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REVIEW ARTICLE

DETECTION AND MANAGEMENT OF LOWER BODY DEFORMITY AND ULCERATION EXTREMITY IN PEOPLE WITH A LIVED EXPERIENCE OF DIABETES

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ABSTRACT

BACKGROUND:

Diabetes is a silent killer, wherein prolonged poor glucose control could lead to acute diabetic ulcers that are responsible for foot ulcers in the lower body extremities. A diabetic foot is a skin sore formed as a result of skin tissue breaking down and exposing the tissue layers underneath. Chronic conditions of the disease lead to amputation of the limb which is a lifelong disability as well as morbidity.

OBJECTIVE:

We have compiled an interesting and informative review on diabetic foot ulceration. Topics and subtopics discussed in the article have scientific relevance for the readers of health management journals.

MAIN OUTCOME AND RESULTS:

The cascade of events that lead to ulceration is responsible for degrading vascular changes in nerve fibers, resulting in poor motor neuropathy in the lower extremities. Therefore, detection of diabetic foot and ulceration in the early stage is crucial for proper disease management. Various tools in this regard have been used to detect and monitor diabetic foot occurrence apart from a conventional assessment such as the severity of the infection, infection of the skin, extent or size of the ulcer, depth of tissue infection, and loss of sensation from various parts of the lower body. Furthermore, recent advancement in medical technology has also given some critical diagnostic tools EMG (Electromyography), NCV (Nerve Conduction Velocity), PPG (Photoplethysmography), and SSEP (Somatosensory Evoked Potential).

CONCLUSION:

The review discusses various complications related to people with a lived experience of diabetic foot ulcers and some advanced tools to diagnose them. Furthermore, a conclusive discussion on a holistic view of diabetic foot diagnosis methods and available treatments has been summarized which could be more explored for better detection/management of the disease.

KEYWORDS

Diabetes; foot ulcerations; diagnosis methods; available treatment; effective management.

INTRODUCTION

Diabetic foot in chronic form gives rise to complications of foot infection, followed by ulceration and destruction of the tissues leading to amputation of the limb [1]. Around 6% of people living with diabetes are affected by diabetic foot complications [2] and other related lower body extremities leading to foot amputations between 0.03% and 1.5% of cases [3]. The complication causes deep lesions in tissues related to peripheral vascular disease and neurological disorders [4]. The occurrence of diabetic foot disease has increased globally due to the prolonged expectancy of people with a lived experience of diabetes [5]. The lower limb in such cases is detached every 30 s, and it costs (average) $US8,659 per diabetic foot patient annually. The total medical cost for treating the disease of diabetic foot ranges from $US9 to $US13 billion in the USA [6]. Thus, the awareness of diabetic foot problems is increasing including by the International Diabetes Federation (https://idf.org/) which could be helpful to reduce substantial medical, social, and economic burdens.

Out of amputated patients with diabetic foot disease, 85% are headed by ulceration in the foot which consequently worsens to an infection (severe) and decay [7]. To the best of our knowledge, no comprehensive study on the prevalence of diabetic foot ulceration has been investigated globally despite the increasing significance of this disease. Reported studies evaluated the prevalence of diabetic foot ulcers only within a certain period in specific areas and varied considerably in the design of the study or analysis of the population. Therefore, a contemporary evaluation and comprehensive epidemiology of diabetic foot ulcers is critical worldwide. Obtained information from such epidemiological studies could be used to prevent diabetic foot ulcers and improve the quality of a person's life by reducing the economic burden. Looking at this scenario, this article aims to discuss some important available studies dealing with diabetes-related lower body part complications, ulcerations, detection tools or methods currently in use, and available treatment for people with a lived experience of diabetes to understand the current prevalence of diabetic foot ulceration.

