Effect of Adjunctive Hyperbaric Oxygen Therapy versus Traditional Wound Care on Diabetic Foot Ulcer

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Abstract: Foot ulcers are significant sources of mortality, morbidity and diminished the quality of life for patients with diabetes. Hyperbaric oxygen therapy has been proposed as a possible treatment. This study aimed at evaluating the effect of adjunctive hyperbaric oxygen therapy on diabetic foot ulcers, and comparing the adjunction of this form of therapy to traditional ulcer care alone. A comparative randomized clinical research design was utilized. The study was conducted in Naser Health Institute. Seventy two patients were recruited in this study conveniently, with grade I or II of foot ulcer. They were randomized into two equal groups, study group mean age 52.89±3.75; they were subjected to traditional ulcer care plus hyperbaric Oxygen therapy. The control group mean age was 54.44±5.50; they were subjected to traditional ulcer care. The results revealed statistically significant differences between study and control groups regarding the process of wound healing, ulcer size and depth after intervention. This concludes that hyperbaric oxygen therapy HBOT could be used as adjunctive therapy for healing of diabetic foot ulcer, so it is recommended to increase the number of specialized center, Evidence-based guidelines should be used to aid the clinician in determining which patients are suitable candidate for HBOT.

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1. Introduction

Diabetes mellitus was diagnosed in the old Egyptian Civilsation and documented in the Ebers papyrus almost for 3500 years ago. However the modern life style (decreased physical activity, and obesity) are causing an alarming bells to ring for the drastic increase in the number of patients suffering from diabetes. In 1985, it was estimated that 30 million people worldwide were diabetic. 15 years later, the number has already passed the 150 million according to different sources. It is estimated that the number of diabetic patient will be more than doubled between 2030 and 2050. (International Diabetes Federation, 2009).

Foot lesions in patients with diabetes mellitus are a major health problem with significant morbidity and mortality, diminished quality of life (Rakel etal., 2006) and is associated with high economic and social costs. It has been estimated that 20-25% of all hospital admissions of patients with diabetes are due to foot lesions and that foot complications are responsible for more inpatient hospital days than all other diabetic complications combined. About 2.5% of persons with diabetes will develop a foot ulcer each year. Some of these lesions will deteriorate to gangrene or sever deep infection, necessitating minor or major amputation. The rate of lower extremity amputations among persons with diabetes is 15-40 times greater than the rate in person without diabetes. Epidemiological data also suggest that foot ulcers precede 85% of amputations ((Reiber etal., 1995). The risk of death is 2.4 times greater for patients with diabetes who have foot ulcers than for patients with diabetes who do not. (Boyko etal., 1999).

Diabetic foot wounds are defined as any break in the cutaneous barrier, usually extending through the full thickness of the dermis. (Cianci, 2000). Diabetic foot ulcers can be generally classified as either neuropathic or ischemic (Miller, 1998). Pathogenesis of diabetic foot lesions that leading to amputation in people with diabetes result from a combination of events, including peripheral neuropathy, autonomic neuropathy, peripheral vascular disease, foot deformities, trauma, ulceration and infection (Fritschi, 2001). A critical triad of neuropathy, minor foot trauma and foot deformity was found in >63% of people with foot ulcers (Reiber, etal., 1999).

Foot ulcers require multidisciplinary management; standard therapy for lower extremity wounds in diabetic patients entails good glycemic control, nutrition, (Rakel, *et al*., 2006), debridement, off-loading of the high pressure areas, correction of comorbidities, education about foot care, and advice on protective footwear. (Apelqvist etal.,2000). Management may need to extend to antibiotic administration and surgical intervention for deep soft-tissue or bone infection (Lipsky etal., 2004), and/or to revascularization procedures to treat peripheral ischemia. (Berendt, 2006). Except surgical revascularization, unfortunately, none of these therapies effectively increases oxygen delivery to the affected tissue. Hyperbaric oxygen therapy may be a noninvasive alternative to surgical revascularization for the treatment of diabetic foot ulcers. (Heyneman,

etal., 2002).

