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EFFECT OF CUSTOMIZED INSOLE ON FOOT PRESSURE DISTRIBUTION AND GAIT KINETIC PARAMETERS IN SUBJECTS WITH DIABETIC NEUROPATHY

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ABSTRACT

BACKGROUND: High plantar pressures have been shown to be a key risk factor for foot ulceration in people with diabetes. The main goal of preventative insoles prescribed for people with diabetes is pressure offloading, so the most reliable method to check if insoles are effective is through kistler force plate. The purpose of the study is to determine Impact of customised insole on Ground Reaction Force and pain in persons with diabetic neuropathy.

SUBJECTS AND METHODS: 30 diabetic subjects with unilateral neuropathy were included in the study by convenience sampling method.

RESULTS: There was no significant improvement in pain and however in ground reaction force there was significant improvement after using customised insole. Center of pressure was significantly differ at eye open with Orthosis in Ax (t=2.048, p<0.05) and Ay (t=2.048, p<0.05). Pain rating mean pre with Orthosis (6.033+0.808) and post with Orthosis (5.9666+ 1.098) is measured by visual analog scale were significantly higher (t=2.001,p<0.789) before use the Orthosis. Ground reaction force were significantly differ Fx (t=2.048, p=<0.005), Fy (t=2.048, p<0.05) and Fz (t=2.048,p=0.002) with Orthosis.

CONCLUSION: Customised insole significantly reduces pain and normalises center of pressure and ground reaction force in subjects with diabetic neuropathy.

Key words: *Customized Insole, Center of Pressure, Ground Reaction Force, Visual Analog Scale, Kistler Force Plate*

CHAPTER 1

INTRODUCTION

Diabetic Mellitus (DM) is a metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism resulting from defects of insulin secretion, insulin action or a combination of both.[1] Diabetes mellitus (DM) is one of the most common and rapidly increasing health problems worldwide.[2] Most alarming is the steady increase in type 2 diabetes, especially among young and obese person.[3] With rising obesity rates, the risk of adolescent Type 2 diabetes is also increasing dramatically amongst Adolescent.[4 & 5]

The major morbidity associated with somatic neuropathy is foot ulceration, the precursor of gangrene and limb loss. Neuropathy Increases the risk of amputation 1.7-fold, 12-fold if there is deformity (Itself a consequence of neuropathy), and 36-fold if there is a history of previous ulceration.[6] Diabetic Neuropathy (DN) also has a tremendous impact on patients quality of life (QOL) predominantly by causing weakness, ataxia, and incoordination, predisposing to falls and fractures.[7] Once autonomic neuropathy sets in, life can become dismal and the mortality rate can approximate 25% to 50% within 5 to 10 years.[8 & 9] Diabetes can cause two problems that can affect the feet- Diabetic Neuropathy (DN), Peripheral Vascular Disease (PVD).[10] Sensory neuropathy leads to loss of the sensation of pain, pressure and heat. Clinical problems of diabetes in foot are athletes foot, fungal infection of nail, callus, corns, blisters, bunions, dry skin, hammer toes, ingrown toenails, plantar warts, foot ulcers, foot drop, equines, prominent metatarsal heads.[11] Neuropathy is present in about 45-60% of foot ulcers in patients with diabetes. Ischemia is a major factor in 45% of case of foot ulcers.[12]

NEUROPATHIC ULCER - Trauma to the foot in the presence of sensory neuropathy is an important component cause of ulceration. While trauma may include puncture wounds and blunt injury, a common injury leading to ulceration is moderate repetitive stress associated with walking or day-to- day activity. This is often manifested by callus formation under the metatarsal heads. Peripheral arterial disease (PAD) rarely leads to foot ulcerations directly. However, once ulceration develops, arterial insufficiency will result in prolonged healing, imparting an elevated risk of amputation.[16, 17] Limited joint mobility has also been described as a potential risk factor for ulceration. Soft tissue changes (other than cheiroarthropathy) in the feet of diabetic patients might also contribute to ulceration through the pathway of altered pressure distributions through the sole of the foot.[18] Two common mechanisms by which foot deformity and neuropathy may induce skin breakdown in persons with diabetes. The mechanism of injury refers to prolonged low pressure over a bony prominence i.e., bunion or hammertoe deformity). This generally causes wounds over the medial, lateral, and dorsal aspects of the forefoot and is associated with tight or ill-fitting shoes.[19] The other common mechanism of ulceration involves prolonged repetitive moderate stress. This normally occurs on the sole of the foot and is related to prominent metatarsal heads, atrophied or anteriorly displaced fat pads, structural deformity of the lower extremity, and prolonged walking.[20] After a patient with diabetes develops an open wound, closure of the foot wound is hampered by both physiologic impairments in wound healing and an increased susceptibility to wound infection. Guidelines for prevention and treatment of diabetic foot ulcers emphasize that healing is accelerated, and that morbidities and amputations are decreased if infection is prevented and adequate offloading is achieved.

