



A Review Article Detailing the Clinical Features, Epidemiology and Management of Buruli Ulcer Infection

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Review Article

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Abstract

Background: Buruli ulcer (BU) is a neglected tropical disease caused by *Mycobacterium ulcerans* and characterized by progressive destruction of skin and soft tissue. Although the disease occurs in several geographic regions, including Africa, Australia, and Japan, its true incidence may be underestimated because of limited access to allopathic healthcare, underreporting, and the predominance of cases in rural and underserved populations.

Pathogenesis and Clinical Manifestations: The major pathogenic factor in BU is mycolactone, a polyketide-derived macrolide produced by *M. ulcerans*. Mycolactone produces cytotoxic, immunosuppressive, and analgesic effects through multiple cellular mechanisms, including inhibition of Sec61-mediated protein translocation and disruption of mTOR-dependent cytoskeletal remodeling. It also contributes to sensory loss through neuronal hyperpolarization and promotes apoptosis through increased expression of pro-apoptotic proteins. Clinically, BU typically begins as a painless nodule, plaque, or edematous lesion and may progress to extensive ulceration with characteristic undermined edges. Approximately 55% of lesions occur on the lower extremities, 35% on the upper extremities, and 10% elsewhere. Although some early lesions may heal spontaneously, healing is often prolonged and may result in scarring, contractures, and functional disability, particularly when lesions involve areas near joints.

Diagnosis and Treatment: Laboratory confirmation is important and can be achieved through polymerase chain reaction, acid-fast bacillary staining, mycobacterial culture, and histopathology. Treatment depends on disease severity. Category I disease may respond to antibiotic therapy alone, whereas Categories II and III generally require antibiotic therapy combined with wound management and, in selected cases, surgical debridement. Despite the role of surgery in advanced disease, standardized and clearly delineated guidelines for surgical debridement remain limited.

Conclusion: Buruli ulcer remains an important but underrecognized infectious disease with substantial potential for tissue destruction and long-term disability. Improved surveillance, early diagnosis, access to appropriate antimicrobial therapy, and standardized approaches to surgical management are needed to reduce morbidity and improve functional outcomes.

Keywords: Buruli Ulcer, Epidemiology, Clinical Features and Treatment

Introduction

Buruli Ulcer (BU) is an uncommon yet debilitating infection caused by *Mycobacterium ulcerans*. This pathogen's impact is particularly profound in regions with limited access to allopathic healthcare, where its underrepresentation creates challenges in fully understanding its quantifiable effects. This infection, characterized by its rapid and debilitating clinical course, is prevalent in diverse geographical areas, spanning Africa, Australia, and Japan. Although there's a suggestion of decreasing BU incidence, various confounding factors such as underreporting, insufficient allopathic medical access and rural environments skew the reported numbers and inaccurately reduce the overall case count.

Given the limited research on BU, patients often dismiss initial lesions as benign, only recognizing their severity once significant body surface areas are affected, or soft tissues and bones are compromised. The lack of knowledge regarding potential vectors, clinical presentations, and genetic lineages places healthcare providers and patients at a disadvantage in effectively encountering and treating this disease. Addressing this challenge requires robust public health efforts to encourage comprehensive case reporting and timely treatment of this overlooked tropical ailment, especially concerning its impact on younger populations.

