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The Effect of Ultraviolet Radiation (Type B) on Decubitus Ulcers

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ABSTRACT

Pressure ulcers heal slowly and this often results in prolonged hospitalization. Wound infection delays healing in decubitus ulcers and standard treatment include wound dressing and use of antibiotics. However, there is increasing resistance of bacteria to antibiotics. The purpose of this study was to determine the effects of ultraviolet radiation (UVB) on the healing of decubitus ulcers. The study was also designed to learn the effect of UVR (Type B) on bacteria. **Methods:** Ten subjects with ascertained bilateral pressure sores (6 at the gluteal region and 4 at the heels) on the left lower extremities were recruited for this study. The left limbs (experimental limbs) were radiated with UVR (B) coupled with normal wound dressing while the right (control) limbs only received normal wound dressing for 6 weeks. The data were analyzed using descriptive and non-parametric inferential statistics (Kruska Wallis test). **Results**: There was 78.9% decrease in the mean surface area of the decubitus ulcers of the experimental (left) limbs while there was only 37.4% decrease in the mean surface area of the decubitus ulcers of the control (right) limbs. Similarly, there was 74.7% decrease in the mean volume of the decubitus ulcers of the experimental (left) limbs while there was only 46.3% decrease in the mean volume of decubitus ulcers of the control (right) limbs. The result of the Kruska Wallis test showed that there was significant decrease in the growth of bacteria ($X^2 = 37.01$, $P<0.00$) and significant increase in the growth of epithelial cells ($X^2 = 36.65$, $P< 0.00$) in the decubitus ulcers that were irradiated with UVR. **Conclusion**: It was concluded that ultraviolet radiation (Type B) had significant effects in destroying bacteria and also promoting wound healing.

INTRODUCTION

A decubitus ulcer is an injury to the skin that results from pressure against the skin.[1] Constant pressure on an area of skin reduces blood supply and this eventually causes cell death, breakdown of skin, and development of an open sore.[1, 2] Tissues are capable of withstanding enormous pressures when brief in duration, but prolonged exposure to pressures even slightly above capillary filling pressure initiates ulceration. Patients who are neurologically impaired, heavily sedated, restrained, or demented are incapable of assuming the responsibility of altering their position to relieve pressure.[2]

Pressure sores occur in approximately 9% of hospitalized patients and this is usually during the first 2 weeks of hospitalization. Also, approximately one fourth of nursing home residents sustain pressure sores.[3] The yearly risk of pressure ulceration in patients with neurological impairment (such as the spinal cord injured patient) is 5 – 8%, with a lifetime risk of approximately 85% and mortality rate of 8%.[3]

The best way to treat pressure sores is to prevent their formation, but prevention of pressures sores has continued to pose challenges to professional nursing practice in both developed and developing countries.[4] Pressure sores are inevitable in developing countries, such as Nigeria, because most teaching and non-teaching hospitals manage bedridden patients without adequate and modern medical technological devices such as water mattresses designed to avoid undue pressure on the skin.[4]

Surgical closure of wounds results in a 38% complication rate, a mean hospitalization period of 40 days, a 26% incidence of unhealed ulcers at discharge and a 79% incidence of ulcer recurrence at the site of the healed flap within a mean of 10.9 months.[5] The treatment of pressure ulcers requires prolonged surgical and nursing care (14 -120 days).[6] During the extended period of treatment required, the patients remain at risk of developing new pressure ulcer at other sites[6].

There are a number of factors that influence the healing of decubitus ulcers. These include malnutrition, underlying medical conditions (such as diabetes mellitus), drugs, presence of residual necrotic tissue in the wound bed, hypoxia, presence of another wound, low temperature, and wound infection.[7] Wound infection is the deposition and multiplication of bacteria in tissues with an associated host reaction.[8] Wound infection is a growing problem and most cases are hospital acquired. It is often associated with extended lengths of stays in the hospital. A Nigerian University teaching hospital reported that virtually all wounds of patients in the hospital at the time of this study were infected with bacteria, with eighty percent (80%) of the wounds infected with Staphylococcus aureus.[10]

Surgical management has been known to sometimes result in complications.[11] There appears to be increasing resistance to both topical and systemic antibiotics for infected wounds. In most situations, endogenous bacteria predominate and many are potentially pathogenic in the wound environment.[5, 12]