Proper off-loading and pressure reduction prevents further trauma and promotes healing. This is particularly important in the diabetic patient with decreased or absent sensation in the lower extremities.[21, 22] The ways of off-loading are- customized insoles, total non- weight bearing: crutches, bed rest, wheelchair, total contact cast, foot cast, removable walking brace with rocker bottom sole, patella tendon bearing braces, total contact Orthosis -custom walking braces. The choice of off-loading modality should be determined by the patients physical characteristics and ability to comply with treatment as well as by the location and severity of the ulcer. Customized insole can prescribe to diabetic neuropathy patients. It distributes equal pressure on the foot and maintains the balance and it also keep the foot safe from ulcers. In case of foot ulcer, offloading can be done to avoid pressure on that area. Peak plantar pressures are highest in the forefoot, compared with the rear foot and medial arch. Irregular biomechanics, such as those caused by limited joint mobility and/or structural foot deformity, can contribute to abnormal pressure on the plantar foot surface. The goal of tissue-load management is to create an environment that enhances soft-tissue viability and promotes wound healing.[23] Various health care centers prefer specific initial modalities, but frequently clinicians must alternate treatments based on the clinical progress of the wound. Even as simple a method as a felted foam aperture pad has been found to be effective in removing pressure and promoting healing of foot ulcers. A study published in 2001 noted that use of a total contact cast (TCC) healed a higher portion of wounds in a shorter time than a half shoe or removable cast walker (RCW). The most important contribution of shoes and orthotic devices to the prevention of ulcers is the redistribution of forces that impact the foot. For vulnerable and intact of skin, forces can be reduced by optimizing the shape and minimizing the stiffness of materials used to fabricate the shoes and insert. When a patient has no ulcer (grade 0) and no deformity and can sense the 10g monofilament, he or she has protective sensation and usually does well with standard shoes of correct size and shape and a simple shock absorbing pad. In other words, this patient will sense pain before damage occurs to the foot. The patient with no ulcer and deformity but without protective sensation requires depth shoes with a total contact accommodative insert to redistribute pressure, thereby reducing forces on areas of potential breakdown. The insert can be moulded to the patient and can be fabricated over a cast of the foot. The cast does not have corrective forces added, only accommodation.[24]

ROLE OF CUSTOMISED INSOLE IN DIABETIC NEUROPATHY FOOT - Custom made insoles or total contact insoles are offering support across the whole plantar aspect of the foot. This type of insole is thought to accommodate deformities and relieve areas of excessive pressure by evenly distributing pressure over the entire plantar surface. Total contact insoles maximize the contact area with the foot and provide arch support in the midfoot region, which has been shown to help to unload the metatarsal and heel regions. By using these subject experiences, a greater uniform pressure distribution, increased comfort, and less pain.

Custom made insoles (CMI) are prescribed primarily, if not exclusively, to protect the plantar surface of the foot. The CMIs were also very effective in the heel region. Plantar pressure (PP) decreased substantially in both medial and lateral heel regions in comparison to the flat insoles, and force time integral (FTI) decreased significantly in the lateral heel. In the medial heel, pressure may have been redistributed within the boundaries of the region by the arch support, which almost certainly extended into the medial heel mask in some cases. Thus, the medial heel FTI was unchanged. The decrease in PP and FTI in the heel is most likely caused by two mechanisms: pressure redistribution through the effect of the medial arch support and bilateral cupping of the heel.

Heel cupping is established by molding the insole around and up the periphery of the heel. The soft tissue of the heel pad is presumably maintained by the CMI in position underneath the bony prominences of the calcaneus, whereas it is displaced in a flat insole. Dramatic pressure reductions were achieved in the heel as a result of load redistribution by the highly effective action of the medial arch support and, presumably, by cupping of the heel. These effects were very consistent across subjects. Despite significant group results at MTH1, the CMIs were variable in their pressure-relieving and load-redistributing effect on an individual level, and no improvement compared with flat, over-the-counter insoles was achieved.[25]

CHAPTER 2
MATERIALS AND METHODS

After approval by the institutional ethical committee, the study was conducted by convenient sampling method of 30 participants with unilateral foot ulcer due to diabetic neuropathy population attending outpatient, Inpatient Rehabilitation ward of N.I.L.D, Kolkata. A convenient sample of 30 diabetic neuropathy patients with unilateral ulcer was selected in this study; they were randomly assigned for offloading modality i.e., customized insole. The subject with diabetic neuropathy reporting to P&O OPD of NILD was first screened through the inclusion and exclusion criteria. The subject fulfilling the criteria was included in the study. The procedure was fully explained to the subject and a written consent.