Hyperbaric oxygen therapy (HBOT) has for long been used in nursing practice. It is widely admitted, that its applications are extremely useful in treating difficult wounds and related traumas (Jin-Ping, 2009). As tissue hypoxia is an important mechanism contributing to the development of diabetic foot, infections and impaired wound healing. Human studies have shown that hyperbaric oxygen exposure increases tissue oxygen levels and thereby results in increased cellular proliferation, improved collagen synthesis and increased angiogenesis. Furthermore, anaerobic organisms are found in low oxygen-tension tissues, which are present in one-third of cases of diabetic foot infections (Bakker, 2000). Hyperbaric oxygen therapy also, increases the killing ability of leukocytes which is lethal to certain anaerobic bacteria (Calhoun etal., 2002, Broussard, 2004, Cianci, 2004, Bakker, 2000). Edema in the periwound area is decreased through the vasoconstrictive action of oxygen and the leukocyte-bacterial-killing ability. Hyperbaric oxygen therapy also enhances phagocytosis of bacteria and inhibits toxin formation (Broussard, 2004, Bakker, 2000& Niinikoski, 2003).

Significance of the study

A series of surveys of diabetes mellitus have been performed in Egypt recently, using a common protocol and WHO criteria for diagnosis and classification found average prevalence for the country as a whole for people above the age of 10 was 4.3%, with distinct geographical differences: 5.7% in urban areas, 4.1% in rural agricultural areas, and 1.5% in rural desert areas. However, the cost of treatment of the various complications of diabetes is higher. So, a tremendous need arise for adjunctive treatments that could reduce the human and economic burden and loss associated with diabetic foot ulcers. As tissue hypoxia is a main pathophysiological characteristic of diabetic ulcers, hyperbaric oxygen therapy has been considered as a therapeutic strategy to reduce tissue hypoxia and enhance wound healing.

Aim of the Study:

This study aimed at

1. Evaluating the effect of adjunctive hyperbaric oxygen therapy on diabetic foot ulcers.
2. Comparing the adjunction of this form of therapy to traditional ulcer care alone.

Research hypothesis:

1. The diabetic foot ulcer patients who exposed to adjunctive hyperbaric oxygen therapy will have their ulcer healed compared to the control group subjects who received the traditional therapy alone.
2. The diabetic foot ulcer patients who exposed to adjunctive hyperbaric oxygen therapy will heal compared to the pre intervention state.

2. Subjects and Methods:

Research design:

A comparative randomized clinical research designed has been utilized in this study.

Research setting:

The study was conducted at Emergency Surgical Department and Hyperbaric Oxygen Unit in Nasser Heath Institute.

Study subjects:

A study subjects of 72 consecutive diabetic patients with grade I or II of foot ulcer who were admitted to Emergency Surgical Department between Jan and July 2011, for the treatment of diabetic foot ulceration. They were randomized into two equal groups, study and control group. Study group patients consisted of 20 males, and 16 females, with their mean age 52.89±3.75, they were subjected to traditional ulcer care plus hyperbaric Oxygen therapy. The control group patients consisted of 16 males and 20 females, with their mean age 54.44± 5.50; they were subjected only to the traditional ulcer care.

Diabetic patients were considered eligible if they were at least 18 years of age, and if they had a foot wound that had been present for at least 4 weeks despite local and systemic wound care. The diabetic patients were excluded if they have any of the following conditions hypoalbuminemia, low plasma protein level, anemia, hyperlipedemia, body mass index more than 30% of average patient weight, smoker, in addition to exclusion of the following cases from the study group as it is contraindicated to be subjected to HBOT (untreated pneumothorax, obstructive pulmonary disease, history of otic surgery, upper respiratory tract infection, febrile state, history of idiopathic convulsion, hypoglycemia, current corticosteroid, amphetamine, catecholamine, or thyroid hormone use).

Tools for data collection:

The following tools utilized to collect data related to this study

Patient data sheet:

It is a patient assessment sheet designed to encompass five parts,

First part concerned with demographic characteristic of the studied subjects regarding age, sex, marital status, and occupation.

The second part include diabetic history that

involve information regarding, type of diabetes, duration of diabetes, glycemic control, presence of diabetic complications, type of diabetic complications (cardiovascular, neurological, ophthalmic, renal, or previous foot ulcer) duration, site(s), wound surface area expressed in square centimeter, depth of ulcer, treatment used (medical or surgical).

Third part concerned with recording of patient medical history (presence of comorbid diseases – liver cirrhosis, heart failure, respiratory failure, and hypertension), smoking hobbit, body mass index, and laboratory parameters (serum albumin, plasma protein, hemoglobin, glycosylated hemoglobin, RBCs, lipid profile).

The fourth part concerned with recording of ulcer characteristic before and after therapeutic intervention and final wound outcome after therapeutic intervention. It includes recording of ulcer surface area expressed in square centimeter, depth of ulcer in centimeters,

The fifth part concerned with hyperbaric oxygen therapy information (no. of sessions, duration of sessions, total time under oxygen therapy, O_2 percentage used, atmospheric pressure used, O_2 dissolved in plasma.