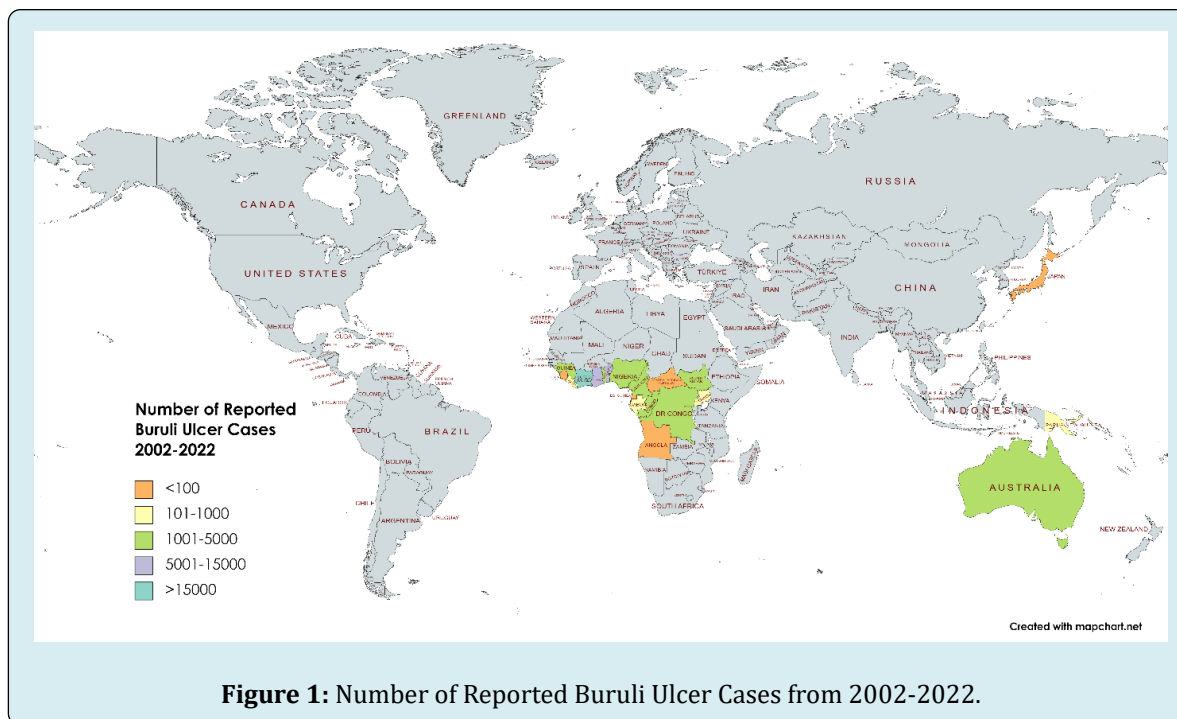
Epidemiology

Initially known as the "Bransdale ulcer," BU was first

identified in Australia during the late 1930s [1]. The genetic similarity between the African and Australian variants, both aligned with the Classical lineage, highlights their commonality. Africa shoulders the heaviest disease burden, accounting for over 95% of reported cases. High incidence rates are concentrated in countries such as Benin, Cote d'Ivoire, and Ghana, where BU occasionally surpasses even tuberculosis in prevalence [2]. Geographical distribution patterns show the Australian variant predominantly along coastal regions, while the African variant primarily affects rural communities—underscoring the interplay between isolation and interconnectedness through trade in disease transmission.

The Japanese variant diverges the most from the Classical lineage, reporting at least 60 cases since 1982. Notably, its distinct mycolic acid composition and biochemical characteristics categorize it as a separate subspecies [3].

Endemic to various tropical regions, BU's demographics and clinical data differ based on ancestral lineage (Americas and Asia) or classical lineage (Australia and Africa). Ancestral lineage BU exhibits no gender preference and affects middle-aged adults more frequently. In contrast, the classical lineage displays a bimodal distribution, with an age range of 42-61 in Australia/Oceania and a younger range of 5-15 in Africa [4]. The evolutionary trajectory of *M. ulcerans*, stemming from *M. marinum*, underscores its association with aquatic environments Figure 1.



Environmental factors significantly influence BU incidence. In African communities, deforestation and the construction of artificial dams correlate with increased cases [5]. Observations link BU incidences with ecological changes such as deforestation, lake flooding, eutrophication, irrigation system creation, and proximity to bodies of water [6]. These factors often result in topographic shifts that contribute to the formation of stagnant water bodies [7]. Climate plays a role, too, with an uptick in cases following dry periods after heavy rainfall. This could be attributed to the creation of small bodies of water post-rainfall, potentially acting as *M. ulcerans* reservoirs [8].

