

Problematic Management of Buruli Ulcer and HIV Co-infection in Tropical Regions

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Editorial

Tropical and sub-tropical regions, in particular sub-saharan Africa account for more than 80% of Human immunodeficiency virus (HIV) infection worldwide. The co-infection of HIV and tropical infectious dermatoses agents is inevitable, and it constitutes a major challenge in term of treatment option and these diseases clinical courses [1].

In Côte d'Ivoire, the most sub-Saharan Africa country affected by Buruli ulcer, more than 2000 new cases per year are diagnosed, and HIV and *Mycobacterium ulcerans* co-infection [2] become more and more frequent.

The HIV infection in Buruli ulcer patients worsens their disease clinical course that leads to bad prognosis and sometimes to treatment failure or to immune reconstitution Inflammatory Syndrome (IRIS). The latest is poorly understood, but well documented [3]. IRIS occurs in the setting of Anti-retroviral treatment (ART) initiation and it is considered as a deregulated immunologic response to a previously existing pathogen such as *mycobacterium ulcerans* in Buruli ulcer. Clinically, we have two types of IRIS as follows, the unmasking IRIS in which a previously unrecognized infection becomes clinically apparent as immune reconstitution occurs, and the paradoxical IRIS which causes clinical deterioration of previously recognized and sometimes treated infections [4].

This new context of the immune system leads to new or worsening clinical manifestations. In fact; IRIS occurs mostly in the first weeks or months after Highly Active Antiretroviral therapy (HAART) initiation. Among the factor known to predispose to IRIS, there are: a very low CD4 cell counts at the initiation of ART and a preexisting infectious disease like Buruli ulcer [4].

These paradoxical reactions have recently recognized to complicate up to 20% of patients receiving antibiotics BU, and sometimes leads to secondary multifocal BU lesions.

Concerning BU, this paradoxical reactions are proposed to result from reversal of the mycolactone toxin induced immune-inhibitory state via the antibiotic mediated killing of *mycobacterium ulcerans* organisms allowing intense immunological reaction to develop against the persisting mycobacterial agents [5,6].

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As, it is already known that paradoxical reactions are common in HIV patients starting ART with varieties of microorganisms such as Tuberculosis, *Cryptococcus* and *Mycobacterium Avium* complex. Moreover, in TB/HIV co-infection, the incidence of IRIS occurrence is increased in patients who start ART within 30 days of TB treatment initiation [7]. Comparing to the case report recently described in Côte d'Ivoire with multifocal BU lesions developing in a HIV patient with severe immunodepression (baseline CD4 cell counts of 51 cell/ mm³), on month after both BU and HIV treatment initiation [2].

The use of corticosteroid agents to reduce these paradoxical reactions was reported by several authors, and evidence from mouse model suggested that corticosteroids use does not lead to BU treatment failure [8].

So, patients co-infected with BU and HIV represents a new challenge for scientist and clinicians in term of disease clinical manifestation and courses and treatment strategy.

As, we know, HIV co-infections may affect the outcome of BU mortality rate, time to healing, recurrence rate and the incidence of paradoxical reactions.

Because of interactions of both antibiotics used to treat BU and Anti-retroviral drugs used to treat HIV infection, many questions arise:

Which treatment option to take and when treatment should be start both antibiotics for BU and ART for HIV?

As a response for these new challenges, initiatives were taken by a team of scientist to establish consensus guidance to manage Buruli ulcer and HIV co-infected patients.

The following suggestions should be taken account as initial guidance for these patients care:

Systematic HIV testing for all BU patients after given informed consent, for eligible patients ART should be commenced as soon as possible after the start of BU treatment, preferably within 8 weeks, before BU treatment, patient must be screened for

Tuberculosis [9]. In addition, Moxifloxacin which is active against both TB and BU, and orally taken should be recommended [10]. But it is not recommended in pregnancy or in children under 18 years of age, and has limited availability due to its high cost in tropical regions. Moreover, Tenofovir should not be combined to streptomycin because it increases renal toxicity risk [9].

Clinicians in Sub-Saharan Africa should be able to distinguish IRIS-associated signs or symptoms to treatment failure or an adverse drug reaction in patients co-infected with HIV and BU after HAART initiation. Therefore, more study should be conducted in order to set up a real treatment protocol for BU-HIV co-infection worldwide, in particular in Sub-Saharan Africa.

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