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Application of Debridement in Diabetic Foot Ulcer

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Abstract

Most assuredly diabetic foot ulcer can influence to infection, gangrene, amputation, and even death if necessary care is not needed. On the other hand, once diabetic foot ulcer has advanced, there is an escalated peril of ulcer progression that perhaps finally influence to amputation. Overall, the rate of lower limb amputation in patients with diabetes mellitus is fifteen fold higher than patients without diabetes mellitus. Debridement appears to de-escalate bacterial counts and enliven generation of local growth factors. This method also decreases pressure, evaluates the wound bed, and facilitates wound drainage. Debridement is the takeoff of necrotic and senescent tissues as well as foreign and exposed materials from a wound, which is considered as the primary and the consummate significant therapeutic step influencing to wound closure and de-escalate in the possibility of limb amputation in patients with diabetic foot ulcer. Enzymatic debridement is a method of debriding devitalized tissue by topical enzymes such as collagenase, fibrinolysin, or papain. The intention of this review article is to encapsulate application of debridement in diabetic foot ulcer advanced from diabetes mellitus as complications and also articulate its advantages for diabetic foot ulcer complication management.

Keywords: Application; Debridement; Diabetic Foot Ulcer

Abbreviations: DMs: Diabetes Mellitus; DFU: Diabetic Foot Ulcer

Introduction

Diabetic foot is still the consummate often rationale of hospitalization of patients with DMs, and diabetes mellitus (DMs) is the chief antecedent of greater than half of non-traumatic lower limb amputations. Diabetic foot ulcer (DFU) is thought as a preponderance source of morbidity and an influencing antecedent of hospitalization in patients with DMs. It is approximated that estimate twenty percent of hospital admissions amid patients with DM are the sequence of DFU. Most assuredly DFU can influence to infection, gangrene, amputation, and even death if necessary care is not needed. On the other hand, once DFU has advanced, there is an escalated peril of ulcer progression that perhaps finally influence to amputation. Overall, the rate of lower limb amputation in patients with diabetes mellitus is fifteen fold higher than patients without DMs [1-7]. Foot ulcers are one of the chief complications in DMs and fifteen percent of diabetic patients advance foot ulcer and fifteen to twenty percent of these will necessitate amputation [8,9]. DFUs are an outcome of multiple factors involving loss of protective sensation owing to peripheral neuropathy where the feet become numb and the damage goes unnoticed. Also, arterial inadequacy complicates the neuropathic ulcer which influences to meager wound curing. Foot abnormality and calluses can sequence in great plantar pressure, which sequences additional peril. Mechanical stress at the wound site is hypothesized to influence wound curing. Multiple distinctive factors contribute to the peril of foot ulceration and its subsequent infection in patients with

DMs. Uncontrolled hyperglycemia, duration of DMs, trauma, inappropriate footwear, callus, history of previous ulcers/amputations, older age, blindness/injured vision, chronic renal disease and meager nutrition have also been observed to play a function in the pathogenesis and progression of DFU [10,11]. The medical management of DFU remains a problem. A better comprehending of the pathophysiology and molecular biology of diabetic wounds perhaps support to result ameliorated and further efficient solutions for their management. It is recently accepted that DFU therapies should be directed to actively promoting wound curing by correcting the expression of those biological factors which are significant in the curing procedure [12].