Systemic antibiotics are generally accepted as being the preferred choice for treating infection, provided that ischemia does not interfere. However, the widespread use of systemic antibiotics is leading to the emergence of resistant bacterial strains such as methicillin-resistant Staphylococcus aureus (MRSA).[13] A systematic review of antimicrobials carried out by O' Meare revealed that antibiotics with wound dressing are not generally effective for management of chronic wound infection.[14] Resistance of bacteria to antibiotics has become a serious problem in recent years.[15] As result of prevalence of slow healing of decubitus ulcer, physiotherapy has played a major role in wound care and management. Therapeutic modalities such as iontophoresis, laser therapy, short wave diathermy, ultrasound, hydrotherapy, infra red radiation and ultraviolet radiation are now being utilized.[16]

It has been found that radiation from ultraviolet radiation (Type C) is universally successful in destroying bacteria.[17] But there is a dearth of empirical data on the use of ultraviolet radiation type B (UVB) in effective destruction of bacteria. The objectives of this study were to determine the effects of ultraviolet radiation (Type B) in the healing of decubitus ulcers and also to determine its effect on bacteria and epithelialization.

METHODOLOGY

Subjects

The population consisted of purposively selected 10 bed ridden subjects (8 females and 2 males) who consented to participate in the study. Six (6) of the subjects were diagnosed to have spinal cord injuries, 2 with head injuries, 1 each with sickle cell anaemia and Pott's disease. Six of the subjects have gluteal sores while 4 sustained the pressure sores on the heels of the left lower limbs. They were all receiving treatment on admission at the Obafemi Awolowo University Teaching Hospital Complex (OAUTHC) Ile-Ife, Osun State Nigeria at the time of this study and have been bedridden for 3 at least months.

Inclusion criteria included absence of previous skin breakdown or wound prior to being admitted, presence of bilateral pressure sores on the lower limbs; a stable regimen of medications during the course of the study including the antibiotic ciproflaxin; a wound duration of at least 8 weeks; and age between 35 – 55 years. Patients with diabetes, malnutrition, dermatitis, or with metallic implants were excluded from this study. The instruments used for this study were fluorescent tube ultraviolet radiation lamp, sterilized cellophane paper, marker, graph paper, swab stick, towel, Vaseline, cotton wool, and Ringer's solution. Other instruments used were syringe, stopwatch, Petri dish and Agar solution ,which was used as liquid medium to culture bacteria.

Procedures

The decubitus ulcers on the left lower limbs served as the experimental limbs and were treated using both ultraviolet radiation and traditional saline-wet-to-moist (WM) wound dressing. The right lower limbs served as the control limbs and were treated only with traditional saline-wet-to-moist (WM) wound dressing.

The source of ultraviolet radiation was a fluorescent tube ultraviolet radiation lamp (UVB). The dosage (distance and exposure duration) used for the study was based on specifications in the literature of the ultraviolet radiation apparatus (Philips 8P3114)—3 inches of distance between the fluorescent lamps and the wound, and a progressively increasing exposure duration with each session (¾, 1, 2, 2½, 3, 4, and 5 minutes for the first seven sessions, respectively). The wounds were radiated once every three days for 6 weeks.

The skin surrounding the wound on the experimental limbs was protected from undesired exposure to ultraviolet radiation by applying 2mm thick layers of Vaseline and cotton wool to the wound edges. Surrounding skin was covered with thick fold of towels. The surface area of each decubitus ulcer (both experimental and control limb) was determined using the planimetric method.[16].The cellophane sheets used for the study were first washed in a disinfectant and dried. The sheets were then sterilized by exposing both surfaces of each sheet to high intensity ultra-violet radiation (B) rays for ten minutes at a distance of 50cm.[16]

First, a sheet was spread over each decubitus ulcer. A second sheet was placed on top of the first sheet and the surface of each of the wounds was traced with a marker. The sheets that were in contact with the decubitus ulcers were discarded while the upper sheets were kept as a record of the surface area of the decubitus ulcers. The outline traced on the sterilized cellophane sheet was transferred to metric graph paper. The area under the traced sterilized cellophane sheet was estimated by counting the number of small squares enclosed within the tracing. The surface areas of the wounds were calculated with the following formula:

Wound surface area (Cm2) = Number of squares within the tracing X 0.0625 (cm^2).[18]

Decubitus ulcers were traced on a weekly basis, and either a reduction or increase in the surface area of the ulcers was computed in percentage. Volumetric measurements of the depth of decubitus ulcers (both experimental and control limbs) were calculated using Ringer's solution with syringe.[19] The base of each wound was covered with foil and filled with Ringer's solution using a syringe. The volume of Ringer's solution required to fill each wound represented the volume of the wound. Volumetric measurements of pressure sores were made on a weekly basis.

