

## A Study of Chronic Foot Ulcers for Diabetic Patients

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### ABSTRACT

The local factors of the diabetic foot ulcer are peripheral neuropathy, anatomic foot deformity, trauma, improperly fitted shoes, and history of foot ulceration or lower limb elimination in the past, oedema, callus, incomplete joint mobility and irregular foot pressures. There is a triad of neuropathy, trauma and deformity seen in about two-thirds of the diabetic foot ulcer patients. The other risk factors are dry or fissured skin, toe-web tinea and onychomycosis. In some studies, there are sexual differences and ethnic elements. The international agreement on the diabetic foot devised a foot risk criteria with increased risk for foot ulceration by categorizing foot risk. Patients who have no neuropathy and no history of foot ulcer in the past are said to be low-risk patients. The annual occurrence of a diabetic foot ulcer is predictable to be 2 to 3%. There are two important functions of the foot. They support the body weight as well as act as a lever to propel the body during walking and running. The foot is composed of many small bones so that it can adapt itself while walking on uneven surfaces, rather than being made of single bone which makes it harder to walk on such surfaces. Several risk factors act together and lead to the formation of the foot in diabetes patients. It can be approximately separated into local factors and general or systemic factors. The general factors include poor glycemic control, the period of diabetes, peripheral vascular disease, chronic renal disease, visual loss or blindness and old age. The local factors are peripheral neuropathy, anatomic foot deformity, trauma, improperly fitted shoes, history of foot ulceration or lower limb amputation in the past, oedema, callus, imperfect joint mobility and abnormal foot pressures. There is a triad of neuropathy, trauma and deformity seen in about two-thirds of the diabetic foot ulcer patients.



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### INTRODUCTION

In this section represents the introduction of this research work. The pathomechanisms of a diabetic foot can be separated into primary causes and secondary causes. [1] The primary causes are polyneuropathy, peripheral arterial disease, microangiopathy including thickened basement membranes and osteoarthropathy. [2] The secondary causes are nearby decreased confrontation to infection and inadequate development to security vessels. The two major pathogenesis in the creation of diabetic foot ulcers is neuropathy and ischemia. Several

mechanisms that cause wound development in a diabetic patient are:

- Nerve hypoxia/ ischemia
- Polyol pathway overactivity
- Auto oxidative stress
- Increase in progressive glycation end products
- Increase in protein kinase C
- Gamma-linolenic acid deficiency
- Dysfunction of cytokines
- Collagen molecules dysfunction (like elastin and proteoglycans)
- Mitochondrial dysfunction
- Endothelial dysfunction
- Alteration in immune mechanism
- Deficiency in growth factors
- Increased proteases

Following damage to the tissues, there is an immediate release of tissue factors and other stimulatory factors like expelled collagen, which leads to mechanical, chemical and biological changes. [3] This causes damage to the nerve fibres as well as the vasculature leading to changes in the molecular level. One crucial event which affects the microcirculation is the endothelial dysfunction. [4] They are endothelial proliferation, basement membrane thickening, increase in blood viscosity, decrease in the nitric oxide synthesis, altered microvascular tone and decrease in blood flow. [5] The next factor is altered immune system. There is immunosuppression due to reduced leukocyte activity, abnormal inflammatory response, reduced fibroblast proliferation, impaired basal keratinocytes and reduced epidermal cell migration. [6]

In this, articles represent division 2 of this paper explains the element on the connected works. In section 3 presents the materials and methods adopted and section 4 presents the specifics of the experimentations and discussions. Finally, section 5 accomplishes the paper by sharing our implications and future plans.

## RELATED WORKS

In this segment represents focuses on the related works of this research work. In any diabetic foot ulcer, the causes can be grouped according to the Rothman's model of causation. [7] The model describes two causes, the component cause and the sufficient cause. [8] The component cause is the one which is not sufficient to cause ulcer by itself but