METHODS

Lower body deformity and ulcerations are chronic and severe complications observed in a diabetic that consist of lesions in the deep tissues, and lower limbs associated with peripheral vascular disease, neurological disorders and other related conditions [8]. We have done a scoping review on existing literature on the topic. We have searched the topic using PubMed and Scopus with "diabetes-related complications in the lower part of the body, methods of detection, and current treatment options". The literature survey included only peer reviewed papers mostly from 2001 and available in the English language.

DIABETES-RELATED ULCERATIVE COMPLICATIONS

People having Type 1 or 2 diabetes mellitus have a lifetime risk that results in various macrovascular and microvascular complications [9] and some have diabetic foot ulcer disease and other complications (cardiovascular disease and neuropathies). We have discussed some diabetes-related ulcerative complications, in brief, to acquaint with the problem.

FOOT DEFORMITIES

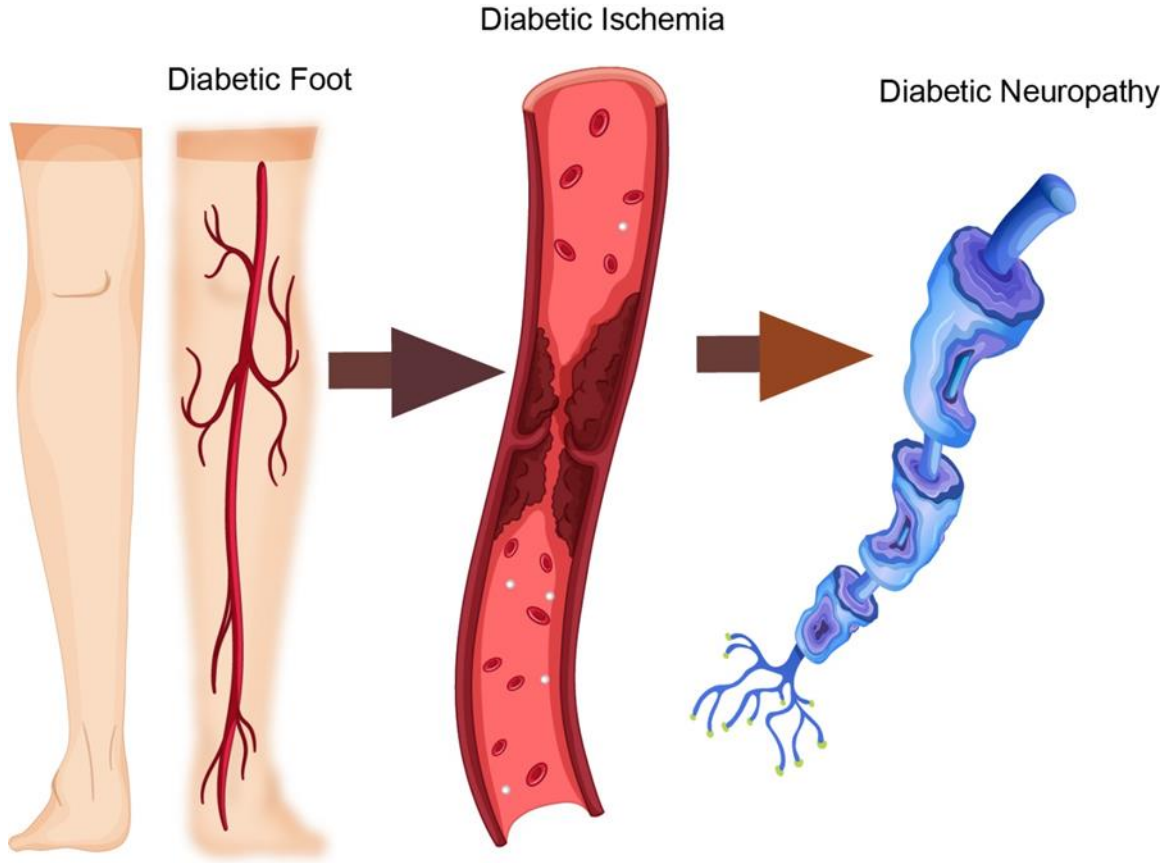
Deformities in the bones, toenails, and soft tissues of the foot are not only prevalent but also more challenging in people with a lived experience of diabetes. Bone deformities cause new pressure points against the foot that can be triggered leading to a cut on the leg or its part. A wound develops once the skin is infected/injured which may cause the collapse of the mid-foot and amputation. It may further induce neuropathy.