The epithelialization of the decubitus ulcers (both experimental and control limbs) was assessed by the Pressure Sore Status Tool.[20] The assessment of the epithelialization was based on a Likert scale of 5. The main researcher graded the epithelialization:

1 - 100% wound covered and surface intact.
2 - 75% to less than 100% of wound covered or epithelial tissues extends greater than 0.5 cm into wound bed.
3 - 50% to less than 75% of the wounds covered with epithelial tissues.
4 - 25% to less than 50% of wound covered
5 - Less than 25% of wound covered.

The epithelialization of decubitus ulcers was assessed on a weekly basis during the study. Swabs of the experimental limbs of the decubitus ulcers were taken at every session of treatment, before and after the wound's exposure to ultraviolet radiation. The swabs of the control limbs were taken once at every session of treatment. The swabs were subjected to medical laboratory analyses. The bacteria in the swabs were cultured on nutrient agar (0.1% of sucrose solution) in a Petri dish .The nutrient agar smeared with the swabs taken from the decubitus ulcers and the Petri dish this were covered for 24 hours. *Staphyloccus aureus*, *Klebsiella species*, *Proteus vulgaris* and *Pseudomonas aureginosa* were isolated in the culture. The bacteria growth was rated on a Likert scale of 5:

1. No growth
2. Slight growth
3. Moderate growth
4. Heavy growth

5. Very Heavy Growth.

Ten (10) laboratory scientists were grouped into 2 (5 in each group) and were blinded to the purpose of grading the bacteria growth to determine the reliability of the Likert scale. The scientists in the first group were asked to grade the bacteria growth one after the other using the scale for a purposively selected wound. The 2nd group was also asked to rate the bacteria growth on the selected wound. The result showed that there was no significant difference ($p>0.05$) in the rating of bacterial growth between the 2 groups. Similarly, there was significant correlation between the rating of the 2 groups ($r= 0.97$). This showed that the scale is reliable. The body parts that were not to be exposed to ultraviolet radiation were properly screened from the UV rays. Both the researcher and the patients used protective goggles.

Statistical Analysis

The data were analyzed using descriptive and non- parametric inferential statistics (Kruska-Wallis test). The level of significance was set at 0.05.

RESULTS

The mean age of the participants was 45.3 ± 18.3 years. For the experimental (left) limbs, the initial (pre-experimental) mean surface area of the ulcers was 76.5 ± 63.7 cm² while the final (post-experimental) mean was 16.6 ± 15.2 cm². Similarly, for the control (right) limbs, the initial mean surface area of the ulcers was 43.8 ± 32.0 cm² and the final mean surface area was 27.4 ±19.7 cm². There was 78.9% decrease in the surface area of the ulcers of the experimental (left) limbs, while there was only 37.4% decrease in the surface area of the ulcers of the control (right) limbs (Table1).

The initial mean volume of the ulcers on the experimental (left) limbs was 34.9 ± 34.2 ml and the final mean volume was 8.2 ± 9.2ml. The initial mean volume of the ulcers on the control (right) limbs was 26.1 ± 25.5 ml and the final mean volume was 14.0 ± 14.3 ml. Thus, there was a 74.7% decrease in the volume of the ulcers on the experimental (left) limbs, but only a 46.3% decrease in the volume of ulcers on the control (right) limbs (Table 2).

The initial mean growth of bacteria on the experimental (left) limbs was 3.90 ± 0.32 on a Likert scale of 5, while the final mean growth was 0.10 ± 0.32. The initial mean growth of bacteria on the control (right) limbs was 4.0 ± 0.00 on a 5-point Likert scale while the final mean growth of bacteria of the control (right) limbs was 2.50 ± 0.52. The result of the Kruska-Wallis test showed that there was a significant difference ($X^2 = 37.01$, $P<0.00$) in the final mean growth of bacteria in the ulcers between the experimental (left) and control (right) limbs (Table 3).

Table 1: SURFACE AREA (CM^2) OF THE DECUBITUS ULCERS.

