Management of Buruli ulcer–HIV coinfection

Technical update

Contents

WHO/HTM/NTD/IDM/2015.01

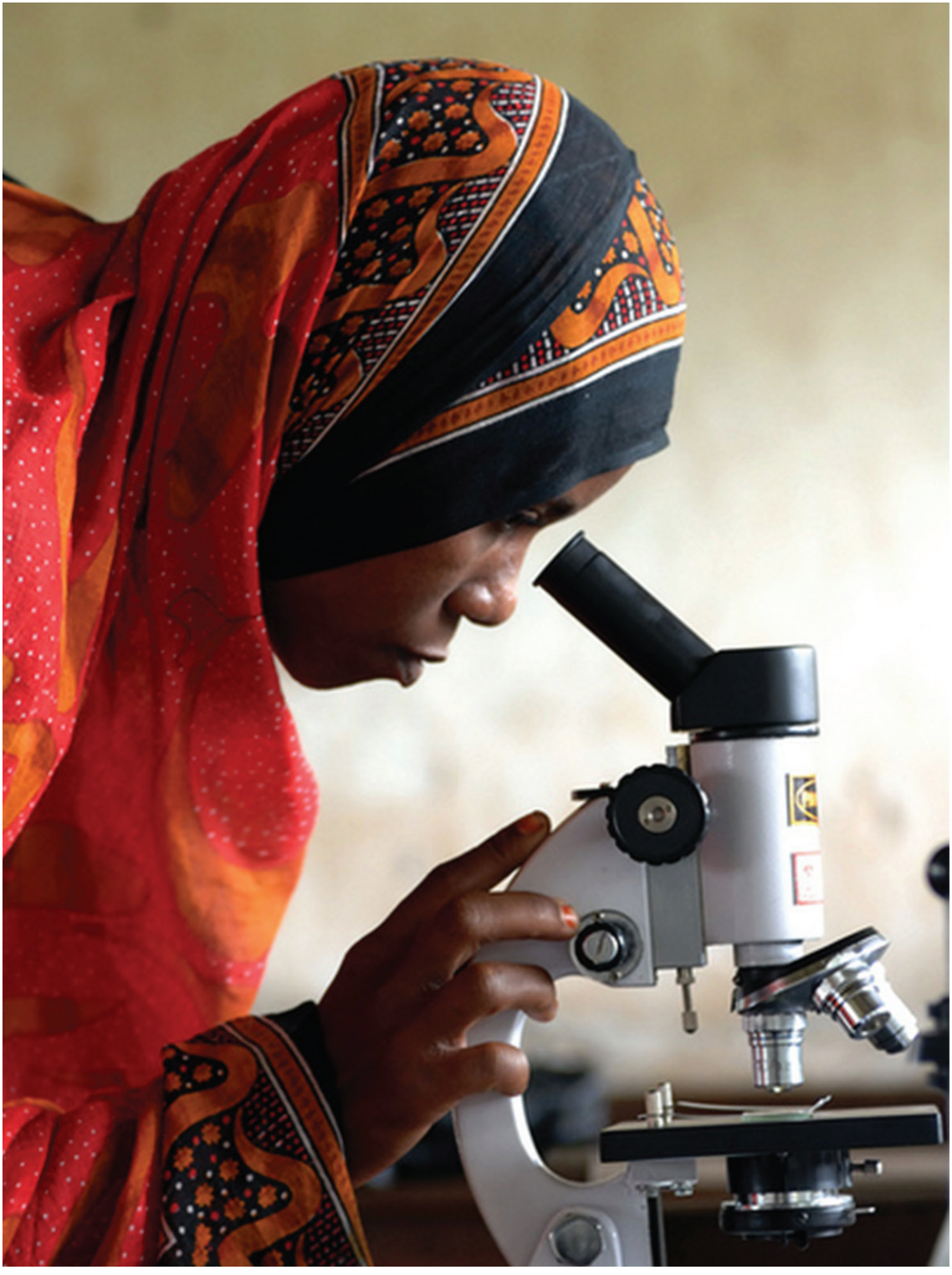
BURULI ULCER–HIV COINFECTION

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.

Management of Buruli ulcer–HIV coinfection

Technical update



Acknowledgements

WHO would like to thank the following people for their contributions to the development of this document:

- Yaw Amoako
  Komfo Anokye Teaching Hospital, Kumasi, Ghana
- Edwin Ampadu
  Ghana Health Service, Accra, Ghana
- Kingsley Asiedu
  WHO Department of Control of Neglected Tropical Diseases, Geneva, Switzerland
- Henry Asse
  Buruli ulcer control programme, Abidjan, Côte d'Ivoire
- Alexandra Calmy
  University of Geneva, Switzerland
- Annick Chauty
  Buruli ulcer Treatment Hospital, Pobe, Benin
- Vanessa Christinet
  University of Geneva, Switzerland
- Eric Comte
  MSF-Switzerland, Geneva, Switzerland
- Meg Doherty
  WHO Department of HIV/AIDS, Geneva, Switzerland
- Serge Eholie
  University Hospital, Abidjan, Côte d'Ivoire
- Nathan Ford
  WHO Department of HIV/AIDS, Geneva, Switzerland
- Haileyesus Getahun Gebre
  WHO Global TB Programme, Geneva, Switzerland
- Serge Mathurin Kaboré
  MSF-Switzerland, Yaoundé, Cameroon
- Anatole Kibadi
  Faculté de Médecine, Université de Kinshasa, Democratic Republic of the Congo
- Charles Kouanfack
  Hôpital Central de Yaoundé, Cameroon
- Johnny Lujan
  MSF-Switzerland, Geneva, Switzerland
- Lisa Nelson
  WHO Department of HIV/AIDS, Geneva, Switzerland
- Daniel O'Brien
  The Geelong Hospital, Victoria, Australia
- Albert Paintsil
  Korle-Bu Teaching Hospital, Accra, Ghana
- Delphin Phanzu
  Institut Médical Evangélique, Kimpese, Democratic Republic of the Congo
- Richard Phillips
  Komfo Anokye Teaching Hospital, Kumasi, Ghana
- Gerd Pluschke
  Swiss Tropical and Public Health Institute, Basel, Switzerland
- Francoise Portaels
  Institute of Tropical Medicine, Antwerp, Belgium
- Fred Sarfo
  Komfo Anokye Teaching Hospital, Kumasi, Ghana

- Micaela Serafini
  MSF-Switzerland, Geneva, Switzerland
- Ghislain Sopoh
  Buruli ulcer Treatment Hospital, Allada, Benin
- Ymkje Stienstra
  University Medical Center Groningen, Netherlands
- Marco Vitoria
  WHO Department of HIV/AIDS, Geneva, Switzerland
- Mark Wansbrough-Jones
  St George's Hospital Medical School, London, UK
- Dorothy Yeboah-Manu
  Noguchi Memorial Institute for Medical Research, Accra, Ghana

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 ≤350 cells/mm^3, 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 coinfected 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 ≤500 cells/mm^3. If CD4 cell-count is not available, BU–HIV coinfected 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 ≤350 cells/mm^3 or WHO clinical stage 3 or 4 disease).

- All BU–HIV coinfected 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% (*Maps*). 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, worlwide, 2013