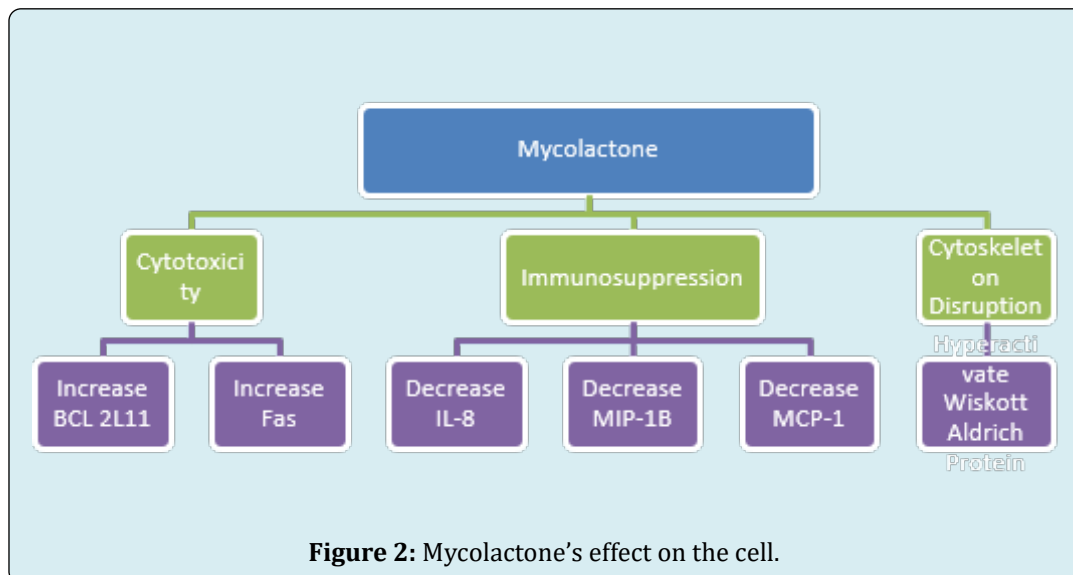
Though the precise mode of transmission remains uncertain, contaminated water exposure and mosquito vectors are proposed mechanisms. Five transmission theories include transmission through amoebae, aquatic invertebrates, mosquitoes, marsupials, and mammals, and the less likely human-to-human transmission. Amoebae and marine invertebrates have been identified as potential reservoirs, while mosquitoes have shown involvement in BU transmission, marked by the decrease in BU cases following insect repellent usage. The role of mosquitoes is further evidenced by the correlation between incidences of Ross River Virus and Buruli ulcer, sharing a proposed vector (*Ae.*

Camptorhynchus) and geographical regions in southeastern Australia [9]. Transmission theories encompass passive transmission via intermediate and high-level trophic organisms, airborne transmission, environmental transmission, and vector-borne transmission.

Microbiology and Pathogenesis

BU stems from *Mycobacterium ulcerans*, yet mycolactone, a polyketide-derived macrolide synthesized by the bacteria, is the true instigator of clinical symptoms, inducing cytotoxicity, immunosuppression, and analgesic effects [10]. Mycolactone has a multifaceted impact on the cell, encompassing the inhibition of Sec61—a pivotal player in creating receptors, cytokines, and antigen-presenting molecules—and mTOR activity, preventing dynamic cytoskeleton remodeling while inducing hypoesthesia through neuron hyperpolarization.

The increased expression of pro-apoptotic proteins BCL 2L11 and Fas heightens mycolactone-driven cytotoxicity. Additionally, mycolactone disrupts pathways like cytoskeleton dynamics by hyperactivating the Wiskott-Aldrich syndrome protein (National Center for Biotechnology Information) [11] Figure 2.



Strains related to *M. ulcerans*, such as *M. pseudoshottsii*, *M. liflandii*, and *M. marinum*, also secrete mycolactone. Phylogenetic analysis reveals polymorphisms within *M. ulcerans* strains, accounting for the divergence from the *M. marinum* progenitor into two distinct lineages.

Understanding *M. ulcerans* involves exploring its immunology. The infection initiates when *M. ulcerans* penetrates an already breached epidermis, as keratinocytes generate bactericidal reactive oxygen species and

chemokines. Within subcutaneous tissue, mycolactone disrupts coagulation cascade factors, forming a necrotic area surrounded by neutrophils, macrophages, and lymphocytes [12].

Mycolactone's influence is pivotal in shaping disease severity. By affecting phagocytosis, inhibiting pro-inflammatory cytokine production, and impeding nitric oxide species, mycolactone skillfully evades immune system responses. *M. ulcerans*'s immune response defects as the

disease progresses are evident in reduced IL-8, MIP-1B, and MCP-1 production during various disease stages [10].

Mycolactone's intense in vitro scrutiny contrasts with the lack of field studies. Consequently, hypothesizing its functions within a human host remains a subject of speculation. Further investigations are essential to unlock its potential roles once it enters a human organism.

Clinical Manifestations:

Since Buruli ulcer progresses quickly and has a poor prognosis, understanding the early stages of the clinical vignette of BU can change clinical outcomes and prognosis greatly. Buruli ulcer disease initially presents as a painless nodule; however, it can also present as a plaque or edematous lesion. 55% of lesions are seen in the lower extremities, with 35% in the upper extremities and 10% comprising the rest of the body [13]. Typically, the lesions are characterized by undermined edges. Up to one third of early nodules will heal spontaneously, however, the healing process is lengthy and can cause functional disabilities through contractures especially with lesions located near joint. On the other hand, the majority of the early stage lesions have the potential to ulcerate within 4 weeks if not treated with antibiotics [14].

