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*Full Length Research Paper*

# Cutaneous leishmaniasis in Uganda: Report of the first case at Mulago National Referral and Teaching Hospital

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**The transmission of *Leishmania* in central Uganda is little known despite that equine leishmaniasis was reported on the mainland in 1926. A case is reported of cutaneous leishmaniasis in an immunocompromised patient, possibly acquired on the Ssesse Islands in Lake Victoria or on the mainland or even introduced from a neighbouring country. The patient's facial and limb skin lesions were suspected to be Kaposi's sarcoma or cutaneous mycobacterial disease and diagnosis was made on histological examination. The possibility of parasite visceralisation could not be excluded, but there was evidence of multi-organ disturbance. Treatment options though limited yielded clinical and histological improvement with Amphotericin B. Parasite isolation was not possible and polymerase chain reaction analysis of unstained formalin-fixed paraffin-embedded skin sections identified *Leishmania major* as the causative organism.**

**Key words**: Cutaneous leishmaniasis, *Leishmania*, Epidemiology, Uganda, case management

## INTRODUCTION

Human infection with *Leishmania* protozoa can result in cutaneous, mucocutaneous or visceral leishmaniasis depending largely on the parasite species and host immune response. A majority of infections worldwide are of the localised cutaneous form, occurring in 82 countries with an estimated annual incidence of 1.5 million cases (WHO, 2008). Several species can cause cutaneous leishmaniasis (CL) which is characteristically benign and self-limiting but can develop into diffuse CL in the Old World, and result in mucosal disease in the New World (WHO, 2010a). In sub-Saharan Africa where *Leishmania* are transmitted to humans and animals predominantly by the sandfly vector, encroachment on transmission areas and migration caused upsurge in leishmaniasis cases and emergence of previously unknown foci (Lemma et al., 1969; Kadaro et al., 1993; Sang et al., 1994). As the number of susceptible individuals increases and as populations move across borders, clinicians are confronted with unusual presentations especially since diagnosis is made by the demonstration of parasites. In this article we report a human immunodeficiency virus (HIV)-infected resident of the Ssesse Islands in Lake Victoria with CL, the first case to be treated at Mulago National Referral and Teaching Hospital (MNRTH) in Kampala, Uganda. We also note the possible local and neighbouring sources of infection, the hardships in patient management and problems of parasite species identification.

### Epidemiological aspects

In Uganda, leishmaniasis was reported in a bay gelding at Entebbe (altitude 1 133 to 1 155 m) directly north of Lake Victoria (Richardson, 1926), spleen-smear images of which resembled those from kala-azar cases (Wenyon, 1926). The WHO's essential leishmaniasis map shows a focus of *L. major* north of Lake Victoria (WHO, 2010b) but literature citing human disease is scarce. Since 1951, anthroponotic visceral leishmaniasis (VL) is known in the north-eastern sub-Region of Karamoja (altitude 1 200 to 2 400 m) where *L. donovani* is transmitted by *Phlebotomus martini* (McKinnon, 1962; Wykoff, 1969). This focus is contiguous with a much larger one in the Kenyan Districts of Pokot North, West Pokot, East Pokot and Baringo. From 2005, VL patients came from increasingly north of the original focus in Pokot territory however; no CL had

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been reported by any Health Unit to the Uganda Ministry of Health.

CL due to *L. aethiopica* was described in Bungoma District of Kenya, on the eastern and south-eastern slopes of Mountain Elgon (Kungu et al., 1972). The animal reservoirs were investigated (Mutinga, 1975a) and the vectors (Mutinga, 1975b) and parasite species identified (Chance et al., 1978). Typical all round Mt. Elgon (Figure 1) are the high altitude rain and bamboo forests, the lower altitude caves and waterfalls and the national park. The Luhya people in whom CL was diagnosed inhabit the Kenya side of the mountain, while the Bagisu and Sabiny tribes inhabit the slopes on the Uganda side. As earlier noted, foci of CL may have existed in neighbouring countries with similar ecology (Mutinga and Mngola, 1974). Also reported in Kenya is cutaneous disease due to *L. major*, *L. tropica* and *L. donovani* (Muigai et al., 1987; Mebrahtu et al., 1988; Mebrahtu et al., 1993).

In Tanzania, CL was diagnosed in a leprosy patient (Andersen, 1964) who may have been subsequent to many cases of 'oriental sore' (Clyde, 1964) and pre- and post-mortem diagnoses made in Malawians who had visited Tanzania (Pharoah, 1993). In the Democratic Republic of Congo VL possibly acquired in Rwanda and an indigenous case (Prévot, 1968; Gigase, 1978) were reported but not CL. While the causative organism in VL was presumed to be *L. donovani*, the species of CL remained undetermined (Pharoah, 1993).

