Persistence of *Mycobacterium ulcerans* Disease (Buruli Ulcer) in the Historical Focus of Kasongo Territory, the Democratic Republic of Congo

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**Abstract.** Fifty years after the last report of *Mycobacterium ulcerans* infections (Buruli ulcer [BU]) in Kasongo Territory, Maniema Province, Democratic Republic of Congo (DRC), we conducted a small-scale cross-sectional survey to assess whether this historical BU focus was still active and to explore the disease epidemiology. Seventy-five active and inactive BU cases were identified on clinical grounds of which two of 28 BU active cases were laboratory confirmed. We used a modified BU02 form to reconstruct the local disease dynamics and we believe that the horrific conflict in eastern DRC and exceptional flooding were the most likely causes of the re-emergence of the disease. There is a need in the DRC to decentralize and integrate surveillance and control activities at local level to increase the effectiveness of patient management.

**INTRODUCTION**

Buruli ulcer (BU), which is caused by the environmental pathogen *Mycobacterium ulcerans*, has been recently designated by the World Health Organization (WHO) as one of the 15 neglected tropical diseases. These diseases are a diverse group of poorly studied diseases that significantly impact the lives of the world’s poorest people. Buruli ulcer is often considered the emerging successor of leprosy, the best-known ancient affliction of stigma and poverty. The disease is characterized by an indolent necrotizing of skin, subcutaneous tissue, and occasionally bones. Disfiguring ulcers and important disabilities are the main consequences of inadequate or delayed treatment.

A better understanding of the transmission and disease dynamics of *M. ulcerans* infections will have a direct impact on the development of appropriate and effective control strategies against this devastating neglected disease. However, the epidemiology, natural history, and biology of BU remain cryptic.

Worldwide, BU has been confirmed in 26, mainly tropical, countries. Within disease-endemic countries, BU has generally been reported from remote rural areas and is characterized by its patchy and focal distribution. There is a strong epidemiologic association between BU and wetlands, particularly those with slow-moving or stagnant water. Indirect evidence, including the recent report of the first cultivation and characterization of *M. ulcerans* from an aquatic insect (a water strider, Gerridae), supports the hypothesis that *M. ulcerans* is an environmental pathogen transmitted to humans.

The prevalence of BU varies significantly between continents, between disease-endemic countries, and at microgeographic level (i.e., between or within villages or hamlets).

Prior to 1980, the incidence of BU was mainly restricted to Uganda and the Democratic Republic of Congo (DRC). However, disease incidence in these countries appears to have decreased, but increased in west Africa. It has been hypothesized that this spatial shift has occurred because of environmental changes and human activities.

To the best of our knowledge, no BU cases were reported in the DRC between 1980 and 2000. Because research and control activities recommenced at the beginning of the 21st century, a number of BU cases have been diagnosed in areas of DRC that were characterized as endemic for BU by Meyers and others in 1974. However, in general, it is not fully understood whether the increased number of cases reported from well-circumscribed areas is caused by intensified surveillance activities alone or to a real re-emergence of the disease.

To better understand the dynamics of BU and the underlying reasons of its patchy, focal distribution in the DRC, we conducted a small-scale cross-sectional study in the historical BU focus of Kasongo Territory, Maniema Province, DRC, in October 2007. This well-defined disease focus was first described in 1959. More recent epidemiologic data are lacking. The primary objective of the study was to examine the prevalence of BU in Kasongo Territory. In this report, we also explore and discuss the dynamics and extent of this disease. This work is part of a larger study exploring the ecologic and epidemiologic aspects of BU at different levels in west and central Africa. Therefore, the ecologic aspects of the present study will be discussed in more detail elsewhere.

**MATERIALS AND METHODS**

**Survey area.** The study area extended to two rural health zones (Kasongo and Kunda), Kasongo Territory, southern Maniema Province, DRC (Figure 1). Maniema Province (0°–5°S, 24°–29°E) covers an area of 132,250 km², approximately 5.6% of the total surface of the DRC. The Congo River bisects the province, flowing from northwest to southeast.
The southern part of the province is covered mainly by savannah, alternating with patchy forests, and the center-western and northern parts are covered by the humid tropical forest that changes to equatorial forest in the extreme northern region. Study villages were selected on the basis of historical reports of BU incidence; anecdotal information from former physicians of the Institute of Tropical Medicine (ITM), Antwerp, Belgium, based at Kasongo Reference Hospital; and recent presumptive data from officers of the rural health zones and Coordination Provinciale Lèpre-Tuberculose-Ulère de Buruli, Maniema Province. Nine health centers and a mobile health post, covering an average of five villages each and all located within a radius of less than 35 km and 55 km from the Congo River and Kasongo City, respectively, were selected (Figure 1).