When it comes to physical exam, early lesions are characterized by undermined edges. When comparing early lesions, edematous lesions have an increased likelihood of ulcerating and having systemic symptoms such as low-grade fever and pain (Vincent, Zingue)[15,6]. In later stages, lesions deepen and eventually causing debilitating sequelae such as osteomyelitis, seen in 15% of cases [15].

Since lesions look very different dependent on clinical course, the World Health Organization classifies lesions into three categories [16].

Category I: single lesions (nodules, plaques and ulcers) less than 5 cm in diameter.

Category II: plaques and ulcers between 5-15 cm in diameter; edematous lesions.

Category III: plaques and ulcers greater than 15 cm in diameter; osteomyelitis or joint involvement.

Differential: (Guarner, UpToDate, Aseidu, and WHO)

Buruli ulcer has a comprehensive clinical picture, as a result, it is a disease that should be considered in any abnormal skin exam as its differential is broad. Since ulcers can have ulcerative, nodular, plaque-like, and edematous properties, it's essential to keep such an underreported disease on a clinician's radar.

Buruli ulcer ulcerative lesion differentials include infectious, arterial/venous insufficiency and inflammatory conditions. Tropical ulcers which formed from a necrotic process caused by anaerobic bacteria; These ulcer edges are not usually raised or undermined as seen in Buruli ulcer.

Yaws ulcers caused by *T. pallidum pertenue*. Lesions begin as papillomas that ulcerates and heals without scarring. Can progress to form on the face, legs, arms, or the anogenital region without treatment.

Cutaneous leishmaniasis begin as a papulonodular lesion that then ulcerates; these lesions are painless, chronic and tend to have raised borders. Pyoderma gangrenosum ulcers are usually seen in the lower extremities and trunk. They start as tender papules that then expand peripherally, leading to ulceration. This is an inflammatory disorder of the skin.

Eumycetoma are subcutaneous mycotic infections; these lesions begin as nodules that develop into tumors and form necrotic abscesses with draining sinus tracts. Feet are usually involved first, then legs and hands. Common in tropical and subtropical regions. Venous stasis ulcers are formed from chronic venous insufficiency between the knee and ankle and usually present with inflammatory changes (stasis dermatitis). These are typically seen at the malleoli. Pain tends to be moderate to minimal.

Diabetic Foot Ulcers Result from Neuropathy and Vascular Insufficiency.

The differential diagnosis of Buruli ulcer nonulcerative lesions (nodules, plaques and edema) includes tumors, infections, bites and inflammatory conditions:

Nodular Lesions

- Squamous cell carcinoma is a malignant keratinocyte tumor that can present as erythematous papules, plaques, or nodules that can then ulcerate.
- Tuberculous lymphadenitis is a form of extrapulmonary tuberculosis. Typically presents as nontender cervical lymphadenopathy. Systemic symptoms are not common
- Onchocerciasis, known as "river blindness", causes blindness and skin disease, can often present with deep subcutaneous nodules, usually over bony prominences
- Lipomas are benign soft-tissue tumors. These are rubbery and nonpainful, mostly on the head, neck, back and shoulders.
- Epidermoid cysts are benign, skin-colored cutaneous nodules, usually with a central punctum. Found mostly on the head, neck, trunk or genitals.

Plaque-like Lesions

Kaposi sarcoma are endothelial cell tumors that usually present as papules and nodules as well as dark plaques on the skin. These lesions are usually painless and nonpruritic and associated with HIV/AIDS, caused by HHV-8. Because HIV infection complicates medical management, the WHO published guidelines to aid health professionals further.

Atopic dermatitis is a chronic inflammatory skin condition. Causes pruritis and can form plaque-like lesions on flexor surfaces. Buruli ulcer lesions are more common on extensor surfaces and tend not to be pruritic.

Edematous Lesions

Cellulitis is a manifestation of skin and soft tissue infections. Can be painful and associated with fever which is not typical of Buruli ulcer edematous lesions. Lymphatic filariasis Roundworm infection (usually *Wuchereria bancrofti*) causing lymphatic obstruction leading to painful lymphadenopathy. Disease process is more chronic than that of Buruli ulcer.

