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Diabetic foot ulcer managed with platelet rich plasma- A case report & review

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Abstract

Diabetic foot is a frequent site for complication in DM. Ulceration occurs as a result of trauma in the presence of neuropathy and/or peripheral vascular disease with infection as a secondary phenomenon following disruption of the protective epidermis. We recorded a case of diabetic foot ulcer in 58 year old female patient managed with platelet rich plasma (PRP).

Keywords: Diabetes mellitus, Diabetic foot, Platelet rich plasma

Introduction

Diabetes mellitus (DM) is one of the most deceitful diseases that affect more than 371 million people all over the world in 2012; by 2030 this will rise to 552 million. Diabetes mellitus is a clinical syndrome characterized by hyperglycemia caused by absolute or relative deficiency of insulin ^[1]. Diabetes mellitus is of two types. Type 1 DM was previously known as insulin dependent diabetes mellitus (IDDM). It is a T-cell mediated autoimmune disease involving destruction of the insulin secreting beta cells of islet of langerhans of pancreas. Hyperglycemia accompanied by the classical symptoms of DM occurs only when 70-80% of beta cells have been destroyed. Type 2 DM was previously termed as non insulin dependent diabetes mellitus (NIIDM) ^[2].

Diabetic foot is a frequent site for complication in DM. Ulceration occurs as a result of trauma in the presence of neuropathy and/or peripheral vascular disease with infection as a secondary phenomenon following disruption of the protective epidermis ^[3]. More than 60% of diabetic foot ulcers are the result of underlying neuropathy. The development of neuropathy in affected patients has been shown in animal and in vitro models to be a result of hyperglycemia-induced metabolic abnormalities ^[4]. One of the more commonly described mechanisms of action is the polyol pathway. In the development of neuropathy, the hyperglycemic state leads to an increase in action of the enzymes aldose reductase and sorbitol dehydrogenase. Hyperglycemia and oxidative stress also contribute to the abnormal glycation of nerve cell proteins and the inappropriate activation of protein kinase C, resulting in further nerve dysfunction and ischemia ^[5]. We recorded a case of diabetic foot ulcer in 58 year old female patient managed with platelet rich plasma (PRP).

Case report

A 58 year old female patient visited the orthopaedics department with complaint of ulcer of right foot since 1.5 years. History revealed that ulcer started 2 year back as small size which gradually increased to attained the present size. Patient was diabetic since 14 years and was not under routine medication.

Her vital sign was within the normal limit. General physical examination was done which revealed a large irregular ulcer approximately 36.2 cm² in maximum dimension. There was necrotic tissue over wound, positive for pus, exudates, and bad odor.

We performed a surgical debridement to extend the wound and drain the pus. Wound was cleaned with normal saline and scrapped & pressed to achieve control of bleeding if any. Then sterile gauze was soaked in PRP and applied over the wound area & dressing was done. Patient was advised to walk with partial weight bearing. Dressing was opened after one week when similar dressing was done again. Then one week later simple normal saline dressing was done & one week later 3rd PRP dressing was given. Then at weekly interval normal saline dressings was done upto 6 weeks and then at 8 weeks. At every dressing, area of the wound was measured by taking maximum length and breadth. There was significant regression in size of the ulcer and prognosis was good.

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Discussion

Peripheral arterial disease (PAD) is a contributing factor to the development of foot ulcers in up to 50% of cases. It commonly affects the tibial and peroneal arteries of the calf. Endothelial cell dysfunction and smooth cell abnormalities develop in peripheral arteries as a consequence of the persistent hyperglycemic state [6]. There is a resultant decrease in endothelium-derived vasodilators leading to constriction. Further, the hyperglycemia in diabetes is associated with an increase in thromboxane A₂, a vasoconstrictor and platelet aggregation agonist, which leads to an increased risk for plasma hypercoagulability [7]. We reported a case of diabetic foot ulcer in 58 year old female patient.

Debridement is a method to facilitate the removal of dead/necrotic tissue, cell debris or foreign bodies from a wound. It improves the healing potential of the remaining healthy tissues. A dead tissue can prevent the wound from healing and make wound vulnerable to infection. There are many techniques of debridement can be used by the healthcare professionals. However, they can be divided into an active debridement and an autolytic debridement [8].