Data collection. A team of researchers conducted a pilot study in selected areas in the early part of October 2007. First, a short training course of BU control activities was organized for 66 local medical staff, including physicians, senior nurses, and coordination staff of both rural health zones. Then, prior to sensitization and passive case-finding activities, villagers were informed of the study by a transmission on local radio. Sensitization of local residents was conducted through the presentation of a WHO documentary film on BU, with a simultaneous translation into the local language (i.e., Swahili), followed by an opportunity for villagers to ask questions about the study. Villagers with any chronic and/or painless (cutaneous) lesion were invited to come to the local health office for clinical screening of BU the morning after the presentation. Any persons who were found not to be infected with BU were referred for appropriate medical care.

Cases of BU were classified according to the WHO case definitions, and photographs of lesions were taken. To reconstruct the dynamics of BU in Kasongo Territory, data were captured using a modified form BU02. Modifications of form BU02 included additional data on disease onset, residence at estimated time of infection, disease course, and type and name of most frequented water bodies prior to disease onset.

Sample collection and analysis. Because of important logistic restrictions in the remote areas, only swab specimens were taken from cases ulcerative forms of BU that were presumptively active. Sterile cotton swabs were used, placed in a semisolid transport medium, and maintained as close to ambient temperature as possible.
to 4°C as possible prior transport to the ITM. At the ITM, microscopic examination was performed after Ziehl-Neelsen staining, suspensions were decontaminated and inoculated on Löwenstein-Jensen medium, and the presence of M. ulcerans DNA was detected by insertion sequence IS2404 polymerase chain reaction (PCR) as described. Genotyping of M. ulcerans was performed on PCR-positive samples by variable number of tandem repeats analysis as reported elsewhere.

**Ethics.** As required and approved by the Congolese Public Health Ministry, data and samples were collected only after written informed consent was obtained from the patients, their responsible relative, or guardian.

**Statistical analysis.** Data were transcribed from BU02 forms using Excel (Microsoft, Redmond, WA) and analyzed with Epi Info version 3.4.3. (Centers for Diseases Control and Prevention, Atlanta, GA). The chi-square test was used to examine associations between dichotomous variables. Continuous variables were analyzed using Student’s t-test or analysis of variance as appropriate. Two-tailed tests were used, with P < 0.05 considered significant.

**RESULTS**

A total of 250 persons had self-diagnosed symptoms of BU. Study clinicians suspected BU in 92 of these patients and BU was diagnosed clinically in 75. Of these patients, 28 (37%) had active lesions and 47 (63%) had inactive cases, i.e., healed ulcers, chronic functional disabilities, and/or amputation (Table 1). The age and sex distribution of all clinically diagnosed BU cases are shown in Figure 2A. The results of analysis of the sex ratio (M:F = 1.2), median age, and age range are shown in Table 1. No significant difference in age and sex were observed (P = 0.705). The estimated year of disease onset of cases is shown in Figure 2B. Twice as many patients (52 versus 23) reported having developed the disease since 1995. The age and sex distribution at estimated time of disease onset is shown in Figure 2C. This distribution was determined by subtracting the estimated year of disease onset from the exact or estimated year of birth. Of 75 patients, 53 (71%) cases were less than 15 years of age at estimated disease onset. More than two-thirds (54) of the patients were residents in the rural health zone of Kasongo.

The severity of illness (graded according to WHO criteria) did not differ significantly between both active and inactive cases (P = 0.172), patients of different sexes (P = 0.334), patients in different age groups (P = 0.923), or the presence of a Bacille Calmette-Guérin vaccination scar (P = 0.110) (Table 1). A total of 46 (61%) patients with lesions satisfied the WHO category III definition, of which 32 (70%) had osteomyelitis and/or multiple lesions (Table 1). However, osteomyelitis was not observed more often in cases without a Bacille Calmette-Guérin than in those with the scar (4 of 43 versus 1 of 28; P = 0.356). The distribution of location of lesions on the body is shown in Figure 3. Sixty-four (85%) of 75 patients had lesions on the extremities, with the lower limbs (39) involved more than twice as often as the upper limbs (19); mixed lesions on the extremities (7) not included in this comparison. The distribution of lesions by location on the body did not show any significant differences with respect to age (P = 0.168) or sex (P = 0.454).