Firstly, the participant was assessed and evaluated. The patient's demographic data such name, age, sex, height and weight were collected on a pretested Performa. The diagnosis of diabetes was made by the Endocrinology specialist from MEHA DIABETIC FOUNDATION, or physician of NILD. Patient will be included in the study by convenient sampling method. Pain was assessed by Visual Analog Scale (VAS), foot pressures and ground reaction force were measured by force plate as a baseline data. The foot ulcer was assessed using Wagner classification system, the cases of Wagner Gr. 0 and Gr. I was included in the study. Orthotic assessment and physical evaluation for diabetic neuropathy and foot ulcer was done. Measurement and casting were taken for fabrication of customized insole (Figure 1). Sensation of foot plantar surface is checked by pin prick and light touch. Pain is measured by VAS scale and if ulcer present then ulcer width and depth are measured. Then measurement of fore foot, mid foot, rear foot and length of foot were taken for customization of insole.

Figure 1: Shoe with Customized Insole

Figure 2: Subject on Force Plate without Orthosis

Figure 3: Subject on Force Plate with Orthosis

The subjects (n=30) were asked for stand and walk on self-selected speed over force plate with and without Orthosis (Figure 2 & Figure 3). Then the standard parameters were recorded. After the pre intervention data the subjects were asked to use the insole for 4 weeks. After 4 weeks the subjects were called for follow up and post intervention data were taken. With the application of insoles, the subjects were asked to walk for 10-meter walkway test at a self- selected speed and the standard parameters for pressure distribution and ground reaction force were measured through force plate.
After the pre and post data collection, data analysis was done using analysis tools. Finally, patient was discharged from the department with advice of care and maintenance of final finished customized insole.

CHAPTER 3
STATISTICAL ANALYSIS

Raw data were exported from kistler force plate into Microsoft Excel, and final data analysis was performed in SPSS version 24.0. The data were explored using appropriate descriptive and graphic techniques. Each data set was examined for a normal distribution prior to conducting any inferential analysis.
For the qualitative or scale score data we used F-test. Statistically the results were analyzed using t - Test. Paired t - test was used to analyze the effect of center of pressure and ground reaction force on the pre post with Orthosis and without Orthosis. The result was considered significant at $p \leq 0.05$. Pain was measured in VAS scale. The t- value and p- value is calculated using t test. In each case, the formula for some test statistics that are exactly follows and closely approximates a t-distribution under the hypothesis is given.
Once a t-value is determined, a p-value can be found using a table from student's t- distribution. If the calculated p-value is below the threshold chosen for statistical significance, then the null hypothesis is rejected in favour of the alternative hypothesis.
$P \leq 0.05$ was considered for statistical significance.

CHAPTER 4
RESULTS

Thirty subjects were evaluated for the study with age range from 40 to 60 years. Their heights, weight was recorded. Table 1 represents the mean and standard deviation of these scores. There was no drop out during the study. Data were collected after the completion 4 weeks of post intervention session.

Table 1: Result of demographic data of 30 subjects

	Age (Years)	Height (cm)	Weight (kg)
Mean_+ SD	50.066+-6.119	163.533-+10.136	62.134-+9.651

Table 2: Distribution of Pain with Orthosis

VAS	Mean	SD	T- Value	P- Value
PRE	6.033333	0.808717	2.001717	0.789837
POST	5.966667	1.098065		

Table 3: Result of Pre post Mean distribution of COP Ax & Ay without Orthosis in eye open condition.

Distribution of Mean Ax (EO) With Orthosis				
	Mean	**SD**	**T- Value**	**P- Value**
PRE	0.015536	0.009993495	2.048407	<0.05
POST	0.025763	0.000674		
Distribution of Mean Ay (EO) With Orthosis				
PRE	0.003137	0.004256703	2.048407	<0.05
POST	0.01082	0.000616		
Distribution of Mean Ax (EO) Without Orthosis				
PRE	0.003342	0.001934303	2.048407	<0.05
POST	0.000529	0.000363543		
Distribution of Mean Ay (EO) Without Orthosis				
PRE	0.003228	0.002302653	2.048407	0.00216
POST	0.009076	0.008775737		