Groups	Wound State	Mean Ulcer Surface Area (cm^2)	SD	Change %
Experimental Limbs	Initial Final	76.5 16.6	63.7 15.2	 .78.9
Control Limbs	Initial Final	43.8 27.4	32.0 19.7	 -37.4

Table 2: THE VOLUME OF DECUBITUS ULCERS (Milliliter)

Groups	Wound State	Mean Ulcer Surface Volume (ml)	SD	Change %
Experimental Limbs	Initial Final	34.9 8.2	34.2 9.2	 -74.7
Control Limbs	Initial Final	26.1 14.0	25.5 14.3	 -46.3

Table 3: COMPARISON OF THE GROWTH OF BACTERIA ON THE LIKERT SCALE

Groups	Wound State	Mean Growth of Bacteria	SD	Mean Rank	X2	P value
Experimental	Initial Final	3.90 0.10	0.32 0.32	29.75 5.50		
Control	Initial Final	4.00 2.50	0.00 0.52	31.00 15.75	37.01	0.00

For the experimental (left) limbs, initial mean epithelial growth of the ulcers was 5.00 ± 0.00 on the Likert scale of 5, while the final mean epithelial growth was 1.90 ± 0.32. On the control (right) limbs, the initial mean epithelial growth in the ulcers was 5.00 ± 0.00, while the final mean epithelial growth was 3.6 ± 0.67. The Kruska-Wallis test showed that there was significant difference ($X^2 = 36.65$, $P < 0.00$) in the epithelial growth between the experimental (left) limbs and control (right) limbs of the ulcers (Table 4).

Table 4: COMPARISON OF THE EPITHELIZATION OF THE DECUBITUS ULCERS ON THE LIKERT SCALE

Groups	Wound State	Mean Growth of Bacteria	SD	Mean Rank	X2	P value
Experimental	Initial Final	5.00 1.90	0.00 0.32	30.00 5.50		
Control	Initial Final	5.00 3.60	0.00 0.67	30.00 16.50	36.65	0.00

DISCUSSION

The aim of ultraviolet radiation is to destroy bacteria, remove slough, and promote repair.[21] This occurs by helping to increase epithelial cell turnover, epidermal cell hyperplasia, and increasing DNA synthesis. Ultraviolet radiation is bacteriocidal in that it has an effect on the DNA of the bacteria. Ultraviolet radiation mostly damages DNA by producing thymine dimers, which are cross-links between adjacent pyrimidine bases in a DNA strand. Short-wavelength UV light has enough energy to damage chemical bonds in DNA molecules, which are very stable under most conditions.

The UV light can cause adjacent pyrimidine bases in DNA (thymine and cytosine), to bond to one another instead of the complementary DNA strand. This disrupts DNA replication. Ultraviolet photons harm the DNA molecules of living organisms in different ways. In one common damage event, adjacent thymine bases bond with each other, instead of across the "ladder." This makes a bulge, and the distorted DNA molecules does not function properly.[22]

Compared to control ulcers, there was a significantly greater reduction in both the surface area and the volume of the ulcers subjected to six weeks of UVB radiation. Our study suggests that UVB radiation is effective in decreasing ulcer size, and lends support to the work of Nussabaum who concluded that ultraviolet radiation can accelerate reduction in the size of pressure ulcers.[5]

Furthermore, compared to control ulcers there was a significantly greater growth of epithelium in the ulcers subjected to UV radiation for six weeks. This supported the study of Thai, who concluded that ultraviolet radiation increases epithelial cell proliferation in pressure ulcers, thus giving the wound surface its typical red-bubbled appearance.[23] This study showed that there was significant reduction of bacterial growth in the UVB irradiated wounds compared to controls. This showed that ultraviolet radiation was greatly effective in killing bacteria in pressure sores. This supported the study of Ruiz who reported that ultraviolet radiation could destroy up to 100% microorganisms present in a wound.[9]

Adedoyin et al. highlighted the importance of physiotherapy in wound care, including such physical agents as electrical stimulation, ultrasound, laser therapy, iontophoresis, infrared and ultraviolet radiation.[16] A pilot study investigating the management of pressure ulcers by South African physiotherapists, however, has revealed that most of them do not use these physical agents for a variety of reasons.[24] This study further highlights the efficacy of physiotherapy in wound care. UV light is

one of the oldest physiotherapeutic modalities available to therapists especially in developing countries. Physiotherapists are advised to consider the adjunctive use of this modality and others in the holistic management of pressure ulcers.[24]

CONCLUSION

This study has demonstrated that ultraviolet radiation is an effective modality for treating pressure ulcers. Ultra Violet Radiation (Type B) was found effective in reducing wound volume and surface area. Also, it increased process of wound epithelization and killing of bacteria in study wounds. The sample size in this study was small, and the grade or stages of the decubitus ulcers were not described. It is recommended that future studies of this nature should use larger samples and compare different grades of pressure ulcers to further validate these finding.

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