Thirty-four (45%) patients mentioned that a family member or relative had previously had or currently had BU. More than two-thirds (68%) of the cases were being treated with traditional treatment. Fifty-two (69%) of the patients reported having had a previous episode of BU. A total of 31% (23) reported no recurrence or did not remember a previous episode.

Forty-four swab samples were taken from the lesions of 22 (29%) of the 28 clinically diagnosed active cases for laboratory confirmation of the presence of M. ulcerans. Samples could not be taken from the other six cases because they had distinct ulcers in an advanced stage of healing. Six (14%) of 44 samples revealed acid-fast bacilli by direct microscopic examination (Ziehl-Neelsen staining). Among these 44 samples, four (9%) were positive for IS2404 PCR, but genotyping of these positive samples by variable number of tandem repeats analysis was not successful. All positive samples (microscopy and PCR) were from two cases (Table 2 and Figure 4). Four clinical samples were positive by microscopy and PCR. Mycobacterial growth was not detected by in vitro culture after one year of observation.

**DISCUSSION**

Six points were considered by study clinicians when making a clinical diagnosis of BU: 1) the origin of suspected cases (i.e., whether a patient was resident in an area with an existing record for BU); 2) the presence of a painless tumefaction (evolving into a chronic ulcer); 3) the presence of a lesion difficult to treat with most commonly available disinfectants and antibiotics; 4) the location of lesions (85% were located on the limbs, Figure 3); 5) the presence of typical stellate-shaped scars (for inactive lesions) after (spontaneous or traditional) healing; and 6) a typical clinical spectrum (Table 1).

The age-sex distribution of cases at the time of estimated disease onset (Figure 2C), was consistent with previous reports: more than 50% (53 of 75, 71%) with BU were children less than 15 years of age (median age = 10 years), with the highest rate of infection in those 10–14 years of age. Rates of infection among males and females were approximately equal (Table 1). The age-sex distribution of all patients at time of diagnosis (Figure 2A and Table 1) is

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**Table 1**

Overview of clinico-epidemiologic data of 75 clinical diagnosed patients with Buruli ulcer, Democratic Republic of Congo

<table>
<thead>
<tr>
<th>Variables</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>40</td>
<td>35</td>
<td>75</td>
</tr>
<tr>
<td>Median age (years)</td>
<td>20</td>
<td>25</td>
<td>23</td>
</tr>
<tr>
<td>Age range (years)</td>
<td>7–79</td>
<td>8–75</td>
<td>7–79</td>
</tr>
<tr>
<td>BCG vaccination (scar)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BCG+</td>
<td>15</td>
<td>13</td>
<td>28</td>
</tr>
<tr>
<td>BCG–</td>
<td>24</td>
<td>19</td>
<td>43</td>
</tr>
<tr>
<td>BCG?</td>
<td>1</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Lesion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active</td>
<td>11</td>
<td>17</td>
<td>28</td>
</tr>
<tr>
<td>Inactive</td>
<td>29</td>
<td>18</td>
<td>47</td>
</tr>
<tr>
<td>Clinical spectrum†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Category I</td>
<td>6</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Category II</td>
<td>12</td>
<td>9</td>
<td>21</td>
</tr>
<tr>
<td>Category III</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Osteomyelitis and multiple lesions</td>
<td>16</td>
<td>16</td>
<td>32</td>
</tr>
<tr>
<td>Unique lesion (&gt; 15 cm diameter) and critical localization</td>
<td>6</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td>35</td>
<td>75</td>
</tr>
</tbody>
</table>

*BCG = Bacille Calmette-Guérin. No significant differences were found in any comparisons.
†Age estimated between 65 and 75 years.
‡According to the definitions of the World Health Organization, 2008.
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most likely caused by the high number of patients with inactive lesion(s) (63%).