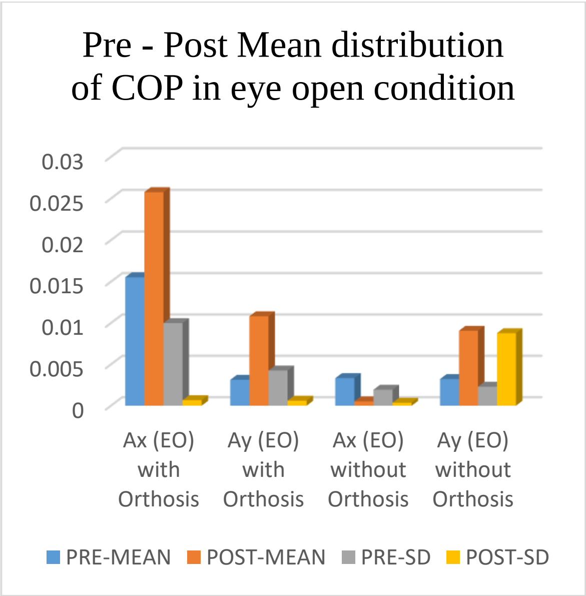
Table 4: Result of Pre post Mean distribution of COP Ax & Ay without Orthosis in eye close condition.

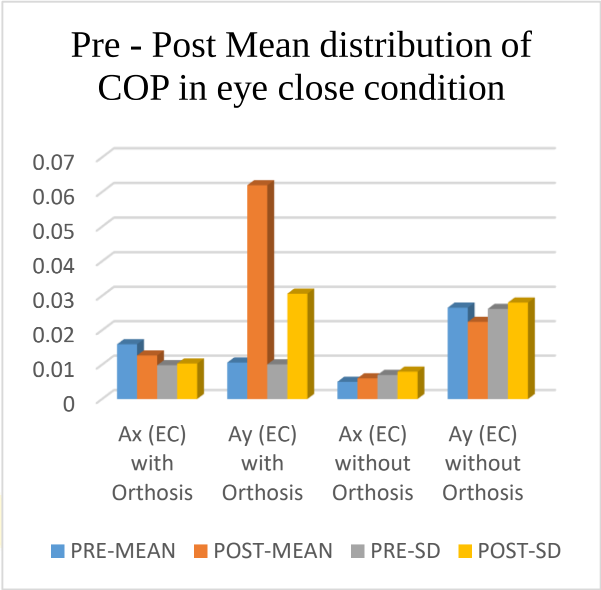
Distribution of Mean Ax (EC) With Orthosis				
	Mean	**SD**	**T- Value**	**P- Value**
PRE	0.015921	0.009821	2.001717468	<0.213
POST	0.012653	0.010317		
Distribution of Mean Ay (EC) With Orthosis				
PRE	0.010596	0.010083656	2.048407	<0.05
POST	0.06193	0.03061111		
Distribution of Mean Ax (EC) Without Orthosis				
PRE	0.005001	0.006999	0.6999942	0.069994
POST	0. 006018	0.007947		
Distribution of Mean Ay (EC) Without Orthosis				
PRE	0.026552	0.026198	2.001717468	<0.684117
POST	0.022426	0.028023		

Table 5: Result of Pre post mean distribution of GRF Fx, Fy & Fz with Orthosis

Distribution of Ground Reaction Force Fx with Orthosis				
	Mean	**SD**	**T- Value**	**P- Value**
PRE	0.397739	0.128690833	2.048407	<0.05
POST	0.247718	0.169565		
Distribution of Ground Reaction Force Fy with Orthosis				
PRE	0.242791	0.141314264	2.048407	<0.05
POST	0.417889	0.153647		

Distribution of Ground Reaction Force Fz with Orthosis				
PRE	0.261607	0.123331412	2.048407	0.002075
POST	0.39295	0.17142		