Pain assessment sheet:

It developed by the researchers to assess if the ulcer is neuropathic or ischemic. It includes asking about the presence of pain, type of pain (throbbing, shooting, acute, aching, erosion, spasm, stabbing, stinging, burning, heaviness, tenderness, tiring, exhausting, cruel ,…….) and pain measurement scale to determine the degree of pain felt.

Peripheral tissue perfusion assessment scale:

It has been adopted from Johnson etal. (2000) to assess peripheral tissue perfusion, it composed of five points Likert Scale ranging from Not compromised (5) to extremely compromised (1). It includes assessment of capillary refill, strength, and symmetry of both distal peripheral, and proximal peripheral pulse, sensation level, skin color, muscle function, skin integrity, extremity temperature, presence of extremity bruits, presence of peripheral edema, presence of localized pain, and the presence of other related abnormalities. It was scored as a total summation of 13 items, scored from 13 to 65 points with the higher the score the better the peripheral tissue perfusion).

Ulcer assessment scales

It is developed by the researchers to assess the characteristics of the foot ulcer. It entails three assessment scales

Stage assessment scale: to stage the ulcer according to University of Texas Classification System of foot ulcer (Armstrong, etal.,1998), this classification system staging the ulcer into 4 stages (no infection or ischemia, infection present, ischemia present, infection and ischemia present). It scored from 0 to 3 with the lowest the score the earliest the stage.

Depth assessment scale: it classifies the depth of ulceration into 4 degrees ranging from epithelialized wound (I), superficial wound (II), wound penetrates to tendon or capsule (III), and wound penetrates to bone or joint (IV). It scored from 0 to 3 with the lowest the score the superficial the ulcer.

Ulcer complication assessment scale: it classifies the ulcer complications into 6 classes (infection, infection with osteomylietis, infection with inactive charcot, infection with osteomylities and inactive charcot, inactive charcot, and active charcot. It scored from 0 to 5 with the the highest the score the worse the complications.

Wound Healing Assessment Scale:

It has been adopted from Nursing Outcome Classification System developed by Iowa University Project published at 1997, refined by 2000, and modified by the researchers to assess degree of wound healing after therapeutic intervention, it composed of five points Likert Scale ranging from Non (1) to complete (5). It includes assessment of skin approximation, granulation, epithelialization, resolution of purulent drainage from wound, resolution of serous drainage from wound, resolution of sanguineous drainage from wound, resolution of serosanguineous drainage from wound, resolution of surrounding skin erythema, resolution of periwound edema, resolution of abnormal surrounding skin, resolution of blistered skin, resolution of macerated skin, resolution of necrosis, resolution of sloughing, resolution of tunneling, resolution of undermining, resolution of sinus tract formation, resolution of wound odor, resolution of wound size, and resolution of skin temperature elevation, It was scored as three parts Including (summation of points related to signs and symptoms of wound healing, resolution of wound drainage, resolution of signs of inflammation), with the highest the score the better the ulcer healing process.

Procedures

The investigators went through literature review to adopt and finalize the study tools. After receiving administrative consent from Naser Health Institute consecutive patients who were admitted to the Emergency Surgical Department for the treatment of

infected diabetic foot ulceration were evaluated for potential inclusion in the investigation. In addition to receiving standard medical assessment, each patient was evaluated to determine whether HBOT was contraindicated, after confirming eligibility, the patients were randomly assigned to the conventional treatment group (control group) or traditional treatment plus HBOT group (study group). Traditional care was defined in this study as performing daily wound care that include dressing changes and local debridement at bedside or in the operating room if needed, use of off-loading devices, as well as amputation when indicated. Infection controls were carried out by clinical follow-up, and by performing culture antibiograms of surgically obtained specimens to determine appropriate antibiotic therapy. In the study group, conventional care was supplemented by hyperbaric oxygen treatment administered at a maximum working pressure of 20 atmosphere absolute (ATA), using a unichamber and multichambers pressure room (ATC decompression chamber) employing a volume of 10 m^3 at 2 to 3 ATA for 90 minutes. Treatment was administered as three different protocols, it include either every day session (6 sessions per week), or day after day (3 sessions per week), or every Two days (2 sessions per week) according to the progression of the case. Participants were given a full explanation of the study aims and procedures. Verbal consent was obtained by each patient prior to completing the study instruments.