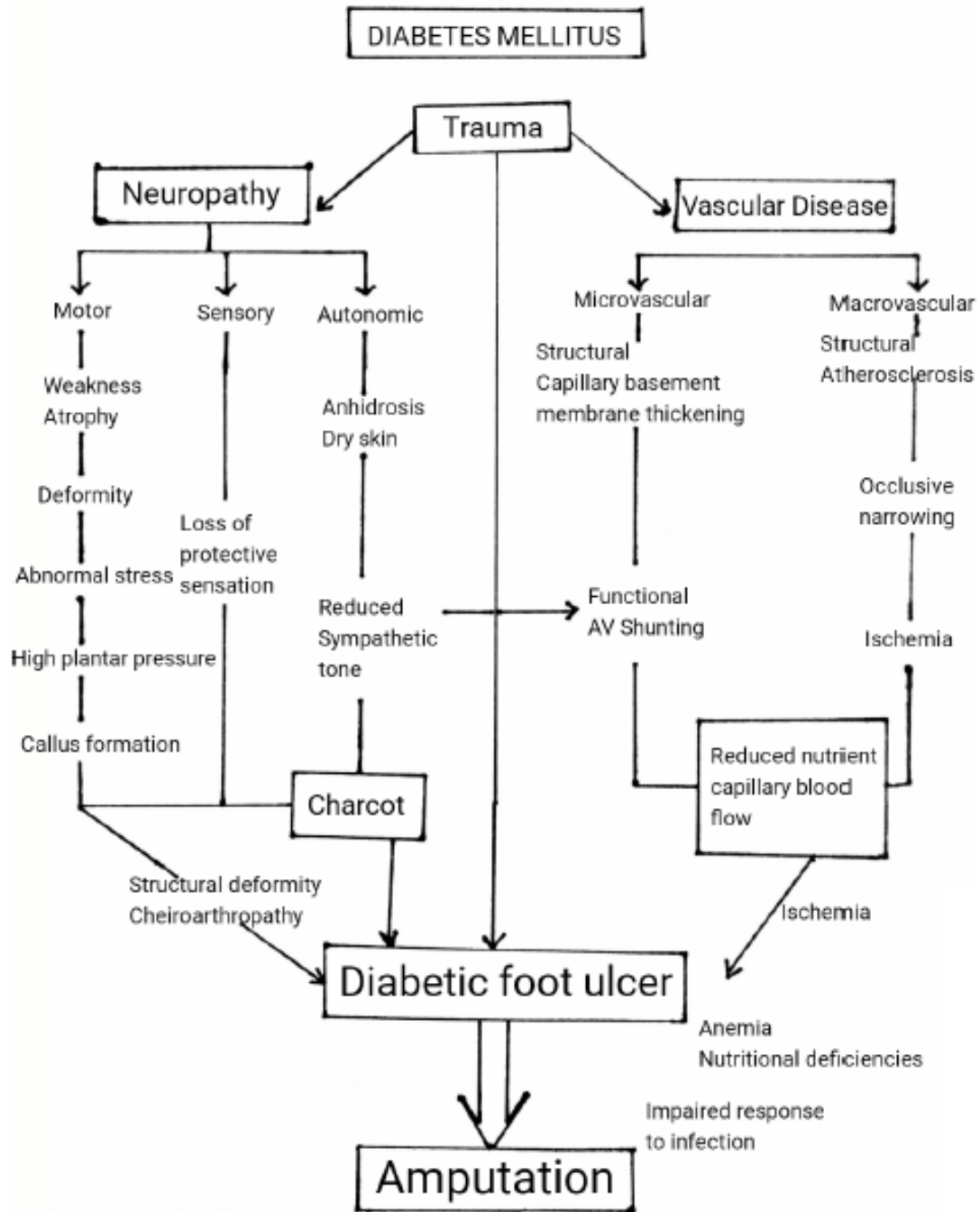
is the required component in the causal chains. [9] The sufficient cause resulting from required component causes is the one which inevitably produces ulcer. [10] By blocking or eliminating the component cause, one can render the action of other components insufficient. [11] For example, neuropathy, trauma, deformity and impaired healing together create a sufficient cause for forming an ulcer. The ulcer cannot be formed in the absence of any of these factors. [12] The third factor is the impairment of nerve axon reflex, which be contingent on the healthy C fibre nociceptor function. [13] The stimulated nerve fibres secrete vasodilators like substance P, histamine, neuropeptide Y and calcitonine gene connected peptide, which leads to vasodilation. This leads to altered Lewis triple flare response which is a compromise in the vasodilatory reaction seen in circumstances of stress, like inflammation or injury in the affected foot of the diabetic patient. [14] This is one of the reasons for slow healing or failure of ulcer to heal even after successful lower limb revascularization. [15] Other associations in diabetes are disturbances in intrinsic wound healing like impairment in matrix metalloproteinase function and collagen cross-linking. [16]