South Sudan has strong ties to Uganda with cross-border exchange of people, goods and animals. Equine dermo-leishmaniasis was also reported in the Sudan (Bennett, 1935) and mucosal lesions (Christopherson, 1914) and cutaneous disease (Kirk and Drew, 1938) noted in areas that were endemic for VL. The vectors and animal reservoirs were investigated (Hoogstraal and Dietlein, 1963; Hoogstraal et al., 1963) but it was not certain whether the causative organism was *L. donovani* (Abdalla et al., 1973). Eventually *L. major* and *L. donovani* were identified in cutaneous lesions of patients (El Safi et al., 1991; Elamin et al., 2008).

## Case history

On 24th November 2009 a 26-year-old female, Muganda by tribe, born and raised in Masaka District and since 2007 a resident of Bbosa village on Serinya Island (altitude 1 156 m) in Kalangala District (Figure 1), presented to the Casualty Department of MNRTH with fever, vomiting and diarrhoea for two weeks, perineal sores for two months and skin lesions for eight months. Other than childhood stays in Mutukula Town on the Uganda-Tanzania border, there was no history of travel anywhere outside Central Uganda (Figure 2). On Serinya Island she sold foodstuffs, and there and on neighbouring Islands she engaged in bush and forest clearing, often sleeping outdoors during the excursions. She was aware of two men from the Karamoja sub-Region (Figure 3); one was a fisherman and the other fried 'chapati'. She had never had a blood transfusion or used injections


**Figure 1.** A map showing Masaka District and the Bugala group of Islands in Kalangala District, Mountain Elgon and the span between Kampala City and Amudat Town in north-eastern Uganda. 1, Masaka District; 2, Kalangala District; 3, Kampala City; 4, Mbale Town; 5, Nakapiripirit Town; 6, Amudat Town; 7, Mountain Elgon.

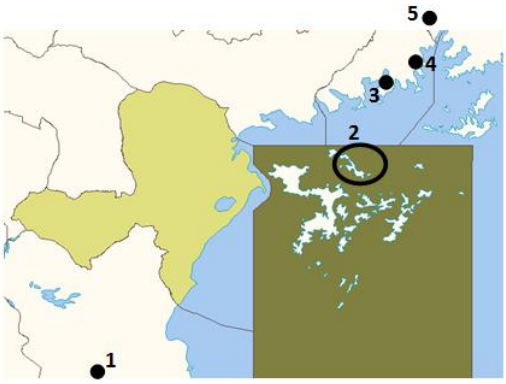


**Figure 2.** A map showing Masaka and Kalangala Districts, Serinya Island and the movement history of the patient. 1, Mutukula Town; 2, Serinya Island; 3, Entebbe Town; 4, Luzira Town; 5, Kampala City.

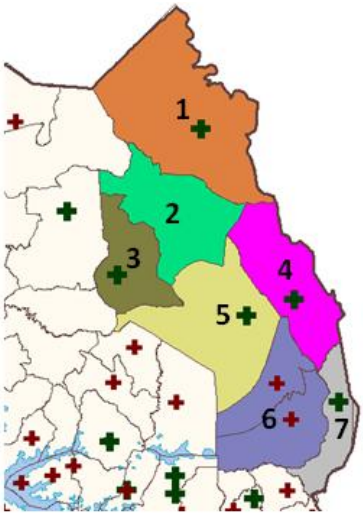


**Figure 3.** A map showing the Districts in the Karamoja sub-Region and the Pokot territory. 1, Kaabong; 2, Kotido; 3, Abim; 4, Moroto; 5, Napak; 6, Nakapiripirit and 7, Amudat (Pokot territory).

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irregularly, and there was no history of chronic alcohol consumption, liver or other organ disease.

While pregnant in 2003 she developed a skin rash on one side of the abdomen, her baby died at two months of age. She tested HIV-positive during postnatal follow-up but was reluctant to seek care. In April 2009 she noticed a 'pimple' on the right cheek, which became swollen and pale. When she attended the Island Health Unit a month later the swelling was red and itching and despite topical applications and tablets it got larger but was painless. Smaller swellings developed on the right arm and leg and a blood report from a mainland acquired immune deficiency syndrome (AIDS) care facility showed total lymphocytes 1 560 /µl, T lymphocytes 1 137 /µl and CD4+ T helper cells 15/µl (reference ranges 1 500-4 000, 690-2 540 and 410-1 590, respectively). She started daily co-trimoxazole prophylaxis therapy (CPT) and counselling sessions in preparation for antiretroviral therapy (ART). There was neither history of ulceration, discharge or bleeding from the skin lesions nor soreness or bleeding from the mouth, throat or nasal space. Six months after the appearance of the facial lesion she developed perineal sores, was seen at the mainland AIDS care facility and with differential diagnoses of Kaposi's sarcoma and cutaneous mycobacterial disease a biopsy of the lesion on the right elbow was taken. A private laboratory in Kampala City, reported *Leishmania* parasites that were seen on microscopic examination of histology sections. The patient was prescribed oral Fluconazole but the skin lesions became more swollen. She developed diarrhoea and vomiting and was referred to MNRTH.