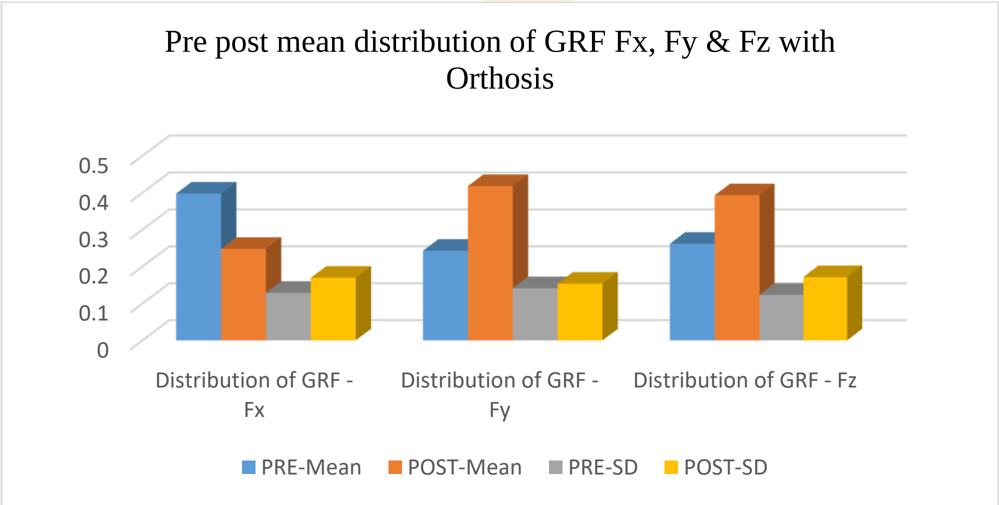




Graph-1

Graph-2

Graph 1: Pre post Mean distribution of COP Ax & Ay without Orthosis in eye open condition.
Graph 2: Pre post Mean distribution of COP Ax & Ay without Orthosis in eye close condition.



Graph-3

Graph 3: Pre post mean distribution of Ground Reaction Force Fx, Fy & Fz with Orthosis

CHAPTER 5
DISCUSSION

The objective of the study was to find the effect pressure distribution in foot pressure in terms of center of pressure GRF vector and pain in pre post condition using foot Orthosis. Logistic regression analyses were performed individually for each insole condition to understand the relationship between the subject characteristics and plantar pressure increase/decrease. These post results were then compared with the pre results characteristics which may be used as a guide for insole prescription.

The purpose of the study was to investigate the impact of customized insole on center of pressure distribution, ground reaction force distribution and pain in persons with diabetic neuropathy. In this study, the presence of ulceration in the neuropathic foot was assumed to be an indicator of a worsening progression of neuropathy.

This study expressed the result of distribution of center of pressure in eye open condition (pre) with Orthosis Ax (0.015536±0.009993495) and post eye open condition with Orthosis Ax (0.025763±0.000674) which is statistically significantly different ($p<0.05$). The distribution of center of pressure eye open condition (pre) with Orthosis Ay (0.003137±0.004256703) and post eye open condition with Orthosis Ay (0.01082±0.000616) and ($p<0.05$) which is a statistically significant (Graph-1).

The result of distribution of center of pressure in pre-eye open condition without Orthosis Ax (0.003342±0.001934303) and post eye open condition without Orthosis Ax (0.000529± 0.000363453) which is statistically significantly different ($p<0.05$). The distribution of center of pressure in eye open condition (pre) without Orthosis Ay (0.003228±0.002302653) and post eye open condition without Orthosis Ay (0.009076±0.008775) and ($p=0.00216$) is a statistically significant (Table-3 & Graph-1).

The result distribution of center of pressure pre-eye close with Orthosis Ax (0.015921±0.009821) and post eye close with Orthosis Ax (0.068667±0.01031) which is ($p=0.2139$) is a statistically non-significant. The distribution of center of pressure pre-eye close condition with Orthosis Ay (0.010596±0.010083) and post eye close with Orthosis (0.06193±0.030611) and which is statistically significant (<0.05) (Graph-2).

The result of distribution of center of pressure pre-eye close without Orthosis Ax (0.005001±0.006999) and post eye close without Orthosis Ax (0.006018±0.007947) which is ($p=0.6999$) statistically non- significant. Same the center of pressure pre-eye close without Orthosis Ay (0.026552±0.026198) and post (0.022426±0.028023) which is ($p=0.0684$) statistically non-significant (Table-4 & Graph-2).

The study expressed the result of distribution of ground reaction force pre with Orthosis Fx (0.397739±0.128690) and post with Orthosis (0.247718±0.169565) statistically significant ($p=0.05$). Like this the ground reaction force pre with Orthosis Fy (0.242791±0.141314) and post with Orthosis Fy (0.417889±0.153647) and this is also statistically significant
($p<0.005$). And the ground reaction of force pre with Orthosis Fz (0.261607±0.1233314) and post with Orthosis Fz (0.39295±0.17142) shows the p value statistically significant ($p<0.002$) (Table-5 & Graph-3)

GRF pathway on foot pressure distribution pattern play a vital role in progression of pain and ulcer in diabetic neuropathy. It was frequently observed that pain and foot ulcer is subjective parameters in diabetic neuropathy. GRF vector have always demanded a wider space to distribution to relief pain or create less pressure.

So as in this present prospective study we hypothesized aim to decrease the COP distribution and compacted the foot pressure distribution by using the foot insole also secondary control the GRF vector to create less expand of foot pressure. As per evident result of this study GRF on gait was significant improved in terms of magnitude in post condition with Orthosis.

