

Buruli Ulcer: Its Impact and Treatment Worldwide: An Interval Report

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Buruli ulcer, a devastating disease first described in 1897 by Sir Albert Cook in Uganda, Africa, is caused by *Mycobacterium ulcerans* and is seen in more than 30 mostly under-resourced, countries worldwide.¹ More than 70% of the patients affected are children younger than 16,² with 90% of the ulcers manifested on the limbs.¹ The ulceration caused by the microorganism is painless due to the cytotoxic and immunosuppressive properties of the bacterial toxin mycolactone. Development of these ulcers is accompanied by marked edema of the affected extremity, and up to 15% of the skin surface can be involved in the ulcerative process.³

Unfortunately, these ulcerative lesions can become very large before treatment is sought because of lack of access to care, lack of funds, superstitious beliefs about the disease, and the stigma of the disease.⁴ Current treatment entails administration of two antibiotics (rifampin and streptomycin) for 8 weeks, followed by excision of the ulcerated area and skin grafting if the ulcer does not show signs of healing by secondary intention. Complications of the disease and its treatment can be seen in up to 24.5% of patients and can include amputation, joint contractures, and death.²

Historically, care for Buruli ulcers during the 8 weeks of antibiotic therapy

has been left to the standards of each healthcare facility treating a patient. This wound care would usually involve washing the wound with water and/or acetic acid and applying Betadine-soaked gauze dressings. In addition, no formal debridement would be done, and no attempt would be made to address the edema of the extremity. If these wounds remained unhealed after 8 weeks of antibiotic therapy, they would be excised and treated with split-thickness skin grafting. In one series, this treatment required an average of 1.45 operations per patient, and 44% of the patients required blood transfusion at the time of operation.²

In an attempt to improve the healing of this devastating disease and to avoid some of the longstanding complications, a clinical trial using good, basic wound care techniques, dressings, and compression therapy in conjunction with antibiotic therapy has been instituted in Ghana, Africa, under the auspices of the World Health Organization.

Methods and Materials

The goal to recruit and treat 20 patients has been undertaken. All patients are treated with rifampin and streptomycin for 8 weeks. Due to the need to provide moist wound healing and treatment of the edema, each patient

is treated with Vaseline gauze, Drawtex hydroconductive dressings, and short-stretch compression therapy. The Drawtex dressing is used because of its superior wicking action, which moves wound fluid away from the wound surface, facilitating autolytic debridement of the wound. Short-stretch compression bandages are used to reduce the marked edema seen in the extremities of patients with this disease. Clinic personnel change dressings three times per week. Wounds are measured, photographed, and evaluated weekly for 8 weeks.

Results

To date, eight of the 20 projected patients have completed the study. Improvement in the wound bed was noted in all patients (**Table 1**). The amounts of granulation tissue in the wound beds improved from 25% to 75% in two patients and from 25% to 100% in six patients. These improvements in the granulation tissue occurred along with reductions of necrotic tissue and slough in the wound beds and through autolytic debridement facilitated by the Drawtex hydroconductive dressing.

Drainage from these large wounds is always a problem. Treatment with the Drawtex hydroconductive dressing had the following results: one patient's

Table 1. Results of Buruli ulcer therapy with Drawtex and short-stretch compression therapy.

Change in % Granulation Tissue	< 25% to 75%: 2	< 25% to 100%: 6	
Change in Drainage	Large to Medium: 1	Large to Minimal: 4	Unchanged: 3
Change in Wound Size	Increased: 3	Decreased: 5	

wound drainage decreased from “large” to “medium”; four patients’ drainages decreased from “large” to “minimal”; three patients’ drainages remained unchanged despite improvements in wound bed granulation tissue responses.

Over the 8-week period of the evaluation, five of the wounds decreased in size, some dramatically (**Figures 1 and 2**). Three of the wounds increased in size during the evaluation period. This is not entirely unexpected, as many wounds have large amounts of necrotic tissue and will enlarge significantly once this tissue is debrided. Seven of the eight patients had a significant improvement in the edema of the extremity during the treatment. Although none of the wounds healed during the evaluation study, three were deemed ready for split-thickness skin grafting before the 8 weeks of the antibiotic therapy were over.

Conclusion

Interim evaluation of eight patients in this Buruli ulcer treatment trial imply that treating these wounds with the hydroconductive dressing, Drawtex, and short-stretch compression bandaging improves the wound bed and facilitates healing before the end of the 8-week antibiotic treatment phase. The majority of the treated patients had a reduction in necrotic tissue and wound slough by autolytic debridement; a reduction in wound drainage; an increase in the granulation tissue in the wound bed; and a reduction in size of the ulcers. The goal of improving the wounds and readying the wounds for split-thickness skin grafting before the end of the 8 weeks of antibiotic therapy seems to have been achieved with this therapy. The results certainly are encouraging enough to recommend continuing the trial until all 20 patients have been enrolled and treated. ■

References

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Figure 1. Buruli ulcer of foot at beginning of evaluation study.



Figure 2. Buruli ulcer of foot after 8 weeks' treatment.