (%4,2) saptandı. Elli altı hastada (%80) birden fazla yara yeri debridmanı ve pansuman, 11 hastada (%15,7) yara iyileşmesinin temini için deri greftlemesi ve 3 hastada (%4,2) amputasyon uygulanması gerekti.

Sonuç: Diyabetin uygun şekilde control edilmesi, günlük yara bakımı, yeterli ve zamanında debridman şeklinde cerrahi müdahaleler ve kültüre duyarlı antibiyotiklerin kullanılması ile diyabetik ayak yaraları düzgün şekilde iyileştirebilmektedir. Ciddi enfeksiyonların varlığında septiseminin önlenmesi amacıyla farklı derecelerde amputasyon uygulamaları hayat kurtarıcı müdahaleler olarak düşünülmeli ve uzvun yada parmağın kurtarılmasındansa hayatın kurtarılması öncelik olmalıdır.

Anahtar kelimeler: Amputasyon, ayak lezyonları, nöropati, yara debridmanı.

ABSTRACT

Objective: Diabetic patients are 15 times more likely to develop gangrene, requiring amputation. Most of the non-traumatic limb amputations are due to complications of diabetes. The aim of this study was to analyse the various anatomical changes occurring in patients with diabetic foot ulcers.

Materials and Methods: A prospective study was carried out in 70 patients presenting to the Diabetic Clinic at a tertiary care hospital in Delhi, India under the Professor and Head of Department Surgery BHDC India, regarding the site, size, nature of foot lesion, etiology of foot lesions and culture and sensitivity patterns of the wound swabs.

Results: Most common lesions were noticed to have skin and nail changes (47 patients,67.1%) in the form of corns, callosities, dry skin, fissures, hypertrophied and brittle nails.20 patients (28.5%) presented with ulcers and 3 patients (4.2%) had gangrene. 56 patients (80%) were managed by multiple wound debridements and serial dressings.11 patients (15.7%) required skin grafting for wound healing.3 patients (4.2%) required some form of amputation.

Conclusion: It was found that with strict diabetic control, daily dressings, surgical intervention in the form of adequate, aggressive and timely debridement and culture specific antibiotics, the diabetic foot wounds healed well. Amputation at appropriate levels should be performed as life-saving measures in severe infections to prevent septicaemia and lifesaving takes precedence over limb / toe saving.

Keywords: Amputation, foot lesions, neuropathy, wound debridement

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INTRODUCTION

Amongst the patients with diabetes, about 15% will develop foot lesions during their lifetime requiring hospitalisation for an average of six weeks.[1,2] The foot lesions in diabetic patients is due to the consequences of peripheral vascular disease and peripheral neuropathy leading to non-healing diabetic foot ulcers.This results in tissue ischaemia and / or altered proprioception, touch or pain sensation, and atrophy of skeletal muscles in the foot. These render the foot less resistant to trauma and infection, requiring frequent hospital admissions.[3]Five out of six non -traumatic limb amputations are due to complications of diabetes.[4]Non-healing wounds are more than just a cost burden, as they have been shown to cause loss of mobility and ability to perform daily tasks, loss of participation in the workforce, and poor quality of life.[5]Approximately, 4% of the US population is diagnosed to have Diabetes, 15% of whom are hospitalized yearly for an average of six weeks with various complications of diabetic foot.[1-3,6]Such problems force a high budgetary and social expenses because of the extended course of the disease or after surgeries running from wound debridement, emergency amputations and wound coverage.[7] The wound healing is slow due to diabetic angiopathy.[8,9]

This study is designed to analyse the various anatomical changes occurring in diabetic foot patients, observe the nature of progression of the lesions, assess the result of the various interventions offered and predict foot at risk of impending complications.

MATERIALS AND METHODS

A prospective study was carried out in 70 patients presenting to the Diabetic Clinic at a tertiary care hospital in Delhi, India. The patients included in the study consisted of in-patients and those presenting in out-patient department for follow-up over a period of two years.

***Ethical Approval**:* Approval for the study was granted by BHDC Ethics Committee (Professor and Head of Department Surgery BHDC), and permission to carry out the study was obtained from him under his guidance and supervision (Permission 04 Apr 2005).