Limitation of the study:

Only single center was available for the collection of data regarding hyperbaric oxygen therapy in Nasser Health Institute.

3. Results:

Table (1): revealed a non significant differences between study and control group regarding age, gender, and occupation at $p > 0.05$

Table (2): demonstrated the patient diabetic history as there is a non significant difference between the two groups regarding type, duration of diabetes, affection by diabetic complications, and previous affection with diabetic foot ulcer, while there is a highly significant difference between the two groups regarding their previous diabetic control and type of treatment used. The table also showed that most of diabetic patients in both groups were affected previously with diabetic foot.

Table (3): revealed that all the patients in both groups are felt pain, with varying degrees from level 4-7, it also shown a non significant difference between the two groups regarding their peripheral tissue perfusion, ulcer complications, ulcer depth, and ulcer grade.

Table (4) showed a highly significant difference between study and control groups regarding signs of wound healing, drainage resolution, and skin inflammation resolution.

Table (5) revealed a non significant difference between study and control groups regarding ulcer area, and ulcer depth before intervention, while showed a significant difference between both groups after intervention.

Table (6) showed a decrease in ulcer area and depth after intervention compared to pre intervention state with a significant difference between the two groups. No one of both groups need for amputation, while 66.6% of the study group subjects' ulcers closed without need for surgical intervention compared to 33.3% of the control group subjects.

Table (1): Demographic characteristics of the studied patients

Items	Group				test	p-value	sig
	Study group (n=36)		Control group (n=36)				
	No.	%	No.	%			
Age							
Mean±SD	52.89±3.75		54.44±5.50		t test =-1.40	0.16	NS
Gender:							
Male	20	55.6	16	44.4	$X^2 = 0.89$	>0.05	NS
Female	16	44.4	20	55.6			
Marital status:							
Married	28	77.8	20	55.6	$X^2 = 4$	<0.05	S
Single	8	22.2	16	44.4			
Occupation:							
Sedentary work	28	77.8	28	77.8	$X^2 = 0.0$	1	NS
Active work	8	22.2	8	22.2			

Table (2): Diabetic history of the studied patients

Items	Group				test	p-value	sig
	Study group (n=36)		Control group (n=36)				
	No.	%	No.	%			
Type of diabetes							
IDDM	12	33.3	12	33.3			
NIDDM*	24	66.7	24	66.7	$X^2 = 0.0$	1	NS
Duration of diabetes							
Mean±SD	7.22±4.08		9.44±6.85		t = -1.67	> 0.05	NS
Diabetes control							
Yes	20	55.6	8	22.2			
No	16	44.4	28	77.8	$X^2 = 8.4$	0.004	HS
Affection by diabetic complication							
Yes	28	77.8	32	88.9			
No	8	22.2	4	11.1	$X^2 = 1.6$	> 0.05	NS
Type of diabetic complications							
No complications	4	11.1	4	11.1			
Cardiovascular complications	8	22.2	0	0.0			
Neuropathic complications	20	55.6	20	55.6			
Retinal complications	4	11.1	4	11.1			
Renal complication	0	0.0	8	22.2	-	-	-
History of diabetic foot:							
Previous affection by diabetic foot							
Yes	32	88.9	32	88.9			
No	4	11.1	4	11.1	$X^2 = 0.0$	1	NS
Ulcer place							
Left	24	66.7	8	22.2			
Right	12	33.3	28	77.8	$X^2 = 14.1$	<0.001	HS
Treatment used							
Medical	36	100	11	30.6			
Surgical	0	0.0	25	69.4	$X^2 = 38.3$	<0.001	HS
No. of hospital stay due to ulcer							
One week	12	33.3	8	22.2			
Two weeks	0	0.0	4	11.1			
Three weeks	12	33.3	24	66.7			
Four weeks	8	22.2	0	0.0			
Five weeks	4	11.1	0	0.0	-	-	-

*IDDM: Insulin Dependant Diabetes Mellitus NIDDM: Non Insulin Dependent Diabetes Mellitus