Preventing progression of DFUs is important. A comprehensive treatment must be done to improve the outcome and to limit the risk for amputation. The main goal of the treatment is the wound healing process. A wound healing is a complex process because it involves growth factors, responses of cells, and a good clinical care. The fundamentals are moist wound care, frequent debridement, offloading, treatment of infection, and revascularization of the ischemic limb [9].

Irawan *et al.* [10] recorded a case in 45-year-old patient with wound on her right foot since 1.5 months ago. The wound did not heal and became larger with bad odor and pus. She had type 2 diabetes mellitus since five years ago with uncontrolled blood sugar. They performed surgical debridement to extend the wound and to drain the pus. They used a combination of hydrogel and hydrocellular foam to treat the wound. The overall performance of a combination of hydrogel and hydrocellular foam was shown to have clinical advantages such as autolytic debridement. Authors observed an increase of wound granulation and epithelialization and a decrease of slough and exudates.

We used PRP for the patient. Use of autologous platelet-rich plasma (PRP) in the form of local application obtained by centrifugation of whole blood and addition of an activator, clotting agent is designed for the creation of local conditions favourable to healing processes. PRP is defined as plasma fraction of autologous blood with a platelet count concentrated above the baseline. It is a repository of growth factors, cytokines, adhesion molecules and clotting agents, and leukocytes [11].

Platelets contain numerous natural growth factors released from their α granulations and stimulating healing processes. Ross *et al.* in the in vitro study noted thrombin-activated platelets as a source of growth factors that could initiate the body's natural healing. Added to platelet-poor plasma, they increased activity of smooth muscle cells and fibroblasts. PRP is obtained by repeated centrifugation of autologous whole blood. The resulting concentrate, combined with activating bovine thrombin, forms a gel that seals the wound. The gel is placed on wound bed and protected by a cover dressing. The dressing may stay in place for up to 7 days [12].

Conclusion

Authors found that wound healing through the application of PRP is recent and advanced method. PRP are responsible for actively extruding growth factors, which initiate soft tissue healing and recruitment of stem cell. Platelet rich plasma is safe and effective method for treatment of DFUs.

References

1. Beckert S, Witte M, Wicke C, Königsrainer A, Coerper S. A New Wound-Based Severity Score for Diabetic Foot Ulcers: A prospective analysis of 1,000 patients. *Diabetes Care*. 2006; 29:988-92.
2. Kavitha KV, Tiwari S, Purandare VB, Khedkar S, Bhosale SS, Unnikrishnan AG. Choice of wound care in diabetic foot ulcer: A practical approach. *World J Diabetes*. 2014; 5:546-56.
3. Atkin L, Stephenson J, Bateman S. Foam dressings: A review of the literature and evaluation of fluid-handling capacity of four leading foam dressings. *Wound UK* 2015; 11:75-81.
4. Clayton W, Elasy TA. A review of pathophysiology, classification and treatment of foot ulcers in diabetic patients. *Clin Diabetes* 2009; 27:52-8.
5. Shaw JE, Boulton AJM. The pathogenesis of diabetic foot problems. An overview. *Diabetes* 1997; (46):S58-61.
6. Zochodne DW. Diabetic polyneuropathy: an update. *Curr Opin Neurol*. 2008; 21:527-33.
7. Foster TE, Puskas BL, Mandelbaum BR, Gerhardt MB, Rodeo SA. Platelet-rich plasma: from basic science to clinical applications. *The American journal of sports medicine*. 2009; 37(11):2259-72.
8. Wasterlain AS, Braun HJ, Dragoo JL. Contents and formulations of platelet-rich plasma. *Operative Techniques in Orthopaedics*. 2012; 22(1):33-42.
9. Marlovits S, Mousavi M, Gäbler C, Erdös J, Vécsei V. A new simplified technique for producing platelet-rich plasma: a short technical note. *Eur Spine J* 13:102-06.
10. Irawan H, Yasa KP. A case report of diabetic foot ulcer underwent an autolytic debridement using hydrogel and hydrocellular foam combination. *Bali Med. J.* 2017; 6(3):93-6.
11. Carter MJ, Fyelling CP, Parnell LK. Use of platelet rich plasma gel on wound healing: A systematic review and meta-analysis. *Eplasty*, 2011, 11.
12. Akingboye AA, Giddins S, Gamston P, Tucker A, Navsaria H, Kyriakides C. Application of autologous derived-platelet rich plasma gel in the treatment of chronic wound ulcer: Diabetic foot ulcer. *The Journal of extra-corporeal technology*. 2010; 42(1):20.