Regular follow-up of the patients during the course of stay in the hospital and on Out-patient basis was carried out. Various interventions offered were strict and adequate glycaemic control, daily dressings, adequate, aggressive and timely serial debridements and culture specific antibiotic therapy. Amputations were carried out at appropriate levels in cases of severe infection to prevent systemic spread of infection.

The wide spectrum of foot lesions were tabulated, anatomical changes occurring in the Indian population compared and conclusion was drawn.

RESULT

Diabetic foot is primarily affected by peripheral neuropathy and angiopathy. The foot is typically insensate and has lost pain which is a protective mechanism as trauma to such feet goes unnoticed. Diabetic autonomic neuropathy makes the foot dry and fissured.

Out of the 70 patients, 2.8% were below 40 years of age, 9 patients (12.8%) were in the group between 40-50.41 patients (58.5%) belonged to the 50-60 age group, 16 patients (22.8%) were in the 60-70 age group and only 2 patients were in the age group above 70 years. Out of 70 patients, 51 patients (72.86%) were males and 19 patients (27.14%) were females who presented with various diabetic foot lesions (Figure 1). Out of the 70 patients, 24 (34.2%) presented with history of Trauma, 19 patients were detected to have peripheral vascular disease and 36 patients (51.4%) suffered from neuropathy. Statistical significant changes occur thus $P \leq 0.05$.

Among the 34 patients with diabetic foot ulcer, 14 patients (41.4%) had ulcers involving the plantar surface under the first metatarsal head, 11 patients (32.3%) had ulcer over the toes, 6 patients (17.6%) has ulcer over the dorsum and 3 patients (11.7%) had ulcers over the heels. 22 patients had discharging wounds with infection, nine of which had osteomyelitis. Statistical significant increases in changes occur thus $P \leq 0.05$.

47 patients (67.1%lesions) were noticed to have skin and nail changes in the form of corns, callosities, dry skin, fissures, hypertrophied and brittle nails.20 patients (28.5%) presented with ulcers and 3 patients (4.2%) had gangrene. Statistical significant increases in changes occur thus $P \leq 0.05$.

As seen in Figure 2, the commonest pathogen was *Staphylococcus* (60.1%) followed by *E. Coli* (27.1%), *Bacteroids* (9.09%) and *Pseudomonas* (3.7%).56 patients (80%) were managed by multiple wound debridements and serial dressings. 11 patients (15.7%) required skin grafting for wound healing as depicted in Figure3. Three patients (4.2%) required some form of amputation.

DISCUSSION AND CONCLUSION

Diabetes Mellitus is one of the oldest diseases known to man.[10] The sweet honey-like taste of urine in polyuric patients which attracted ants was reported during the 5th and 6th century AD by Sushruta.[11,12]

Diabetics are 25 times more likely to develop blindness, 17 times more likely to develop gangrene and twice as likely to develop heart disease and neuropathy. The concurrent neurologic and vascular involvement was noticed by Levin in 1983, making it prone to the various anatomic and morphological changes that is attributed to diabetic foot. The foot lesions in diabetics may be caused by either the underlying neuropathy or peripheral vascular disease or a combination of both.[3,4] Each of them manifests differently and early recognition helps in the institution of correct intervention.

Most significant changes in the foot occur in the presence of neuropathies.[4] Examination of these feet demonstrate the alteration of sensory impulses transmitted proximally as evidenced by reduced vibratory, pinprick and light touch sensation. This sensory deficit is accompanied by motor involvement characterized by muscular weakness and atrophy. This leads to imbalance between the flexors and extensors of the toes resulting in a deformed foot, altering the biomechanics of the foot. The common foot lesion are[3-5]blisters, corns and calluses, dry skin, fissures, hair loss, brittle deformed nails, toe deformities in the form of hammer toes, hallux valgus, tailor bunion, clawing of toes, infections presenting as paronychia, abscesses, cellulitis, interdigital fungal infections or ulcers.