Regarding COP distribution, we found significant decreases of COP to make it compact, as foot insoles always trying to make it closed packed position. So, COP pressure over Ax Ay decreases in pre to post conditions.

Paula M H Akashi et al. showed that decreased tactile plantar sensitivity with a larger number of plantar areas with sensory deficit. The sensitivity data confirmed that the history of ulceration indicates more developed state of neuropathy in diabetic neuropathic subjects. The findings of symmetry the association of autonomic neuropathic complications may itself lead to plantar ulceration in diabetic subjects. The EMG time activation and GRF were influenced by the progression and the worsening of plantar sensitivity caused by diabetic neuropathy. Because of the vastus lateralis delay at the moment of initial heel contact, we suggest that mechanism of loading and shock attenuation might be weakened at this stage of diabetic neuropathy, even with no change in the first GRF peak. In my study the ground reaction force decreases after using insole for 4 weeks. This result is due to Orthosis provides little cushioning and less shock absorption which resulting in a greater vertical impact force.

Bunnie Yuk San Tsung et.al. demonstrated that the use of custom molded polyurethane insole could provide a mean reduction of peak pressure of the whole foot. For the mean peak pressure, the percentage changes from shoe only condition in the whole foot was similar for both non weight bearing insole and semi weight bearing insole conditions (about 20% reduction) but the full weight bearing condition had a smaller reduction (14%), while the mean peak contact area was increased by 20-30%. [26] The result of present study shows significance in reduction of pressure in some instances. And the mean Ax eye close with Orthosis in pre post, the mean Ax eye close without Orthosis in pre post, the mean Ay eye close without Orthosis pre post are statistically non-significant. In these conditions the reduction of peak pressure is comparatively less.
Raspovic et.al conducted a study was to evaluate the amount of pressure reduction achieved at focal areas of the foot with the use of custom-made Orthosis. A statistically significant reduction was found in peak pressures between the insole versus no-insole situation ($P<0.01$). Pressure reduction with the orthotic ranged from as much as 93% reduction to as little as 6% reduction which can possibly be explained by the variety of orthotic types and materials used. The reduction in the pressure/time integral with the insole compared to without the insole was statistically significant. This, however, was likely to be due to the degree of pressure reduction with the Orthosis rather than a change in the duration of loading, as duration of loading alone was not significantly altered by the Orthosis.[27] It may be concluded that dynamic plantar pressure is an important parameter which provides information about the human gait. Pressure reduction on the plantar surface shows the reduction in pain and the gait pattern after using insole. Use of insole is beneficial to the patient which is statistically significant ($p<0.05$).
Tatiana Almeida Bacarin et.al showed that neuropathic groups, both non-ulcerated and ulcerated, presented alterations in plantar pressure distribution patterns, and the ulcerated patients presented higher loads than non-ulcerated. The history of foot ulcers in the clinical history of the diabetic neuropathy subjects influenced plantar pressure distribution, resulting in an increased load under the midfoot and rear foot and an increase in the variability of plantar pressure during barefoot gait. The neuropathic groups were composed of diabetics with similar disease characteristics ($p\leq0.05$) according to the duration of disease, type of diabetes and diabetic neuropathy symptoms, which is significant to the study.[28] Similarly in my study the pre post data eye open with and without Orthosis are statistically significant ($p<0.05$). After using insole, the contact surface area under the foot increased and pressure be more evenly distributed. The difference in surface area with and without the orthotic in this study was highly significant, with the surface area increasing during the wearing of customized insole.
Wen-Ming Chen et.al. conducted a study having aim to establish relationships between insole modification, including thickness and MP placement, and stress distributions under metatarsal heads (MTH). It was indicated the pressure seems to drop most substantially due to initial thickness increases, and then becomes less sensitive to further changes in thickness. This is reflected as an _asymptote_ in the pressure reduction curve when insoles are very thick (10.2–12.7 mm). Some important aspects of interactions between the foot and insole/MP may be gained through the stress/strain analysis within the foot structures and the study showed the plantar soft tissue under MTHs gradually and more deeply indented into the adjacent insole materials as thickness of the insole increased.[29] In the present study, a reduction in the difference between the maximum and minimum pressure values indicates that the insoles can really disperse the pressure and this indicates the significance customized insole in diabetic neuropathic foot care.
J. Burns et.al. states in a study foot pain and foot function scores improved with both custom foot orthoses and the sham at 8 weeks of using. Two groups were compared with customized insole and sham Orthosis and there were no significant differences between groups. This study is the first randomized controlled trial to investigate the effectiveness of custom foot orthoses for the treatment of the painful diabetic foot and have significant ($p<0.014$) independent predictor of foot pain reduction at 8 weeks. Same likely in my study the customized insole using duration was 4 weeks for diabetic neuropathic patients having foot pain. We identified predictive variables for foot pain improvement and determined whether changes in plantar pressure were correlated to changes in pain and function. But there is not much improvement in foot pain in such short duration of use of insole which shows the pain is not statistically significant ($p<0.789$) to the study.[30]
The customized insole was manufactured according to the plantar geometry of the users foot in conjunction with accommodative arch support and heel cup mechanisms. This produced a high degree of conformity between the contact surface of insole and the foot contours. The close fit of the insole resulted in a uniform distribution of the pressures in the heel, arch and fore foot region which is significant ($p<0.05$) value in eye open condition. Some studies are focused on the ulcerated subjects showed higher peak pressures under the midfoot and pressure-time integrals under the midfoot and rear foot. The higher peak pressure under the midfoot can be explained by the shift of the loading pattern from the lateral toward the medial part of the

