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Antimicrobial Susceptibility Patterns of Mycobacterium ulcerans Isolates: Implications for Buruli Ulcer Treatment

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Introduction

Buruli Ulcer (BU) is a neglected tropical disease caused by Mycobacterium ulcerans, a slow-growing bacterium. It primarily affects the skin and subcutaneous tissues, leading to destructive lesions and ulcers. BU is prevalent in several countries, including sub-Saharan Africa, Australia and certain parts of Asia and the Americas. The conventional treatment for BU involves a combination of surgical intervention and antibiotics. However, the emergence of antimicrobial resistance among M. ulcerans isolates has raised concerns about the efficacy of antibiotic therapy. This review aims to explore the antimicrobial susceptibility patterns of M. ulcerans isolates and discuss their implications for Buruli ulcer treatment [1, 2].

Mycobacterium ulcerans is naturally resistant to several commonly used antibiotics, including β-lactams, aminoglycosides and macrolides. However, specific antibiotics have demonstrated activity against this pathogen. Clarithromycin, rifampicin and streptomycin are the primary antibiotics used in the treatment of BU. Clarithromycin inhibit protein synthesis, rifampicin targets RNA polymerase and streptomycin disrupts protein synthesis by binding to the 30S ribosomal subunit. These antibiotics have shown good efficacy in the management of BU lesions and preventing disease progression [3].

Rifampicin resistance in ulcerans isolates is a growing concern. Recent studies have reported an increasing prevalence of rifampicin resistance, primarily due to mutations in the rpoB gene, which encodes the β-subunit of RNA polymerase. Rifampicin resistance compromises the effectiveness of combination therapy and poses a significant challenge in the treatment of BU. Alternative antibiotics, such as rifabutin, have shown potential for rifampicin-resistant cases. However, their efficacy and optimal dosing regimens require further investigation [4].

Clarithromycin resistance is another emerging issue in the treatment of BU. Clarithromycin resistance has been linked to mutations in the roll gene, encoding the 23S rRNA. These mutations reduce the binding affinity of clarithromycin to its target site, leading to reduced efficacy. Studies have reported increased rates of clarithromycin resistance in M. ulcerans isolates, highlighting the need for alternative treatment options. Azithromycin, another macrolide antibiotic, has shown promising *in vitro* activity against clarithromycin-resistant strains. Combination therapy with azithromycin and rifampicin has demonstrated favourable outcomes in some cases of BU. Streptomycin have traditionally been a key component of BU treatment; however, its role is evolving due to concerns about toxicity and emerging resistance. M. ulcerans isolates generally exhibit susceptibility to streptomycin, but reports of streptomycin resistance have also emerged. As a result, the World Health Organization (WHO) no longer recommends streptomycin as a first-line drug for BU treatment. Alternative injectable antibiotics, such as amikacin, may be considered in cases of streptomycin resistance or intolerance.

Combination therapy is crucial in the treatment of BU to enhance efficacy and prevent the development of resistance. Currently, the recommended combination consists of rifampicin and clarithromycin or azithromycin. However, with the increasing prevalence of resistance to these antibiotics, alternative combinations and newer agents are being explored. Fluor quinolones, such as moxifloxacin and ciprofloxacin, have demonstrated *in vitro* activity against M. ulcerans and may have potential as alternative treatment options. Clinical trials are needed to assess their safety and efficacy in the management of BU [5].

Conclusion

The antimicrobial susceptibility patterns of M. ulcerans isolates have significant implications for the treatment of Buruli ulcer. The emergence of resistance to rifampicin and clarithromycin

poses challenges in the management of BU, necessitating the exploration of alternative antibiotics and combination therapies. Streptomycin sensitivity remains an important consideration, although its role is evolving. Continued research and surveillance are vital to monitor antimicrobial resistance trends and develop effective treatment strategies for Buruli ulcer.

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