Foot deformities may take several forms such as clawing of toes, varus deformity, hammer toe or hallux valgus. These changes lead to pressure distribution disturbance, increased shear stress and friction, ultimately leading in foot ulceration.[13]Autonomic neuropathy is associated with foot oedema resistant to diuretics which results due to hyperkinetic circulation, further predisposing the foot to ulceration.[14]

Semmes-Weinstein monofilament is a widely used tool for the assessment of the diabetic peripheral neuropathy.[15] It assesses the protective ability of the foot through the application of gentle pressure to the handle until the nylon filament is buckled for two seconds. 90% of patients with insensate diabetic foot can be detected on testing four planter sites of the foot viz. the great toe and the bases of first, third and fifth metatarsals.[16]Impaired perception of vibration sensation is one of the earliest signs of diabetic peripheral neuropathy which can be assessed by 128 Hz tuning fork at the medial malleolus or the tibial tuberosity. Tuning fork assessment carries about 53% sensitivity as compared to monofilament test which has shown a sensitivity in the range of 66%-91% proving that tuning fork is less predictive in comparison to Semmes- Weinstein monofilament for development of foot ulceration.[17] Nerve conduction studies are rarely used in diagnosis of diabetic neuropathy but are non-invasive and reliable and can detect the type of nerve injury, extent, symmetry and severity of the lesion.[18]

Friction is the resistance that anybody meets in moving over another body. The more irregular surfaces in contact, the greater is the friction, and greater is the chance of friction induced tissue breakdown.[5]Constant and rapid friction against the skin forms blisters. Intermittent and slow friction causes calluses and corns.[3,5]

"Pressure" is the force exerted by one body on another by its weight. It is inversely proportional to the "area" the body rests on. Unremitting direct pressure against tissue causes ischemia and tissue necrosis resulting in ulcers.

During the dynamic action of walking both the above forces combine to form the "shear force". In the gait cycle, at heel contact there is a direct downward force of the body through the substance of the heel and an upward, a rapid supination movement occurs at the foot as the body moves over the foot. The interface between the foot and the ground is the deep soft tissue and the skin. Smooth, efficient locomotion, is attributed to appropriate skeletal and connective tissue alignment along with phasic skeletal muscle action.[19]

This optimal function cannot be maintained in the presence of Diabetic angiopathy and neuropathy. Lack of protective pain caused by sensory neuropathy allows damage to go unheeded. There is loss of vibratory and touch sensation, hence the patient may not appreciate trauma to the foot for instance from ill-fitting new shoes, a hot water bottle or excessive friction.[19] There is loss of vibratory and touch sensation, hence the patient may not appreciate trauma to the foot for instance from ill-fitting new shoes, a hot water bottle or excessive friction.[19]

Patients may either have a painful neuropathy or a painless one as a result of neuropathy affecting the small non-myelinatedC-fibres.[20] Painful neuropathy results in a persistent burning, aching or tingling feel in the feet, and worst in the bed at night. The risk of

ulceration in painless neuropathy is greater.[20] Failure of perfusion by the circulatory system impedes healing, resulting in the formation of ulcers.[21,22] Motor neuropathy can be seen as atrophy of small foot muscles resulting in claw toes. Loss of Achilles tendon reflex is an early sign of motor neuropathy.[23] In our study, 47 patients (67.1% lesions) were noticed to have skin and nail changes in the form of corns, callosities, dry skin, fissures, hypertrophied and brittle nails. Diabetic autonomic neuropathy may result in sudomotor dysfunction leading to abnormal sweating and dry skin with cracking and fissuring facilitating the bacterial infection of the foot.[24] Diabetic neuropathy usually presents as distal, bilateral, symmetrical neuropathy usually starting in the lower limbs but may involve the upper limb too and gradually proceeding proximally as the severity of nerve dysfunction increases and presents in a glove and stocking pattern of abnormal sensation.[25] Pryce[26] recognized about 100 years ago, that diabetic neuropathy led to painless ulceration. Martin[27] and Ellenburg[28] noticed that neuropathic feet often had palpable pulses. Armstrong and Colleagues, have cautioned, that the ulcer severity was more important than the ulcer site in determining the final outcome.