foot and from the rear foot to the anterior part of the foot at the roll over process, which is even more evident in ulcerated patients. But pressure reduction in current study using customized insole is measured in pre post condition in AP and ML view which is statistically significant except Ax, Ay eye close without Orthosis and Ax eye close with Orthosis.

CHAPTER 6
CONCLUSION

The present study has shown that diabetic neuropathic foot decreases the propulsion of vertical ground reaction force during gait. With the application of customized insole there has been seen a significant difference in vertical ground reaction force and center of pressure, thus reducing pain by aligning the foot in normal position. In this current study the comparison made between pre and post intervention data during gait cycle, with and without insole among subjects with diabetic neuropathic foot. The result shows some parameters with significant difference and some with non- significant statistically. However, with the use of customized insole the subject showed a wide acceptance and alleviation from the constant pain. The customized insole is simple and non-invasive methods of offloading in diabetic foot ulcer. The use of customized insole improved the gait parameters, acceptance of the Orthosis and quality of life in subjects significantly. In this study the majority of patients reported improved ability to walk using customized insole. The customized insole allows the clinician with another useful pressure relief method in neuropathic foot ulcers having grade 0 and grade I. Therefore, it may be concluded that the use of customized insole should be encouraged in the diabetic foot ulcer population and needs to be implemented clinically as a treatment option. Further study needed with advance mode of clinical diagnosis to check ulcer healing and improved gait analysis system to collect more accurate gait data.

AUTHORS' CONTRIBUTIONS

The entire clinical course of Effect of Customized Insole on Foot Pressure Distribution and Gait Kinetic Parameters in Subjects with Diabetic Neuropathy service delivery was done by Ms Arpita Nayak towards the fulfilment of a master's degree research project under the guidance of Mr Aratatran Patra & Mr Dhruti Sundar Das. The manuscript preparation is done by Mr. Parthasarathi Swain and Ms Arpita Nayak. All the clinical service delivery to patients and research study was carried out in the premises of NILD, Kolkata.

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REFERENCES

[1] DeFronzo RA. International Textbook of Diabetes Mellitus. 3rd ed. Chichester, West Sussex; Hoboken, NJ: John Wiley; 2004.

[2] Aboderin I, Kalache A, Ben Shlomo Y, Lynch JW, Yajnik CS, Kuh D, Yach D. Life course perspectives on coronary heart disease, stroke and diabetes: key issues and implications for policy and research.

[3] Boulton AJ, Vileikyte L, Ragnarson-Tennvall G, Apelqvist J. The global burden of diabetic foot disease. The Lancet. 2005 Nov 12;366(9498):1719-24.

[4] Centers for Disease Control and Prevention. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the United States, 2011. Atlanta, GA: US department of health and human services, centers for disease control and prevention. 2011 Jan;201(1):2568-9.

[5] Boyko EJ, Ahroni JH, Stensel V, Forsberg RC, Davignon DR, Smith DG. A prospective study of risk factors for diabetic foot ulcer. The Seattle Diabetic Foot Study. Diabetes care. 1999 Jul 1;22(7):1036-42.