NEUROPATHY

Foot bone deformities cause peripheral neuropathy, which further results in muscle atrophy. Peripheral neuropathy affects feet, toes, and legs. Insensitivity to pain, numbness, prickling sensation, etc. are the symptoms of peripheral neuropathy that may include loss of coordination and balance. Peripheral neuropathy may cause loss of reflexes in the ankle and weakness in muscles. Due to unnoticed pressure and injury, blisters and sores may appear on numb areas of the foot. The infection may spread to the bone if an infection occurs and there is no prompt treatment, which ultimately may lead to the removal of a foot from the body [10]. At the site of pressure, loss of sensation takes place, which is accompanied by trauma. Increased pressure further contributes to skin breakdown which is often accompanied by the formation of ulcers. Elevated pressure on the plantar foot contractures in particular areas at the ankle joint, anterior leg musculature, and weakness may contribute to equinus deformity with lack of ample

dorsiflexion and upraise plantar pressures in the forefoot [11]. Vascular complications in diabetic feet further develop diabetic ischemia and ultimately diabetic neuropathy (Figure 1). Ischemia may contribute to the persisting or developing foot ulcers in people with a lived experience of diabetes. [12].

FIGURE 1: VASCULAR COMPLICATIONS OF DIABETES FOOT PLAY A KEY ROLE IN THE INITIATION AND DEVELOPMENT OF DIABETIC ISCHEMIA AND DIABETIC NEUROPATHY



Source: Authors artwork

VENOUS ULCERS

Venous hypertension is the cause of venous ulceration. Venous hypertension contributes to venous obstruction. With the local effects, the result leads to the transmission of elevated venous pressure from the deep to the superficial system of the veins to the ulceration [13]. Multiple hypotheses exist that attempt to explain the direct cause of ulceration the most accepted one is venous hypertension plays a major role in ulceration development. Venous hypertension results in decreased fluid flow and circulation in the capillaries, which results in white blood cell accumulation. These white cells may interfere with tissue oxygenation and release proteolytic enzymes [14]. In the case of venous hypertension, another hypothesis suggests that various macromolecules leak into the region of the dermis and trap the growth factors [15].

ARTERIAL ULCERS

Peripheral arterial occlusive disease is caused due to atherosclerosis. This predominantly affects the superficial popliteal and femoral vessels, reducing the flow of blood to the lower body extremities [16]. Ulceration develops when the ischemia is enough severe. The inflammatory segmental thrombotic disease of the small and medium vessels of lower body extremities is usually associated with smoking. This is considered a prime cause of ulceration and peripheral arterial disease (PAD). When proximal plaques break off and travel distally, atheroembolism may cause peripheral arterial occlusion. This is called blue toe syndrome or cholesterol emboli [17].

DETECTION TOOLS/METHODS CURRENTLY IN USE

Physical examination, claudication, skin examination, palpation of arteries, auscultation of the vessels, etc can be performed in all people with a lived experience of diabetes having trouble in lower body extremities. We have compiled some methods (Table 1) that are used for the detection of the complication.

TABLE 1. DETECTION TOOLS AND METHODS IN DIAGNOSIS OF DIABETES WITH SOME CASE STUDY.