In conclusion; It was found that with strict diabetic control, daily dressings, surgical intervention in the form of adequate, aggressive and timely debridement and culture specific antibiotics, the diabetic foot wounds healed well. Amputation at appropriate levels should be performed as life saving measures in severe infections to prevent septicaemia and life-saving takes precedence over limb / toe saving. Normal saline dressings have been found to be better as compared to other cleaning agents including povidone iodine once healthy granulation tissue appears in diabetic foot lesions.

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Conflict of Interest: No conflict of interest was declared by the authors.

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REFERENCES

1. Reiber GE, Lipsky BA, Gibbons GW. The burden of diabetic foot ulcers. Am J Surg. 1998;176(2A Suppl):5S-10S.
2. Sanders LJ. Diabetes mellitus:Prevention of amputation. J Am Podiatr Med Assoc. 1994;84(7): 322-328.
3. Quebedeaux TL, Lavery LA, Lavery DC. The development of foot deformities and ulcers after great toe amputation in diabetes. Diabetes Care. 1996;19(2):165-167.
4. Singh G, Chawla S. Amputation in Diabetic Patients. Med J Armed Forces India. 2011;62(1):36–39.
5. Edwards et al.: Health service pathways for patients with chronic leg ulcers: identifying effective pathways for facilitation of evidence based wound care. BMC Health Services Research. 2013;13:86. doi:10.1186/1472-6963-13-86
6. Trautner C, Haastert B, Giani G, Berger M. Incidence of lower limb amputations and diabetes. Diabetes Care. 1996;19(9):1006-9.
7. Naves CC. The Diabetic Foot: A Historical Overview and Gaps in Current Treatment. AdvWound Care (New Rochelle). 2016;5(5):191–197. doi:10.1089/wound.2013.0518
8. Apelqvist J, Ragnarson-Tennvall G, Persson U, Larsson J. Diabetic foot ulcers in a multidisciplinary setting. An economic analysis of primary healing and healing with amputation. J Intern Med. 1994 ;235(5):463-71.
9. Giugliano D, Ceriello A, Paolisso G. Oxidative stress and diabetic vascular complications. Diabetes Care. 1996;19(3):257-67.
10. Olokoba AB, Obateru OA, Olokoba LB. Type 2 diabetes mellitus: A review of current trends. Oman Med J. 2012;27(4):269–273. doi: 10.5001/omj.2012.68.
11. Karamanou M, Protogerou A, Tsoucalas G, Androutsos G, Poulakou-Rebelakou E. Milestones in the history of diabetes mellitus: The main contributors. World J Diabetes. 2016;7(1):1–7. doi:10.4239/wjd.v7.i1.1
12. Vecchio I, Tornali C, Bragazzi NL and Martini M. The Discovery of Insulin: An Important Milestone in the History of Medicine. Front. Endocrinol. 2018; 9:613. doi: 10.3389/fendo.2018.00613
13. Amin N, Doupis J. Diabetic foot disease: From the evaluation of the "foot at risk" to the novel diabetic ulcer treatment modalities. World J Diabetes. 2016;7(7):153–164. doi:10.4239/wjd.v7.i7.153
14. Nabuurs-Franssen MH, Houben AJ,Tooke JE et al..The effect of polyneuropathy on foot microcirculation in Type II diabetes. Diabetologia. 2002;45:1164–1171. doi:10.1007/s00125-002-0872-z