Debridement

The method of debridement perhaps the significant management for diabetic foot ulcer. Several types of products have been used to keep the wound dry and covered (hydrogels, hydrocolloids, alginates and foams). Debridement includes takeoff of dead, injured, or exposed tissue, which ameliorates the healing potential of the remaining healthy tissues. Based on the wound tissue type, distinctive debridement techniques are recommended: (1) Surgical debridement or sharp debridement-recommended for necrotic and exposed wounds. The terms surgical debridement and sharp debridement are frequently used intercalate, certain clinicians refer to surgical debridement as being settled in an operating room, whereas sharp debridement is settled in a clinic setting. Sharp surgical debridement is the mostly effective and quickest method of debridement; (2) Autolytic debridement-a selective process in which the necrotic tissue is liquefied. A wound covered with an occlusive dressing permits concentration of tissue fluids containing macrophages, neutrophils, and enzymes, which takeoff bacteria and digest necrotic tissues. Autolytic debridement is not advisable for the management of exposed pressure ulcers; (3) Mechanical debridement-includes takeoff of unhealthy tissue using a dressing, which is altered regularly by wound irrigation (pressure: 4-15 psi), without injuring healthy/new tissues. Scrubbing the wound aids in takeoff of exudates and devitalized tissues, although this influences to bleeding as well as pain sequencing from wound trauma. This technique is used in the treatment of surgical wounds and venous leg ulcers. The shortcomings of the method is that it is time consuming and expensive; (4) Enzymatic debridement-a method of debriding devitalized tissue by topical enzymes such as collagenase, fibrinolysin, or papain. Recommended for sloughy, exposed, necrotic wounds where surgical debridement is contraindicated; and (5) Maggot debridement-a technique in which maggots or fly larva that are accelerated in a sterile environment are used [13-18]. Debridement is the takeoff of necrotic and senescent tissues as well as foreign and exposed materials from a wound, which is thought-out as the primary and the consummate significant therapeutic step influencing to wound closure and de-escalate in the possibility of limb amputation in patients with DFU. Debridement appears to de-escalate bacterial counts and enliven generation of local growth factors. This method also decreases pressure, evaluates the wound bed, and facilitates wound drainage. There are distinctive kinds of debridement involving surgical, enzymatic, autolytic, mechanical, and biological. Amid these methods, surgical debridement has been revealed to be most effective in DFU curing. Surgical or sharp debridement includes cutting away dead and exposed tissues pursued by daily application of saline moistened cotton gauze. The chief objective of this type of debridement is to turn a chronic ulcer into an acute one. Surgical debridement should be repeated frequently as necessitated if fresh necrotic tissue continues to form. The method of debridement based on characteristics, preferences, and practitioner degree of expertise. When surgical or sharp debridement is not expressed, then distinctive types of debridement could be used [19-27]. The peril of the lesion worsening in terms of both progressive deep tissue decrement and infection is linked to the co-existence of an ischemic component. Thereupon, peripheral vascular disease must be precluded in the primary assessment of an ulcerated lesion with clinical characteristics appropriate to those of a neuropathic lesion. Appropriate debridement must pursue the evaluation of an ulcer. It should completely takeoff the callus that surrounds the lesion and entire non-healthy tissues, until healthy bleeding edges are revealed. Sharp debridement permits for thorough takeoff of entire necrotic material and destructs the bacterial load, thus promoting curing. It is then mandatory to go on an accurate “probe to bone” maneuvre in order to settle the involvement of deeper structures such as tendons, joint capsules and bones. In the preponderance of cases, the ‘probe-to-bone’ maneuvre with a sterile blunt instrument is sufficient to diagnose osteomyelitis. It is thereupon solely necessary to use multiple complex methods (such as Nuclear Magnetic Resonance and/or radiolabeled leukocyte scanning) in a small percentage of cases [28].

Conclusion

DFU is considered as a majority source of morbidity and an influencing antecedent of hospitalization in patients with DMs. Foot ulcers are one of the chief complications in DMs and fifteen percent of diabetic patients advance foot ulcer and fifteen to twenty percent of these will necessitate amputation. Debridement is the takeoff of necrotic and senescent tissues as well as foreign and exposed materials from a wound, which is thought-out as the primary and the consummate significant therapeutic step influencing to wound closure and de-escalate in the possibility of limb amputation in patients with DFU. Autolytic debridement is a selective process in

which the necrotic tissue is liquefied. A wound covered with an occlusive dressing permits concentration of tissue fluids containing macrophages, neutrophils, and enzymes, which takeoff bacteria and digest necrotic tissues.