Table (3): diabetic foot ulcer assessment for subjected patients

Items	Group				test	p-value	sig
	Study group (n=36)		Control group (n=36)				
	No.	%	No.	%			
Feeling of pain							
Yes	36	100	36	100			
No	0	0.0	0	0.0	$X^2 = 0.0$	1	NS
Type of felt pain							
Spasm	8	22.2	0	0.0			
Erosion	0	0.0	4	11.1			
Burning	8	22.2	4	11.1			
Aching	4	11.1	0	0.0			
Heaviness	12	33.3	16	44.4			
Tiring	0	0.0	8	22.2			
Exhausting	4	11.1	4	11.1	-	-	-
Pain level							
Level 4	4	11.1	12	33.3			
Level 5	16	44.4	4	11.1			
Level 6	12	33.3	8	22.2			
Level 7	4	11.1	12	33.3	$X^2 = 16$	<0.01	HS
Peripheral tissue perfusion							
Extremely compromised	0	0.0	0	0.0			
Substantially compromised	0	0.0	0	0.0			
Moderately compromised	0	0.0	0	0.0			
Mildly compromised	28	77.8	32	88.9			

Not compromised	8	22.2	4	11.1	$X^2 = 1.6$	> 0.05	NS
Ulcer complications							
Infection	23	63.88	16	44.4			
Infection + osteomylities	13	36.22	20	55.6			
Infection + non active Charcot	0	0.0	0	0.0			
Infection + osteomylities+ non active Charcot	0	0.0	0	0.0			
Non active Charcot	0	0.0	0	0.0			
Active Charcot	0	0.0	0	0.0	$X^2 = 0.89$	>0.05	NS
Ulcer depth							
Superficial	36	100	36	100			
Deep	0	0.0	0	0.0	$X^2 = 0.0$	1	NS
Ulcer stage (Texas Classification System)							
No infection or ischemia	4	11.1	12	33.3			
Infection present	4	11.1	8	22.2			
Ischemia present	28	77.8	16	44.4			
Infection and ischemia present	0	0.0	0	0.0	$X^2 = 8.6$	<0.05	S
Ulcer grade							
Epithelialized wound	32	88.9	28	77.8			
Superficial wound	4	11.1	8	22.2			
Wound penetrate to tendon or capsule	0	0.0	0	0			
Wound penetrate to bone or joint	0	0.0	0	0.0	$X^2 = 1.6$	> 0.05	NS

Table (4): comparison of wound healing process after intervention in both study and control group

Items	Group				test	p-value	sig
	Study group (n=36)		Control group (n=36)				
	No.	%	No.	%			
Signs and symptoms of wound healing							
Non	0	0.0	4	11.1			
Slight	0	0.0	4	11.1			
Moderate	4	11.1	20	55.6			
Substantial	12	33.3	8	22.2			
Complete	20	55.6	0	0.0	Z= 6.2	<0.001	HS
Drainage resolution							
Non	0	0.0	0	0.0			
Slight	0	0.0	8	22.2			
Moderate	4	11.1	16	44.4			
Substantial	4	11.1	12	33.3			
Complete	28	77.8	0	0.0	Z= 6.4	<0.001	HS
Skin inflammation resolution							
Non	0	0.0	0	0.0			
Slight	0	0.0	4	11.1			
Moderate	4	11.1	12	33.3			
Substantial	12	33.3	20	55.6			
Complete	20	55.6	0	0.0	Z= 5.1	<0.001	HS

Table (5): Comparison of ulcer size and depth before and after intervention in both groups

Items	Group				test	p-value	sig
	Study group (n=36)		Control group (n=36)				
	No.	%	No.	%			
Ulcer area before intervention							
1 cm^2	4	11.1	4	11.1			
2 cm^2	16	44.4	8	22.2			
3 cm^2	4	11.1	0	0.0			
4 cm^2	4	11.1	16	4.4			
5 cm^2	0	0.0	0	0.0			
6 cm^2	8	22.2	8	22.2	Z= 1.94	>0.05	NS
Ulcer area after intervention							
0 cm^2	4	11.1	4	11.1			
1 cm^2	20	55.6	8	22.2			
2 cm^2	8	22.2	16	44.4			
3 cm^2	4	11.1	4	11.1			

4 cm^2	0	0.0	4	11.1	Z=2.37	<0.05	S
Ulcer depth before intervention							
1 cm^2	36	100	36	100			
>1 cm^2	0	0.0	0	0.0	0.00	1	NS
Ulcer depth after intervention							
Non	16	44.4	8	22.2			
1 cm^2	20	55.6	28	77.8	X^2=4	<0.05	S