## MATERIALS AND METHODS

In this segment represents the materials and methods of this research work. The approval was obtained from the ethical committee review board. Patients diagnosed with psoriasis and fulfilling the inclusion criteria visiting the department of Dermatology, Venerology and Leprosy in Chennai from January 2019 to January 2020 were chosen. Informed Consent was taken from all the patients. A detailed history was collected regarding the extent of the disease, onset, Past history of Diabetes/ Hypertension, history of burning and alcohol intake were noted. Height and weight of the patients were recorded. Patients BMI was calculated using the formula weight in kilograms by height in meter square. Waist circumference was measured. BP was recorded. Patients were asked to do laboratory blood investigations such as Fasting Lipid Profile, Fasting Blood sugar, Liver function test and ECG. The associated metabolic comorbidities, such as diabetes mellitus, hypertension, dyslipidemia, obesity, cardiovascular disease, were assessed (Figure 1).

## RESULTS AND DISCUSSION

In this segment focuses on the results and discussions of this research work. The total number of patients in the study was 42. They were randomly separated into two groups. The PRP group had



**Figure 1: Pathogenesis of Diabetic Foot Ulcer**

21 patients (50%), and the control group had 21 patients (50%).

At the end of the study (5 weeks), the mean area of the ulcer was 2.89 cm<sup>2</sup> in the PRP group and 6.41 cm<sup>2</sup> in the control group. The standard deviation for the PRP group and control group was 2.12 and 2.65, respectively. The mean area of ulcer among the PRP group and the control group showed important statistical alteration. This shows there is a significant overall discount in the mean area of the ulcer in the

PRP group than that of the control group.

In the PRP group, out of 21 patients, two patients showed complete re-epithelialization of ulcer at the end of 3 weeks, four patients at the end of 4 weeks and eight patients at the end of 5th week. A total of 14 patients (66.67%) showed complete healing with PRP therapy. There was no complete closure of ulcer in the remaining seven patients (33.33%).

In the control group, out of 21 patients, none showed complete re-epithelialization of the ulcer within the

study duration (five weeks).

The mean percentage development in the ulcer area at the end of 5 weeks was 82.19% (SD 9.15) in PRP group and 37.50% (SD 13.16) in the control group with the significant statistical difference between both the groups. This shows that the percentage of ulcer healing is significantly better and faster than that of the control group. A study conducted a study on diabetic foot ulcers with a total number of 14 patients for a period of five weeks. [16] The percentage discount in the wound area was  $71.9 \pm 22.5\%$  in the treatment group and  $9.2 \pm 67.8\%$  in the control group. Reduction of at least 50% in wound area or thorough healing of the wound was observed in 5 patients (71.4%) in the treatment group and two patients (28.6%) in the control group.

A study on diabetic foot ulcers for 12 weeks, showed a favourable outcome in diabetic lower limb wound healing with the use of autologous platelet-rich plasma gel [17]. Out of 19 patients in the treatment group, 13 showed complete healing, and out of 21 patients in the control group, 9 showed comprehensive healing. Also, the mean duration of ulcer healing was smaller in the treatment group than that of the control group. A study on diabetic foot ulcers on 50 patients, where 52% of the patients presented comprehensive healing in the study group, and 20% presented comprehensive healing in the control group. [18]

The following two studies show the use of autologous platelet-rich plasma in chronic non-healing ulcers of various etiologies. The deliberate 24 patients with 33 ulcers. [19] The mean percentage of discount in the area of the ulcer was 91.7%, and complete healing was observed in 24 ulcers (76%). A study on 24 patients out of which 17 (70.83%) patients showed more than 90% reduction in ulcer size. [20]

## CONCLUSION

Finally, this work concludes that the treatment with PRP is found to show significant improvement in ulcer healing, reduction in the duration of treatment and sustainability of improvement as well as shorter length of hospital stay. It is an unassuming, safe, cost-effective and painless process, which also improves the quality of life in the patients. Hence, it is a more desirable treatment option for patients with chronic diabetic foot ulcers, especially when other modalities of treatment have failed or surgical management is contraindicated.

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## Conflict of Interest

Authors declared no conflict of interest.

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