Detection Tool	Case Study	Diagnosis/Observation	References
EMG	Patients with diabetic peripheral neuropathy were tested by EMG	Lower limbs than in upper limbs were found to have abnormal motor nerve conduction. However, EMG was not recommended as standalone technique for diabetes management	[21]
	EMG patterns in diabetic neuropathic observed during step ascending and descending	EMG, three-dimensional motion capture techniques was used to determine the lower limb muscle relaxation and motor neuron behavior. It showed the relation between Diabetic peripheral neuropathy and early muscle activation	[22]
	Ppatients with diabetic neuropathy-distal and proximal nerve behavior was studied	Irregular sensory potential was found in diabetic neuropathy with multiple peaks of short duration demonstrating muscle wasting and dysfunctional motor neuron conduction	[23]
	Use of EMG for Type 2 diabetic neuropathy patient's prognosis based on oxidative stress markers and Body fat mass.	Increased oxidative stresses were observed in Type 2 diabetic patients correlating with decreased nerved conduction. Increased body mass further assisted the increased oxidative stress and Type 2 diabetes	[24]
NCV	Patients with Long duration diabetes leading to diabetic neuropathy	Various factors (Age, duration of diabetes and gender (prominent in male) could significantly contribute to abnormal nerve conduction velocity abnormal (with ankle and knee motor nerve being most affected.	[18]
	10 years survey of NCV correlation with retinopathy and diabetes Type 2	Decrease in NCV was noted when retinopathy increased, even maintaining the HbA1c to balanced level had had no major effect to NCV reduction	[25]
	Type 2 Diabetes patients and occurance of Diabetic Retinopathy	Diabetic Retinopathy patients were found to have reduced sural sensory conduction velocity and tibial motor conduction velocity. The reduced NCV thus was correlated with early onset of Diabetic Retinopathy	[26]

	Patients with Type 2 diabetes mellitus correlating with duration of glycaemic control, NCV and Diabetic neuropathy	Glycosylated haemoglobin had association with HbA1c levels and sural nerve amplitude.	[27]
SSEP	Tibial nerve stimulation was observed in healthy subjects and diabetic patients dependent on insulin	The SSEP result correlated with the peripheral and autonomic response test. The finding was that in insulin dependent individuals somatosensory dysfunction is dependent upon extant of peripheral neuropathy and not on the duration of diabetes or glycaemic control.	[28]
	Patients with Type 2 diabetes	It was observed that central as well as peripheral somatosensory were affected in the diabetic Type 2 group, evident from the latency in sensory nerves responsible for progression to severe diabetic neuropathy condition.	[29]
	Study in Diabetes mellitus women patients	Abnormal findings in motor and urodynamic studies correlated with diabetic neuropathy (Urinary dysfunction) and diabetic cystopathy (sexual dysfunction).	[30]
	Chronic diabetes mellitus effect to the brain (cranial diabetic neuropathy)	Lengthening of SSEP in diabetes mellitus Type 2 were observed and postulated for occurrence of diabetic encephalopathy	[31]
PPG	Healthy control and diabetic subject were taken to study Auto-Regressive Moving Average using a black box system	State of diabetes were predicted close to 92.1% specificity leading to control of insulin administration	[32]
	Direct Non-invasive Monitoring of Blood Glucose	Deuterated water with 5% glucose showed distinct absorption spectra was observed by developed TensorTip Combo Glucometer	[33]
	Measuring of Arterial Stiffness by PPG for patients with HbA1c<8% and those with HbA1c>10%	Patients with HbA1c<8% showed higher curve area as compared to those with HbA1c>10% in PPG graph, corresponding to increase in patients with HbA1c>10%	[34]
	Use of Logistic Regression Modeling on PPG data for diabetes management	The model showed the confidence rate of 92.3%, predicting early onset of diabetes by correlating with Type 2 diabetes if HbA1c value exceeds 6.5%.	[35]

ELECTROMYOGRAPHY (EMG)

It is a diagnostic procedure that is highly useful in the management of diabetic neuropathy through electrical stimulus-based tests to determine the health of muscle and motor neurons. According to the action potential pattern in the electromyography, the nervation ratio could be determined. Also, motor conduction could be determined from distal conduction time, peak-to-peak amplitude, and shape of the polyphasic (composed of various phases). Another parameter that could be investigated by EMG is sensory conduction by distal conduction velocity, shape, and duration of conduction potential.