[6] Vinik EJ, Hayes RP, Oglesby A, Bastyr E, Barlow P, Ford-Molvik SL, Vinik AI. The development and validation of the Norfolk QOL-DN, a new measure of patients' perception of the effects of diabetes and diabetic neuropathy. Diabetes technology & therapeutics. 2005 Jun 1;7(3):497-508.
[7] Stoffers HE, Rinkens PE, Kester AD, Kaiser V, Knottnerus JA. The prevalence of asymptomatic and unrecognized peripheral arterial occlusive disease. International journal of epidemiology. 1996 Apr 1;25(2):282-90.
[8] Ross R. The pathogenesis of atherosclerosis—an update. New England journal of medicine. 1986 Feb 20;314(8):488-500.
[9] Fowkes FG, Housley E, Cawood EH, Macintyre CC, Ruckley CV, Prescott RJ. Edinburgh Artery Study: prevalence of asymptomatic and symptomatic peripheral arterial disease in the general population. International journal of epidemiology. 1991 Jun 1;20(2):384-92.
[10] Stess RM, Jensen SR, Mirmiran R. The role of dynamic plantar pressures in diabetic foot ulcers. Diabetes care. 1997 May 1;20(5):855-8.
[11] International Working Group on the Diabetic Foot. Diabetes and foot care: time to act. Brussels: International Diabetes Federation. 2005.
[12] Van Deursen RW, Simoneau GG. Foot and ankle sensory neuropathy, proprioception, and postural stability. Journal of orthopaedic & sports physical therapy. 1999 Dec;29(12):718-26.
[13] Boyko EJ, Ahroni JH, Stensel V, Forsberg RC, Davignon DR, Smith DG. A prospective study of risk factors for diabetic foot ulcer. The Seattle Diabetic Foot Study. Diabetes care. 1999 Jul 1;22(7):1036-42.
[14] Pecoraro RE, Ahroni JH, Boyko EJ, Stensel VL. Chronology and determinants of tissue repair in diabetic lower-extremity ulcers. Diabetes. 1991 Oct 1;40(10):1305-13.
[15] Fernando DJ, Masson EA, Veves A, Boulton AJ. Relationship of limited joint mobility to abnormal foot pressures and diabetic foot ulceration. Diabetes care. 1991 Jan 1;14(1):8- 11.
[16] Reiber GE, Vileikyte L, Boyko EJ, del Aguila M, Smith DG, Lavery LA, Boulton AJ. Causal pathways for incident lower extremity ulcers in patients with diabetes from two settings. Diabetes Care 22:157-162, 1999.
[17] Brand PW. Repetitive stress in the development of diabetic foot ulcers. In: The Diabetic Foot, 4th ed, pp 83-90, edited by ME Levin and JK Davidson, Mosby, St Louis, 1988.
[18] Armstrong DG, Lavery LA, Harkless LB. Validation of a diabetic wound classification system: the contribution of depth, infection, and ischemia to risk of amputation. Diabetes care. 1998 May 1;21(5):855-9.
[19] Cavanagh PR, Lipsky BA, Bradbury AW, Botek G. Treatment for diabetic foot ulcers. The Lancet. 2005 Nov 12;366(9498):1725-35.
[20] Llewelyn JG, Gilbey SG, Thomas PK, King RH, Muddle JR, Watkins PJ. Sural nerve morphometry in diabetic autonomic and painful sensory neuropathy: a clinicopathological study. Brain. 1991 Apr 1;114(2):867-92.
[21] Kantor J, Margolis DJ. A multicentre study of percentage change in venous leg ulcer area as a prognostic index of healing at 24 weeks. British Journal of Dermatology. 2000 May;142(5):960-4.
[22] Ling E, Lepow B, Zhou H, Enriquez A, Mullen A, Najafi B. The impact of diabetic foot ulcers and unilateral offloading footwear on gait in people with diabetes. Clinical Biomechanics. 2020 Mar 1; 73:157-61.
[23] Tsung BY, Zhang M, Mak AF, Wong MW. Effectiveness of insoles on plantar pressure redistribution. Journal of rehabilitation research and development. 2004.
[24] Raspovic A, Newcombe L, Lloyd J, Dalton E. Effect of customized insoles on vertical plantar pressures in sites of previous neuropathic ulceration in the diabetic foot. The Foot. 2000 Sep 1;10(3):133-8.
[25] Bacarin TA, Sacco IC, Hennig EM. Plantar pressure distribution patterns during gait in diabetic neuropathy patients with a history of foot ulcers. Clinics. 2009 Feb;64(2):113-20.
[26] Chen WM, Lee SJ, Lee PV. Plantar pressure relief under the metatarsal heads– Therapeutic insole design using three-dimensional finite element model of the foot. Journal of biomechanics. 2015 Feb 26;48(4):659-65.
[27] Burns J, Wegener C, Begg L, Vicaretti M, Fletcher J. Randomized trial of custom orthoses and footwear on foot pain and plantar pressure in diabetic peripheral arterial disease. Diabetic medicine. 2009 Sep;26(9):893-9.