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References

1. Yazdanpanah L, Nasiri M, Adarvishi S (2015) Literature review on the management of diabetic foot ulcer. World J Diabetes 6(1): 37-53.
2. Shahbazian H, Yazdanpanah L, Latifi SM (2013) Risk assessment of patients with diabetes for foot ulcers according to risk classification consensus of International Working Group on Diabetic Foot (IWGDF). Pak J Med Sci 29(3): 730-734.
3. Aalaa M, Malazy OT, Sanjari M, Peimani M, Tehrani M (2012) Nurses' role in diabetic foot prevention and care; a review. J Diabetes Metab Disord 11(1): 24.
4. Leone S, Pascale R, Vitale M, Esposito S (2012) Epidemiology of diabetic foot. Infez Med 20(S1): 8-13.
5. Iraj B, Khorvash F, Ebneshahidi A, Askari G (2013) Prevention of diabetic foot ulcer. Int J Prev Med 4(3): 373-376.
6. Fard AS, Esmaelzadeh M, Larijani B (2007) Assessment and treatment of diabetic foot ulcer. Int J Clin Pract 61(11): 1931-1938.
7. Snyder RJ, Hanft JR (2009) Diabetic foot ulcers--effects on QOL, costs, and mortality and the role of standard wound care and advanced-care therapies. Ostomy Wound Manage 55(1): 28-38.
8. Larijani B, Shirin HR (2008) Overview of diabetic foot; novel treatments in diabetic foot ulcer. Daru 16 (1): 1-6.
9. Larijani B, Forouzandeh F (2003) Diabetic foot disorders. Iran J Diabetes and Lipid Disord 2(2): 103-93.
10. Kavitha KV, Tiwari S, Purandare VB, Khedkar S, Bhosale SS, et al. (2014) Choice of wound care in diabetic foot ulcer: A practical approach. World J Diabetes 5(4): 546-556.
11. Farahani RM, Kloth LC (2008) The hypothesis of 'biophysical matrix contraction': wound contraction revisited. Int Wound J 5(3): 477-482.
12. Oloughlin A, Obrien T (2011) Topical stem and progenitor cell therapy for diabetic foot ulcers. Stem Cells Clin Res 978: 579-604.
13. Sibbald RG, Williamson D, Orsted HL, Campbell K, Keast D, et al. (2000) Preparing the wound bed--debridement, bacterial balance, and moisture balance. Ostomy Wound Manage 46(11): 14-22.
14. Landis S, Ryan S, Woo K, Sibbald RG (2007) Infections in chronic wounds. In: 4th (Edn.), HMP Communications, pp: 299-321.
15. Driver VR (2004) Treating the macro and micro wound environment of the diabetic patient: managing the whole patient, not the hole in the patient. Foot and Ankle Q Sem J 16: 47-56.
16. Cervo FA, Cruz AC, Posillico JA (2000) Pressure ulcers. Analysis of guidelines for treatment and management. Geriatrics 55(3): 55-60.
17. Rolstad B, Ovington L, Harris A (2000) Principles of wound management. In: 2nd (Edn.), Bryant R, (Eds.), St. Louis: Mosby, pp: 85-124
18. Ramundo J, Gray M (2008) Enzymatic wound debridement. J Wound Ostomy Continence Nurs 35(3): 273-280.
19. Lebrun E, Canic MT, Kirsner RS (2010) The role of surgical debridement in healing of diabetic foot ulcers. Wound Repair Regen 18(5): 433-438.
20. Edwards J, Stapley S (2010) Debridement of diabetic foot ulcers. Cochrane Database Syst Rev 2010(1): CD003556.
21. Brem H, Sheehan P, Boulton AJ (2004) Protocol for treatment of diabetic foot ulcers. Am J Surg 187(5A): 1S-10S.
22. Enoch S, Harding K (2003) Wound bed preparation: the science behind the removal of barrier to healing. Wounds 15(7): 213-229.
23. Jain AC (2014) A New Classification (Grading System) of Debridement in Diabetic Lower Limbs-an Improvization and Standardization in Practice of Diabetic Lower Limb Salvage around the World. Medicine Science 3(1): 991-1001.
24. Tallis A, Motley TA, Wunderlich RP, Dickerson JE, Waycaster C, et al. (2013) Clinical and economic assessment of diabetic foot ulcer debridement with collagenase: results of a randomized controlled study. Clin Ther 35(11): 1805-1820.
25. Lebrun E, Canic MT, Kirsner RS (2010) The role of surgical debridement in healing of diabetic foot ulcers. Wound Repair Regen 18(5): 433-438.

26. Saap LJ, Falanga V (2002) Debridement performance index and its correlation with complete closure of diabetic foot ulcers. Wound Repair Regen 10(6): 354-359.

27. Cardinal M, Eisenbud DE, Armstrong DG, Zelen C, Driver V, et al. (2009) Serial surgical debridement: a retrospective study on clinical outcomes in chronic lower extremity wounds. Wound Repair Regen 17(3): 306-311.

28. Paola LD, Faglia E (2006) Treatment of Diabetic Foot Ulcer: an overview strategies for Clinical Approach. Current Diabetes Reviews 2(4): 431-447.