Table (6): final wound outcome in study and control groups

Items	Group				test	p-value	sig
	Study group (n=36)		Control group (n=36)				
	No.	%	No.	%			
Decrease in ulcer area							
4 cm^2	4	11.1	0	0.0			
3 cm^2	8	22.2	4	11.1			
2 cm^2	0	0..0	20	55.6			
1 cm^2	24	66.7	12	33.3	Z=1.98	<0.05	S
Decrease in ulcer depth							
Non	20	55.6	28	77.8			
1 cm^2	16	44.4	8	22.2	X^2=4	<0.05	S
Final wound intervention							
Total closure without need for surgical intervention at besides or in OR	24	66.6	12	33.3			
Graft or flap closure required	12	33.3	24	66.7			
Amputation proximal to metatarsophalangeal joint	0	0.0	0	0.0			
Amputation distal to metatarsophalangeal joint	0	0.0	0	0.0	X^2 = 0.0	1	NS

4. Discussion:

Management of diabetic foot ulcers is becoming more challenging and controversial with increasing number of wound care products, protocols, and algorithms available. This difficulty is especially pronounced for health care professionals without specialized training in wound care. (Calhoun, etal.2002). When all the trials that used foot amputation as a primary outcome variable are considered, amputation was prevented in most of the diabetic patients treated with adjunctive hyperbaric oxygen therapy (Heyneman, & Lawless-Liday, 2002).

In order to make the comparisons between the two groups valid, their comparability regarding the factors that could affect wound healing was examined. They had similar age, gender, working status and past medical history, where there was no statistical significant difference between the two groups regarding their diabetic type, diabetes duration, affection by complications and previous affection with diabetic foot ulcer. The diabetic history exhibits that most of the studied subjects in both groups were affected previously by foot ulcer, findings similar to Iversen, etal., (2009), who mentioned that a history of previous diabetic foot ulceration increases the risk for new ulceration. The history also showed a highly significant difference between the two groups regarding treatment used as many of the control group patients exposed to surgical procedure, which may be explained by their poor previous diabetic control as evident by more than three third of the control group patients have previous uncontrolled diabetes, with a highly statistically significant difference between both groups.

Concerning the ulcer assessment, the results of the present study revealed also a non significant difference between the two groups regarding their peripheral tissue perfusion, ulcer complications, ulcer depth, ulcer degree, and ulcer area before intervention which support the similarities between the two groups.

Comparing the process of wound healing, the results of the present study revealed a highly significant difference between the study and control group regarding the signs of wound healing, resolution of wound drainage, and resolution of signs of wound inflammation, as well as more than half of the study group subjects experience complete healing compared to non of the control group subjects (Hypothesis I). Similar results were achieved by (Ong 2008, Abidia etal., 2003, Kalani etal. 2002) who reported that half of the HBOT group subjects have healed completely compared to less than forty percent of the control group. Kessler etal., (2003) also reported similar findings. These findings is also supported by the present study results concerning the size and depth of the ulcer before and after intervention in both group, the study results revealed

a non significant difference between the two groups regarding the ulcer size and depth before intervention, while revealed a significant difference between study and control groups after intervention. This is attributed to the adjunction of HBOT to the treatment plan of study group subjects. This effect is explained by Gill and Bell, (2004), Barnes, (2006), Cimşit etal. (2009), Lipsky, and Berendt (2010) who explained the experimentally demonstrated effects of HBOT on improving wound tissue hypoxia, enhancing perfusion, reducing edema, down regulating inflammatory cytokines, promoting fibroblast proliferation, collagen production, and angiogenesis make it a useful adjunct in clinical practice for "problem wounds," such as diabetic foot ulcers. HBOT is also touted for eradicating difficult to treat soft tissue and bone infections by mechanisms that include killing microorganisms, improving leukocyte and macrophage function, and enhancing the effect of antimicrobials. Ong (2008) was supporting this explanation when reported the role of oxygen in wound healing.

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The present study also showed a decrease in area and depth in both group compared to the pre intervention level, with a significant difference between the two groups. (Hypothesis II). This was in congruent with Zamboni etal., (1997) and Duzgun, etal., (2008), who reported significant greater reductions in wound size in HBOT group compared to the control, which support the idea that HBOT has a strong beneficial effect on wound healing in diabetic patients. Duzgun etal.,.(2008) added that HBOT is effective even in the presence of risk factors that are typically harmful, more patients healed their lower extremity wound. This was the case in the present study as 44.4% of the study group was not controlling their diabetes, in addition to 77.8% of them was affected by other diabetic complications, and 77.8% of them exhibit signs and symptoms of ischemia and 11.1% of them, their ulcers were infected