15. Mayfield JA, Sugarman JR. The use of the Semmes-Weinstein monofilament and other threshold tests for preventing foot ulceration and amputation in persons with diabetes. J FamPract. 2000;49(11 Suppl):S17-29.
16. Smieja M, Hunt DL, Edelman D, Etchells E, Cornuz J, Simel DL. Clinical examination for the detection of protective sensation in the feet of diabetic patients. International Cooperative Group for Clinical Examination Research. J Gen Intern Med. 1999;14(7):418-24.
17. Boyko EJ, Ahroni JH, Stensel V et al. A prospective study of risk factors for diabetic foot ulcer. The Seattle Diabetic Foot Study. Diabetes Care. Jul 1999;22(7):1036-42.
18. Gooch C, Podwall D. The diabetic neuropathies. Neurologist. 2004;10(6):311-22.
19. Boulton AJ, Kubrusly DB, Bowker JH et al. Impaired vibratory perception and diabetic foot ulceration.Diabet Med. 1986;3(4):335-7.
20. Armstrong DG, Lavery LA. Diabetic foot ulcers: prevention, diagnosis andclassification. Am Fam Physician. 1998;57(6):1325-32.
21. Brand PW. Management of the insensitive limb. Phys Ther. 1979;59(1):8-12.
22. Guy RJ, Clark CA, Malcolm PN, Watkins PJ. Evaluation of thermal and vibration sensation in diabetic neuropathy. Diabetologia. 1985;28(3):131-7.
23. Andersen H. Motor dysfunction in diabetes. Diabetes Metab Res Rev. 2012 ;28Suppl 1:89-92.
24. Shaw JE, Boulton AJ. The pathogenesis of diabetic foot problems: an overview. Diabetes. 1997 ;46Suppl 2:S58-61.
25. Chung T, Prasad K, Lloyd TE. Peripheral neuropathy: clinical and electrophysiological considerations. Neuroimaging Clin N Am. 2014;24 (1):49–65. doi:10.1016/j.nic.2013.03.023.
26. Pryce TD. A case of perforating ulcers of both feet associated with diabetes and ataxic symptoms. Lancet . 1887:11-12.
27. Martin MM. Neuropathic lesions of the feet in diabetes mellitus. Proc R Soc Med. 1954;47 (2):139-40.
28. Ellenberg M. Diabeicneuropathic ulcer. J Mt Sinai Hosp. 1968;35:585-594.

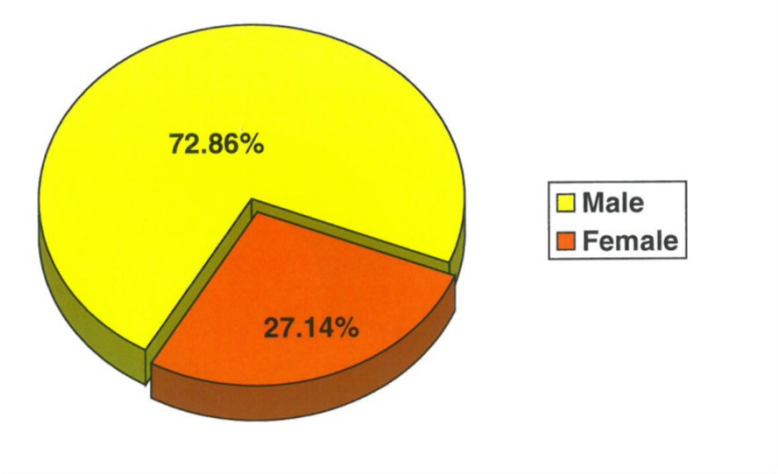

Figure 1. Percentage of gender affected by diabetic foot lesions.