It is evident from the survey that monitoring and treatment of BU in the study area is inadequate. Health records show no recording of pre-ulcerative symptoms, a high proportion of patients presented with category III lesions (61%). We would expect that even in a region with minimal BU surveillance, lesions would be diagnosed and treated while still category I or II. The high number of inactive cases (63%), the high number of persons seeking traditional treatment (68%) and the high recurrence rate (69%) are further indications that patients were unable to get satisfactory care in the study area.

Although only two patients were clinically and laboratory (microscopy and PCR) confirmed with BU infection, 73 patients were diagnosed on clinical and epidemiologic grounds. This relatively low number of laboratory-confirmed cases is most likely caused by the fact that most patients had ulcers in an advanced stage of healing. Furthermore, lesions developed in the two laboratory confirmed cases just prior to diagnosis.

A high contamination rate (approximately 80%) was observed in samples taken from lesions. This finding is probably caused by poor skin hygiene of people in this highly disadvantaged region. The contamination rate could probably have been reduced if the appropriate resources for (punch) biopsies and/or fine needle aspiration had been present. Based on the results of clinical and laboratory diagnosis, our study confirmed that the Kasongo Territory is still (or again) an active BU focus.

Because presentation at health centers was voluntary, we assume that our results have underestimated the actual prevalence, as reported previously. To reconstruct the dynamics of BU, we used a modified form BU02. The estimated time of disease onset of BU cases, shown in Figure 2B, indicates that BU was already had a low prevalence in Kasongo Territory in the early 1950s. This observation is supported by

Figure 2. A. Age (in years) and sex distribution in 75 clinically diagnosed patients with active and inactive Buruli ulcer at diagnosis (October 2007). B. Sex distribution of 75 clinically diagnosed patients with Buruli ulcer at estimated time of disease onset. C. Age (in years) and sex distribution in 75 clinically diagnosed patients with Buruli ulcer at estimated time of disease onset. Figure 2C was obtained by combining Figures 2A and 2B.

Figure 3. Distribution of lesions in 75 clinically diagnosed patients with Buruli ulcer.
the fact that of 160 hospitalized patients with cutaneous lesions, 23 patients with active cases of BU were reported during a 2.5-year meticulous surveillance in 1959,18,19 By 1960, 33 cases were recorded.4,14 The disease continued to be present at a low prevalence until the mid 1990s (Figure 2B), a finding that is consistent with observations of former ITM physicians at the Kasongo Reference Hospital, who confirmed they had treated 15 patients with active cases of BU during a 3.5-year period after 1994 (Figure 2B). Analyzing this apparent emergence more in detail, we discovered a first peak in the number of cases (8) in 1997, followed by a decrease the next two years and a second peak (10) in 2000, followed by a more steady decrease.

It is plausible to attribute the first peak to the start of the conflict that engulfed Rwanda, Burundi, and the DRC in the mid 1990s. As a result of this horrific conflict, huge populations of people were displaced and there was a marked deterioration in already poor living conditions. Maniema Province, in particular Kasongo Territory, became inaccessible to health and aid workers for many years because of lack of security and its geographic isolation.20,38,39 We believe that the unsanitary and inhuman conditions in the province caused local residents to have greater contact with the infection source. An association of BU with unsanitary conditions has already been demonstrated, and it has been suggested that simple hygiene measures, such as the regular use of soap and the thorough cleaning of injuries, could be important in preventing BU.40

Furthermore, we assume that an immunogenetic explanation for this emergence, caused by refugees from regions not endemic for BU (i.e., influx of new susceptible persons) as reported in Kinyara, Uganda, is unlikely.5,41 All 75 patients reported in Kinyara, Uganda, is unlikely.5,41 All 75 patients were resident in Kasongo Territory. Furthermore, there are no records of refugee camps or major migrations of refugees were resident in Kasongo Territory. Furthermore, there are no records of refugee camps or major migrations of refugees residing within a radius of 10 km of the Mwasa Village (Figure 1).4,18,19

The second observed peak may be explained by the unusual heavy rainfall in the vast Congo River basin between December 2007 and April 2008.42 The subsequent flooding, the worst in the past 35 years, affected some seven million persons living in the catchment of the river.42 This observation is consistent with those of previous reports.13,43,44 Barker suggested that the emergence of BU and the extension of its area in the Busoga District, Uganda, in 1965–1970 was related to the unprecedented flooding of rivers and lakes between 1962 and 1964.44 Such flooding may have resulted in marked ecological changes and changes in human activities. For instance, land use, land cover, and/or habitat changes may have led to an increase in the number of reservoir hosts, an increase in incidence of infection in reservoir hosts, an increase in the density of human hosts in the pathogen’s habitat, and/or a change in exposure between human host and infection source.45