Insight on the physiologic explanations for these results is provided by Duzgun etal., (2008), who emphasized the effect of O_2 in wound healing. The rational for the adjunctive use of HBO_2 stems from its beneficial effects on the microenvironment of the wound. Although HBO_2 will not significantly increase O_2 saturation of hemoglobin, it can significantly increase the fraction of O_2 physically dissolved in plasma. Under hyperbaric conditions, wound tissue O_2 tension can be increased 10 to 15 fold. Elevation of O_2 tension in hypoxic wounds enhances neutrophil oxidative killing of bacteria and stimulates fibroblast proliferation, collagen production, revascularization, and epithelization; In addition, O_2 is directly toxic to anaerobic organism. Furthermore, HBOT has also been shown to have synergistic effects with aminoglycosides, trimethoprime, notrofurantoin, and sulfisoxazole, and hyperoxic vasoconstriction that takes place during HBOT reduces capillary pressure and trans capillary fluid transfer increases extra vascular fluid resorption, which reduces lower extremity edema.

Inspite of a non significant difference between study and control group in their final wound outcome, the present study showed that 66.6% of the study group subjects' ulcer closed without the need for surgical intervention at bedside or in operating room compared to 33.3% of control group subject, which support the adjunction of HBOT to the treatment plan of diabetic foot ulcer patients. Surprisingly, no one of the studied subjects were amputated. Zamboni etal.(1997) reported similar findings. These findings may be explained in the light of their wound characteristics, that both group were affected by grade I or II ulcer, their peripheral tissue perfusion was not compromised or mildly compromised, all ulcers were superficial. This explanation in congruence with Ong (2008) who reported that the patient with a relatively intact peripheral blood supply will respond better to HBOT.

Conclusion:

Foot ulcers are a significant source of mortality, and morbidity, and diminished quality of life for patient with diabetes. Hypoxia can cause trivial lesions to progress rapidly to infection, gangrene, and limb amputation. Hyperbaric oxygen therapy increases the amount of oxygen dissolved in plasma, allowing tissues to achieve level of oxygenation that would otherwise be impossible. HBOT is adjunctive treatment that could be added and will never replace good wound care.

Recommendations

The results of this study should provide impetus and basis for larger multicenter prospective, so it is recommended to increase the HBOT centers as there are only two centers in Nasser Health Institute and Air Force Military Hospital, only one of them is available for such kind of data.

Evidence-based guidelines that based on transcutaneous oximetery and ulcer grade classification should be used to aid the clinician in determining which patients are suitable candidate for HBOT. Measuring the level of oxygen dissolved in plasma (T_cPo_2) may be useful in selecting patients for adjunctive HBOT.

From socioeconomic perspective, this beneficial form of therapy is still expensive for a single patient as the complete course still very

expensive for individual patient, so it should be allowed for diabetic patient under the umbrella of health insurance.

