

# Management of Buruli ulcer–HIV coinfection

Technical update



World Health  
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Areas of Africa endemic for Buruli ulcer (BU), caused by *Mycobacterium ulcerans*, also have a high prevalence of human immunodeficiency virus (HIV), with adult prevalence rates between 1% and 5%.

However, there is a lack of information on the prevalence of BU–HIV coinfection. Further study is needed to clarify this association and enhance knowledge about the prevalence of BU–HIV coinfection in endemic areas.

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## Key learning points

- All Buruli ulcer (BU) patients should be offered high-quality provider-initiated HIV testing and counselling.
  - Cotrimoxazole prophylaxis should be commenced immediately for all HIV patients with a CD4 count  $\leq 350$  cells/mm<sup>3</sup>, or if CD4 count is not available and the patient has advanced symptomatic HIV disease (WHO clinical stage 3 or 4). In settings with high prevalence of malaria and/or severe bacterial infections, cotrimoxazole prophylaxis should be initiated in all individuals regardless of CD4 cell-count.
  - Combination antibiotic treatment for BU should be commenced before starting antiretroviral therapy (ART) and given for 8 weeks' duration. The recommended combination is rifampicin plus streptomycin. An alternative regimen is rifampicin plus clarithromycin, although due to drug interactions this regimen should be used with caution.
- ALL BURULI ULCER (BU) PATIENTS SHOULD BE OFFERED HIGH-QUALITY PROVIDER-INITIATED HIV TESTING AND COUNSELLING.
- ART should be initiated in all BU–HIV coinfecting patients with advanced symptomatic HIV disease (WHO clinical stage 3 or 4) regardless of CD4 cell-count and in those asymptomatic individuals with CD4 count  $\leq 500$  cells/mm<sup>3</sup>. If CD4 cell-count is not available, BU–HIV coinfecting individuals with WHO category 2 or 3 BU disease should be offered ART.
  - For eligible individuals, ART should be commenced as soon as possible within 8 weeks after commencing BU treatment and as a priority in those with advanced HIV disease (CD4  $\leq 350$  cells/mm<sup>3</sup> or WHO clinical stage 3 or 4 disease).
    - All BU–HIV coinfecting patients should be actively screened for tuberculosis before commencing BU treatment and before starting ART.
    - Programmes should implement a monitoring and reporting system to monitor and evaluate the outcomes of BU–HIV interventions.

## Background

Areas of Africa endemic for Buruli ulcer (BU), caused by *Mycobacterium ulcerans*, also have a high prevalence of human immunodeficiency virus (HIV), with adult prevalence rates between 1% and 5% (Map<sup>1</sup>). However, there is a lack of information on the prevalence of BU–HIV coinfection. Preliminary evidence suggests that HIV infection may increase the risk of BU disease (1–3). In the Médecins Sans Frontières project in Akonolinga, Cameroon, HIV prevalence was approximately 3–6 times higher among BU patients than the regional estimated HIV prevalence (3). Similarly

in Benin and Ghana, BU patients were 8 times and 4 times respectively more likely to have HIV infection than those without BU (1,2). Further study is needed to clarify this association and enhance knowledge about the prevalence of BU–HIV coinfection in endemic areas.

HIV may affect the clinical presentation and severity of BU disease, with a reported increased incidence of multiple, larger and ulcerated BU lesions in HIV-infected individuals (3–6). Additionally in the Akonolinga project, the main lesion size was significantly increased with decreasing CD4 cell-count levels (3).

Distribution of Buruli ulcer, worldwide, 2013



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