Fifty years ago, all cases of BU were recorded in persons living within a radius of 10 km of the Mwasa Village (Figure 1).4,18,19 However, our results, show that the disease-endemic area appears to have extended south toward the Congo River. Moreover, our data indicate that the main BU focus has apparently been displaced and is now located within a radius of 15 km from the Kankumba Village, which is located between Mwasa and the Congo River (data not shown). In addition, the time of this spatial shift seems to correspond to the period after the mentioned flooding, which is consistent with the observation of Barker in Uganda.44 Finally, we observed that most of the cases observed during this survey lived within 10 km of the Congo River (54 of 75 cases). In addition, as previously observed by other researchers, we determined that all cases observed during this survey came from patients resident in savannah, which in this region is composed of a close network of rivers and fish ponds.8,9,13,41 None of the cases observed during this survey came from persons resident in forested areas.

### Table 2
Overview of characteristics of two laboratory-confirmed (PCR and microscopy) Buruli ulcer patients, Democratic Republic of Congo*  
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Patient 1</th>
<th>Patient 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>F</td>
<td>M</td>
</tr>
<tr>
<td>Age (years)</td>
<td>15</td>
<td>79</td>
</tr>
<tr>
<td>Place of origin: village (rural health zone)</td>
<td>Yamba-Yamba (Kunda)</td>
<td>Lamba (Kasongo)</td>
</tr>
<tr>
<td>Profession</td>
<td>Student</td>
<td>Fisherman</td>
</tr>
<tr>
<td>Date of disease onset</td>
<td>December 2006</td>
<td>August 2007</td>
</tr>
<tr>
<td>Location</td>
<td>Left knee</td>
<td>Left upper arm</td>
</tr>
<tr>
<td>Clinical spectrum</td>
<td>Category III (single lesion &gt; 15 cm)</td>
<td>Category II (5 cm &lt; single lesion &lt; 15 cm)</td>
</tr>
<tr>
<td>Form</td>
<td>Ulcerative</td>
<td>Ulcerative</td>
</tr>
<tr>
<td>BCG vaccination scar</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Knowledge of trauma or previous disease signs/symptoms</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Previous episodes (recurrence)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Traditional treatment</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Relatives with Buruli ulcer</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Most frequented water body for domestic and/or professional activities</td>
<td>Chamakala River, ponds</td>
<td>Congo River</td>
</tr>
</tbody>
</table>

*PCR = polymerase chain reaction; BCG = Bacille Calmette-Guérin.

**Figure 4.** A. Typical active lesion (laboratory confirmed) on the left upper limb of a 79-year-old man (fisherman). B. Typical active lesion (laboratory confirmed) on the left knee of a 15-year-old woman (student). This figure appears in color at www.ajtmh.org.
Meyers and others described five foci of Buruli ulcer in DRC.\textsuperscript{14–16} The persistence of BU in foci in Bas-Congo and Bandundu were reported recently.\textsuperscript{4,15,16} Our data support the need to explore the disease status in the other two remaining historical foci. The DRC is a vast country whose health, sanitation, transport, and social infrastructure has been destroyed by decades of neglect and conflict. The apparently scattered focal distribution but with nevertheless persistent incidence of BU implies that a more targeted approach to control and surveillance activities would be invaluable in developing a greater understanding of this devastating neglected disease. It seems likely that the current decentralization and reorganization of the public health system in the country will ultimately improve the effective implementation and integration of BU control and surveillance activities at local level, within existing synergetic control programs (i.e., tuberculosis and leprosy), and will eventually be of great benefit to patient care.\textsuperscript{13,17}

We conclude that although there have been no reports of BU from Kasongo Territory for more than half a century, this historical BU focus is still active, that BU subsisted at low prevalence for decades, but that it has probably recently re-emerged and expanded because of conflict and exceptional flooding. However, further investigation is needed to assess the exact burden and extent of the disease in this region. Furthermore, until no substantial support is available, we recommend assessing the exact prevalence and extent of the disease in historical foci first, prior to the exploration of unreported regions in the DRC. This approach may increase our understanding of disease dynamics in scattered foci. However, detection of cases should always be combined with proper case management.

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