While clinically, skin condition can have a wide variety of differential; it is important for clinicians to have a broad differential and to include Buruli ulcer on said differential in order to prevent morbidity and mortality in extreme cases.

Diagnosis: (Guarner, Rondhini, Uptodate, Aseidu, WHO, Roltgen, Portaels, Sakyi)

While it can be clinically diagnosed, laboratory confirmation is often required for diagnosis. The standard for Buruli ulcer confirmation can be achieved by four laboratory modalities: polymerase chain reaction, acid-fast staining, culture, and histology.

Polymerase chain reaction (PCR)— This technique uses insertion sequence 2404 of the *M. ulcerans* genome. PCR has high sensitivity, with a reported 70 to 80 percent sensitivity in most series [17]. PCR is typically achieved with surgical or punch biopsy specimens but can also be achieved with swabs from the edges of undermined ulcers. Because of limited resources in developing countries, the WHO formed the Buruli ulcer Laboratory Network in Africa so as to increase PCR confirmations [18].

Histopathology— This modality requires considerable training and lab sophistication to perform. However, it has high sensitivity, up to 90 percent, and can help in monitoring response to treatment. Early disease characteristics include many acid-fast bacilli, epidermal hyperplasia, inflammation, hemorrhage and necrosis of subcutaneous tissue [19]. Granulomas are more common in later stages of disease.

Microscopy— Quick, cheap and relatively easy laboratory confirmation of Buruli ulcer; especially useful in areas of limited resources. It is done by direct smear examination with Ziehl-Neelsen staining to detect acid-fast bacilli. Sensitivity is on the lower side (40 – 78%), lessening the overall effectiveness of this technique. Moreover, this method does not differentiate between other infectious mycobacteria.

Culture— This method necessitates more sophisticated laboratory tools given difficulties with slow growing bacteria. Lowenstein-Jensen culture is typically used, however other media have been used. Cultures take 9-12 weeks of incubation. Sensitivity has been recorded at 35 – 60%. Culture yield is highest from plaque specimens and lowest from edematous lesions. Even with the difficulties inherent in culturing, this method is considered useful as a confirmatory test to detect viable bacilli in clinically suspected relapses and patients with nonhealing lesions after treatment.

Serological tests and interferon-gamma release assays have yet to be developed for Buruli ulcer. Moreover, Tuberculin skin testing is not recommended as it does not discriminate between mycobacterial infections; these tests also tend to be negative during the early course of infection [20]. A promising POCT for Buruli ulcer is currently under study^{***}. The test is an assay that uses thin-layer chromatography for the detection of mycolactone. Suspected osteomyelitis should be further examined with imaging modalities per society guidelines.^{**}

Treatment (National Institute of Biotechnology, WHO, and Amoako)

The most effective treatment for Buruli ulcer is a combination of antibiotics and complementary therapy, depending on the severity of the disease. When it comes to the treatment of Buruli ulcer Category I, antibiotic treatment alone can offer an effective cure. For Category II and Category III Buruli ulcers, a combination of antibiotics and surgical debridement of the wound offers the best cure.

When it comes to surgical debridement, there are not clearly delineated guidelines. Treatment is however on a case-by-case basis. In general, surgical debridement helps reduce the mycobacterial burden and mycolactone production in infected persons but does not fully clear bacteriuria from the patient's system.

Antibiotic treatment is the single most important treatment option for clearing the bacterial infection. Early detection, antibiotic treatment, and surgical debridement would provide the best outcome for the patient. However, as

most cases of Buruli ulcer are seen in rural areas with lack of resources, patient typically do not have access to surgical debridement and as a result, have physical deformities that can only be treated with plastic surgery restoration.

The choice of antibiotics for the treatment of Buruli ulcer depends on the geographical location of the disease. For instance, in Australia, a combination of rifampicin (10 mg/kg once daily) and moxifloxacin (400 mg once daily) is often used with excellent outcomes. The effectiveness of double antibiotic therapy used in Australia has yet to be proven in any randomized clinical trial. In Africa however, especially in Ghana, where the disease is prevalent, rifampicin (10 mg/kg once daily) and clarithromycin (7.5 mg/kg twice daily) is the recommended treatment option which offers a good outcome. These regimens, while in practice, have yet to be extensively studied in clinical trials.