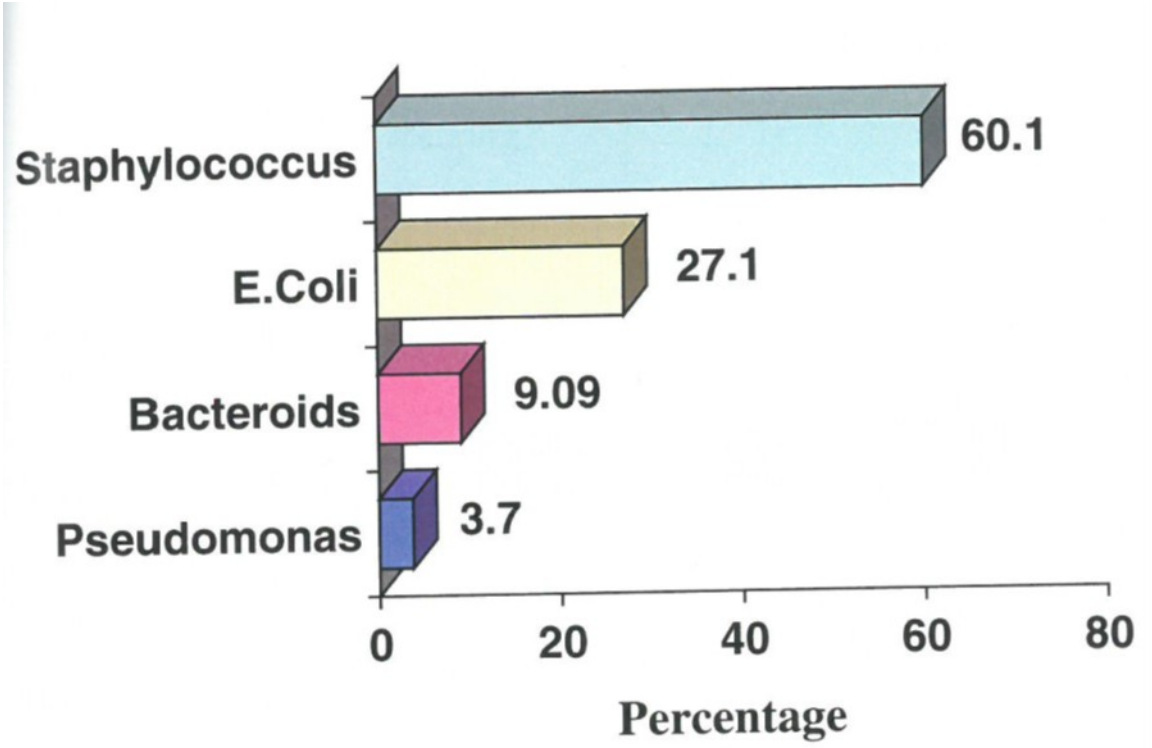

Figure 2. Wound swab culture results: Micro-organisms grown from wound swabs of diabetic foot lesions.

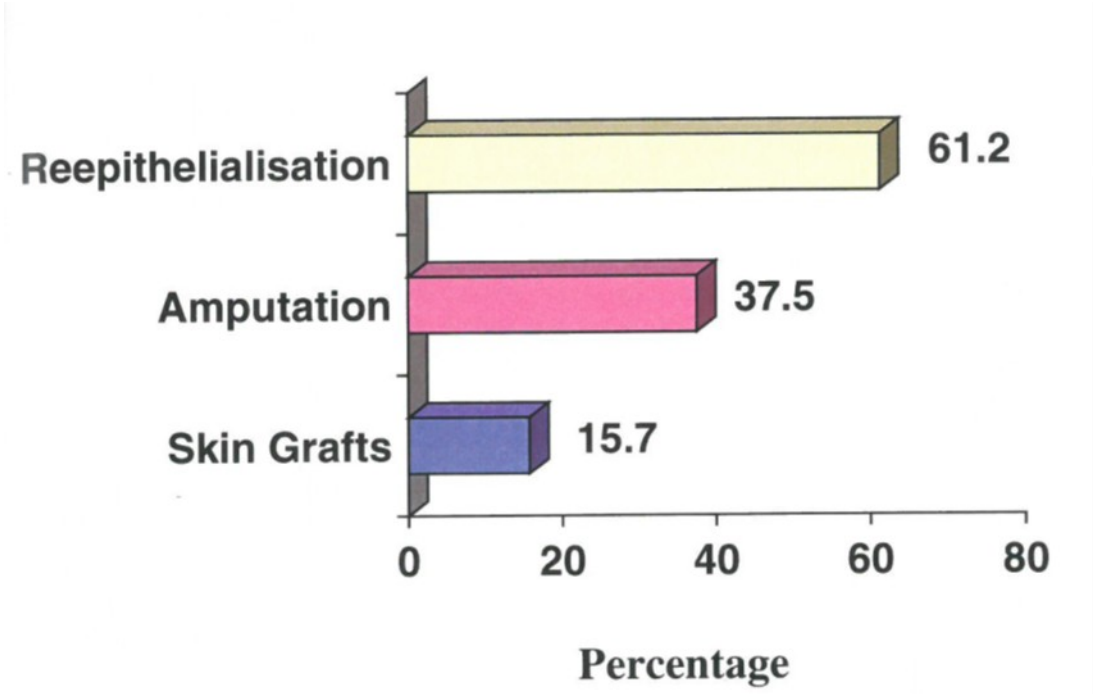

Figure 3. Fate of diabetic foot lesions after treatment.