NERVE CONDUCTION VELOCITY (NCV)

This method evaluates the nerve conduction velocity in people with a lived experience of diabetes by relating it with the tendon reflexes. The procedure for NCV is to observe the conduction in sensory branches and motor (Peroneal nerve, Ulnar nerve, Sensory, and Sural nerve). According to the study by Tehrani et al. investigating NCV across wide age groups have a normal conduction frequency of 81.8% [18].

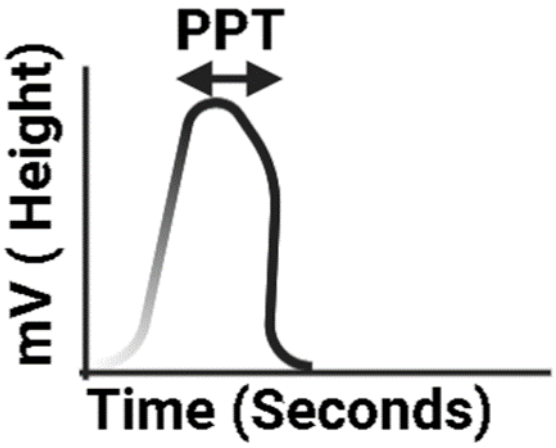
SOMATOSENSORY EVOKED POTENTIAL (SSEP)

This is one of the non-invasive techniques to determine the neurophysiological phenomenon in the dorsal column-lemniscal system (brain) and spinothalamic system (spinal cord). The SSEP potentials are elicited by stimuli that are largely used for the diagnosis of neural disorders. Important to note that SSEP evaluates motor function by detection of vibration and texture including determination of joint position and force movement. At present, the SSEP method still needs more studies to understand the inconsistent deviation readings that are largely affected by age, body type, and glycemic index of people with a lived experience of diabetes.

PHOTOPLETHYSMOGRAPHY (PPG)

This technique specifically targets PAD, which occurs commonly in people with a lived experience of diabetes [18]. PAD warrants special attention due to being a life-threatening disease with high chances of amputation and morbidity. 8-MHz Doppler probes are generally used for Doppler imaging of prominent body areas such as finger, toe, ankle, and brachial. However, in the case of PPG, the predictive model is used to classify diabetes based on the waveform data from the photodiode. This non-invasive technique applies statistical regression, machine learning, and artificial neural networks to determine the severity and classification of diabetes. The electrical output from optical interference thus determines the systolic and diastolic phases, wherein the diastolic notch in-between is of prime significance such as closer of valve and receding of blood flow. Another important data that could be derived from the PPG is arterial stiffness (calcification due to diabetes) by knowing the subject height and PPT, as represented in Figure 2.

FIGURE 2: DETERMINATION OF STIFFNESS INDEX THAT COULD BE DETERMINED FROM SUBJECT HEIGHT/ PPT.



AVAILABLE TREATMENT AND FUTURE THERAPY

The best treatment for lower extremity ulceration is prevention and mechanical therapy is the considered gold standard for treatment for venous ulceration. The best prevention strategy for diabetic foot people is never to go barefoot. Washing of feet every day is advised in warm water, not hot water, and dry feet completely before applying lotion. Elevation of the legs above the heart level may reduce and improve ulcer condition if it is done for 30 minutes 3-4 times daily. Swelling in the lower extremities can be reduced by elevating the legs while sleeping at night. A compression stocking is considered a good approach for the treatment once a venous ulcer develops. For the individual with a lived experience of diabetic foot, the compression stocking is used not only to increase the rate of ulcer healing but also to reduce the rate of recurrence. Compression therapy has the confidence to exert a positive effect on venous ulcers by reducing venous hypertension, improving microcirculation at the cutaneous level, and increasing fibrinolysis. Exerting a pressure of 30-40 mm of Hg is typically useful in settling venous ulceration.