Buruli ulcer is foreign to the United States of America

therefore there is little known or research done about it. Due to a lack of research on this disease, the CDC recommends following the World Health Organization's (WHO) [16] treatment recommendations. Other countries like Ghana have developed local guidelines or modifications to the WHO's treatment recommendations to combat the disease. (Add in local guidelines and modification here)

The treatment approach to Buruli ulcer offered by the World Health Organization (WHO) is based on the size of the lesion and the tissue damage caused by the lesion. Antimicrobial therapy is the initial therapy of choice. For lesions that require some form of surgery, delayed surgical intervention is preferred, since deterioration during antimicrobial treatment is uncommon and extensive early debridement leaves more tissue damage than necessary. The progression of lesion size on antimicrobial therapy may reflect a paradoxical reaction.

Category I



Above images show plaque, edematous and nodular forms of the disease respectively. Category I lesions are under 5 cm.

Category I lesions often heal spontaneously with less or no medical intervention. This is treated with 4 weeks of antibiotics. There is often no need for surgical intervention or debridement of the disease in category I.

Category II



Above pictured ulcerative forms of the disease. Category II lesions are within 5 and 15 cm.

This requires typically long-term antibiotic treatment and surgical intervention for proper restoration of organ function. The use of antibiotics for 8 weeks provides a good outcome with total recovery. The use of debridement and surgical intervention is often appropriate. There are

Category III



Above left category III lesion involving the eye. Above right depicting contracture, a disease sequela.

It often involves critical sites like the eye the nose and the genitalia. Prolonged antibiotic course and surgical debridement are necessary for complete healing. Following a prolonged course of antibiotic regimen for the treatment category III of the disease, plastic reconstructive surgery is often key to restoring bodily function and preventing disability in those patients that develop contractures from the ulcers. Long-term physical therapy is core to returning patients to full body function. Infectious disease experts should be consulted for appropriate treatment of osteomyelitis associated with Buruli ulcer.

Other Treatment Options

The use of other treatment options for the treatment of Buruli ulcer has been explored. The use of topical therapies like nitrogen oxide, phenytoin powder, and the use of hyperbaric chamber has been used with effective outcomes. In Africa, locals use doxycycline mixed with either palm oil or palm cannula as a topical application to Category I, Category II, and Category III lesions with moderate success. The use of boiling water to clean the wounds among begun in patients is very common with moderate success largely due to the temperature sensitivity of mycobacteria ulcerans with ultimate growth conditions at 29 to 33° C.

Category II and III are recommended to have some surgical debridement as part of the treatment plan for good outcomes. Surgical debridement along with wound

reported cases of relapses with category II, and the repeat of antibiotic therapy is appropriate. Physical therapy is often offered for full-body function without limitations after treatment of category II. Skin grafting may also be necessary depending on the location of the lesion, especially lesions involving the face. If surgical intervention is warranted, eight weeks of antibiotic therapy is recommended before surgical intervention.

care should be delayed for at least 14 weeks following the completion of successful antibiotic therapy for better outcomes. Paired with physical therapy and analgesics, it leads to better outcomes and prognosis.

Psychological factors also play a significant role when it comes to the clinical course of Buruli Ulcer. Most patients develop depressive symptoms requiring psychological counseling and SSRIs. There has also been a correlation between neglected tropical diseases and mental illness, as many of these diseases cause a decrease in quality of life [21]. Not only are patients left debilitated, but they can also be isolated from society. The social stigma of the disease comes from the belief that ulcers were caused by curses or witchcraft [22]. As transmission is still debated and not fully understood, the mystery of the disease causes many to steer clear of the affected.

The Future of Buruli Ulcer

To offer a better preventive measure to the spread of Buruli ulcer, a better understanding should be established of how the disease spreads. There needs to be a clear understanding of how the disease is spread to interrupt the mode of transmission. Currently, the Bacillus Calmette-Guérin (BCG) vaccination appears to provide limited protection to those who receive it [23-25].

When it comes to the future of Buruli Ulcer, the WHO has mentioned five topics that future research should look at. These five areas include (1) mode of transmission (2) Early Diagnostic Methods (3) New Treatment regimens that shorten treatment time (4) Cultural studies (5) Complete

mapping of Buruli Ulcer. Overall, while research has helped a great amount when it comes to research, more time and resources should be put into not only understanding the disease but also creating more effective interventions so morbidity and mortality can decrease, not because the disease is being forgotten from the world, but because the local communities don't feel its impact on its members.

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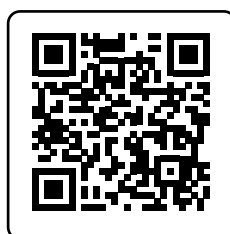
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