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References

1. Abidia, A., Laden, G.. & Kuhan, G.. (2003): The role of hyperbaric oxygen therapy in ischemic diabetic lower extremity ulcers: a double-blind randomized-controlled trial. Eur J Vasc Endovasc Surg.; 25:513-518. In: M Ong (2008): Hyperbaric oxygen therapy in the management of diabetic lower limb wounds. Singapore Med J. 49(2):105-109.
2. Apelqvist, J., Bakker, K. & Houtum, W., Franssen H.& Schaper, N.(2000): International consensus and practical guidelines on the management and the prevention of the diabetic foot. International Working Group on the Diabetic Foot. Diabetes Metab 16(Supp 1):584–92.
3. Armstrong, D.G.., Lavery, L.A., Harkless, L.B. (1998): Validation of a diabetic wound classification system. The contribution of depth, infection, and ischemia to risk of amputation. Diabetes Care; 21(5):855-9.
4. Bakker, D.J. (2000): Hyperbaric oxygen therapy and the diabetic foot. Diabetes Metab Res Rev.16 (Suppl) 555-558.
5. Barnes, R.C. (2006): Hyperbaric oxygen is beneficial for diabetic foot wounds. Clin Infect Disease 43: 188– 192.
6. Berendt, A. R. (2006): Hyperbaric Oxygen Not Effective • CID: 43.
7. Boyko, E.J., Ahroni, J.H., Stensel, V., Forsberg, R.C., Davignon, D.R& Smith, D.G.. (1999): A prospective study of risk factors for diabetic foot ulcer: the Seattle Diabetic Foot Study. Diabetes Care. 22:1036-1042.
8. Broussard, C.L. (2004): Hyperbaric oxygenation and wound healing. J. Vasc Nurs. 22:42-48.
9. Calhoun, J.H., Overgaard, K.A., Stevens, C.M., Dowling, J.P. & Mader, J.T. (2002): Diabetic foot ulcers and infections: current concepts. Adv Skin Wound Care.15:31-45.
10. Cianci, P. (2004): Advances in the treatment of the diabetic foot: Is there a role for adjunctive hyperbaric oxygen therapy? Wound Repair Regen. (12):2-10.
11. Cianci, P. (2000): Consensus Development Conference on Diabetic Foot Wound Care: A randomized controlled trial does exist supporting use of adjunctive hyperbaric oxygen therapy. Diabetes Care; (23):873- 874.
12. Cimist, M., Uzun, G.&Yildiz S., (2009): Hyperbaric Oxygen Therapy for Diabetic Foot Wounds: Has hope hurdled hype?. Diabetes Care. 33 (5): 1143-1145.
13. Duzgun, A.P., Satir, H.Z., Ozozan, O., Saylam, B., Kulah, B., & Coskun, F. (2008): Effect of hyperbaric oxygen therapy on healing of diabetic foot ulcer. The Journal of Foot and Ankle Surgry.47 (6).
14. Fritschi, C. (2001): Preventive care of the diabetic foot. Nurs Clin North Am. 36:303-320.
15. Gill, A.L., Bell, C.N. (2004): Hyperbaric oxygen: its uses, mechanisms of action and outcomes. QJM; 97: 385– 395.
16. Heyneman, C.A. & Lawless- Liday, C., (2002): Using hyperbaric oxygen to treat diabetic foot ulcers: safety and effectiveness. Crit Care Nurse.22:52-60.
17. International Diabetes Federation, (2009): http://www.idf.org/
18. Iversen, M.M., Midthjell, K., Tell, G.S., Moum, T., Ostbye, T., Nortvedt, M.W., Uhlving, S. & Hanestd, B.R., (2009): The association between history of diabetic foot ulcer, perceived health and psychological distress: The NordTrondelag health study. BMC. Endoccrine Disorders, 9:18
19. Jin-ping, L., (2009): Hyperbaric oxygen in treating diabetic foot and corresponding nursing measures. Journal of Shandong medical College.
20. Johnson, M., Mass, M.& Moorhead, S. (2000): Nursing outcome classification. IOWA Outcome Project. (2nd ed). 431, 445-446.
21. Kalani, M., Jorneskog, G., Naderi, N., Lind, F.& Brismar, K. (2002):
22. Kessler, L., Bilbault, P.& Ortega, F., (2003): Hyperbaric oxygenation
23. Lipsky, B.A., Berendt, A.R. & Deery, G.H.(2004): Diagnosis and treatment of diabetic foot infections. Clin Infect Dis. 39:885–910.
24. Lipsky and Berendt (2010): Hyperbaric Oxygen Therapy for Diabetic Foot Wounds: Has hope hurdled hype? Diabetes Care 33 (5): 1143-1145
25. Miller (1998): Management of diabetic foot ulcers. J Cutan Med. Surg. 3 (suppl 1): 13-17-51.
26. Niinikoski, J., (2003): Hyperbaric oxygen therapy of diabetic foot ulcer, transcutaneous oxymetry in clinical decision making. Wound Repair Regen; 11:458-461.
27. Ong, M. (2008): Hyperbaric oxygen therapy in the management of diabetic lower limb wounds. Singapore Med J. 49(2):105-109.
28. Rakel, A., Houtm, C., Ekoe, J. (2006): Canadian diabetes association technical review: the diabetic

foot and hyperbaric oxygen therapy. Canadian Journal of Diabetes. 30(4):411-421.
29. Reiber, G.E., Boyko EJ& Smith, D.G. (1995): Lower extremity foot ulcers and amputations in diabetes. In: Harris, M.I., Cowie, C.C., Stern, M.P., Diabetes in America. (2nd ed). Washington, DC: US Government Printing Office; 409-428. Publication NIH 95-1468.
30. Reiber, G.E., Viberti, L. & Boyko, E.J. (1999): Causal pathway for incident of lower extremity ulcers in patients with diabetes from two setting. Diabetes Care.22:157-162.
31. Zamboni, W.A., Wong, H.P., Stephenson, L.L.& Pfeifer, M.A., (1997): Evaluation of Hyperbaric oxygen for diabetic wounds; A prospective study. Undrsea and Hyperbaric Medical Society.; 24(3):175-179.

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