Multilayered compression bandages are also effective in the reduction of amputation. Some patients require compression therapy to attain the healing of ulcers. Some patients are reported with venous ulcers in association with arterial occlusive disease. It needs extreme pharmacologic and compression therapy. Pentoxifylline has been shown to improve the healing of venous ulcers with and without compression therapy. A clinical trial (randomized, and double-blind) of twenty patients treated with enteric-coated aspirin daily dose of 300 mg, was performed by Layton et al., 1994 [19]. They found that the aspirin-treated group had a better reduction in the ulcers' size.

Neuropathy is the entryway to foot ulceration development in people with a lived experience of diabetes. Therefore, the risk of foot ulceration could be reduced by regular screening of neuropathy conditions, the use of custom footwear, intensive podiatric care, etc. Once ulceration develops, treatment should focus on pressure relief and avoidance of infection. For off-loading the diabetic foot, the total contact casting method is a gold standard. With pressure relief, the cast cannot be removed, which further reduces the risk of patient noncompliance. The cast application is more demanding, but they are applied in the presence of technical expertise with proper care. Cast application is not applied in the presence of infection or excessive drainage. Removable cast walkers are used commonly to off-load the diabetic foot in place of the total contact cast. Because the device is removable, therefore, the wound can be supervised daily and can be used even in the presence of infection because infection management is the most critical aspect of diabetic foot treatment. People with a lived experience of diabetes may remove the device during bathing and sleep. Infected foot ulcers are life-threatening. Infections should be treated empirically, and beta-lactamase and cefazolin are used in case of infections. Definitive therapy may then be instituted when results are reported with resistant microbial culture. Incision and drainage should be performed when an abscess or deep infection is observed. Antibiotic therapy combined with surgical removal of infected bone is necessarily done in case of osteomyelitis.

A specialist from vascular surgery and vascular medicine should be consulted for the treatment of arterial ulcers. Gangrenous tissue must be removed, and it often requires amputation partially. Vascular consultation is needed in amputation to determine the appropriate level of care. It must be determined whether the patient is a candidate for the process of peripheral revascularization. The wound does heal without sufficient oxygenation in tissue. Therefore, radiologists also perform procedures which is less invasive vascular, which may increase blood flow peripherally. To promote granulation and prevent infection, wounds must be kept moist and clean. Due to these complications immunity of the body becomes weak. Therefore, the immunopathy approach could be effective and may delay the healing of the wound. Some of the complications of immunopathy include adverse reactions to drugs and foreign body reactions [20] but it could be a futuristic therapy.

CONCLUSIONS

Hyperglycemia, vascular abnormalities, and metabolic changes play a vital role in the development of foot deformities and ulcerations in people with a lived experience of diabetes. There are some products in terms of wound care and dressings available in the market that give temporary relief only to people with a lived experience of diabetic foot and there should be a better longer-term treatment option. Living skin equivalents, negative pressure wound therapy, silver-impregnated dressings, topical growth factors, etc. are some modalities available and used for ulcerative diabetic foot treatment and care. However, these things are not satisfactory for people with a lived experience of diabetic feet. Wounds in the case of diabetic foot are resistant to healing and some peoples benefit from an available treatment option, however, effective modalities for the healing of chronic diabetic foot ulcers and their appropriate management are needed by establishing some cost-effective therapies. Clinicians have to think about some new approach for its early detection and proper management because cellular events involved in its development is the common denominator between the vascular abnormalities and metabolic factors detected in the case of people with a lived experience of diabetic foot ulcer. Available detection and treatment methods could be explored more judiciously in the future for better detection and to provide well-coordinated care and management to the people with a lived experience of diabetic foot ulcers and further clinical studies are needed for the development of new approaches for the treatment of diabetic foot ulcers.

DECLARATIONS

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CONFLICT OF INTEREST

The authors do not have any conflict of interest.

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