## Case evaluation

Clinical evaluation at MNRTH, eight months since the onset of the skin lesions, revealed a fever of 38°C, dehydration, severe underweight for age, gender and height [height 150 cm, weight 30 kg (42-56 kg), BMI 13.3 (18.5-25 kg/$m^2$)], sparse silky hair, rough unevenly darkened skin, a hoarse voice but intact non-inflamed nasal, buccal and pharyngeal mucous membranes and no jaundice or bleeding tendency. There were 6 firm non-tender skin lesions of two types, a large (3.5 cm) raised well-circumscribed erythematous non-ulcerated granular-surfaced lesion on the right cheek with satellites at the edges, a similar medium-sized (2.5 cm) lesion at the paronychium of the left ring finger and four smaller (1-1.5 cm) smooth erythematous plaques on the extensor surfaces of the right arm, right leg and left thigh (Figure 4 A and B). Punctate raw perineal ulcers oozed serous non-bloody non-foul-smelling fluid. There was bilateral inguinal lymphadenopathy but no enlargement of the head, neck, deltopectoral, axillary, epitrochlear or popliteal lymph nodes.

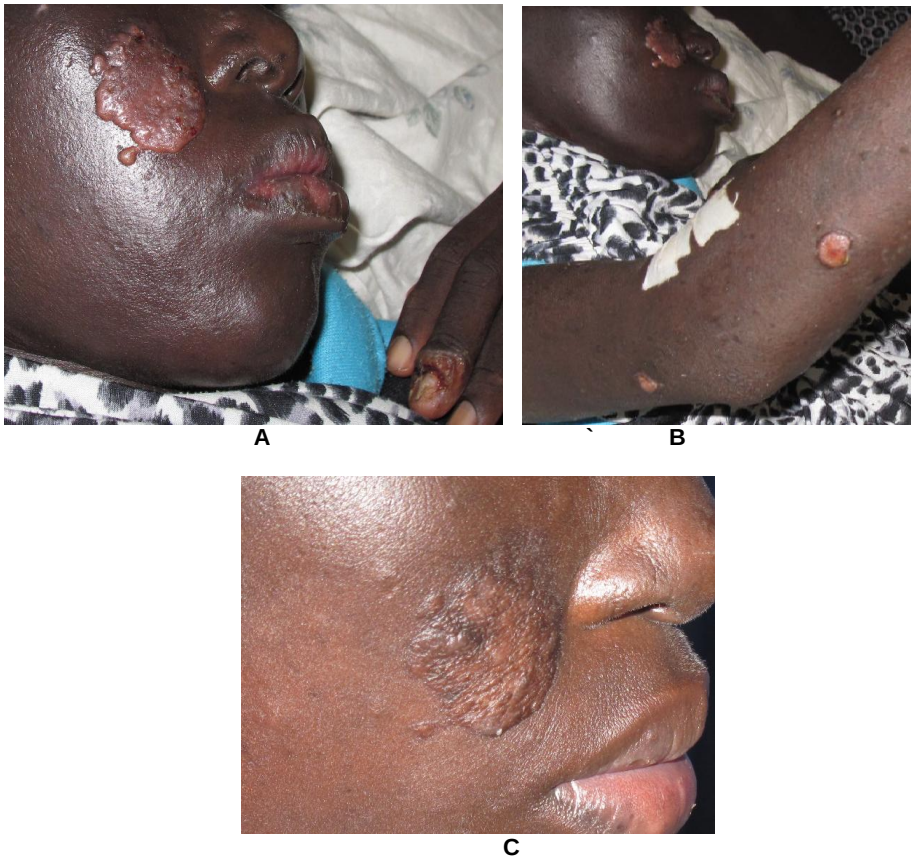


**Figure 4A.** Hyperaemic granular-surfaced patches on the face and at the paronychium of the left ring finger; **B.** Pale smooth-surfaced plaques on the right arm; **C.** Depigmented scar of the facial lesion after treatment with Amphotericin B.

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The chest findings and x-ray report were normal, abdominal examination showed no tenderness or guarding, no liver or spleen enlargement, and an ultrasound scan of the abdomen had reported normal liver, gallbladder, spleen, pancreas and kidneys. The haemogram showed a microcytic anaemia with a haematocrit of 26.5% and haemoglobin of 8.4 g/dl, a leukopenia of 3 600 /µl and neutropenia of 1 000/µl (reference ranges 36-47%, 11.5-16.5 g/dl, 4-11 x $10^3$ /µl and 2-7 x $10^3$ /µl respectively). No malaria parasites had been seen in a finger prick blood smear. There was a hypoalbuminaemia of 28.8 g/l with a normal total proteinaemia of 63.9 g/l (reference ranges 35-50 g/l and 63-83 g/l). Abnormal liver function results were an aspartate aminotransferase (AST) of 77.1 U/l and gamma glutamyl transferase (GGT) of 71.4 U/l (reference ranges 0-40 U/l and 0-53 U/l respectively). Kidney function tests showed low creatinine 38.8 µmol/l and urea 1.6 mmol/l levels (reference ranges 44-106 µmol/l and 2.7-6.4 mmol/l); blood electrolytes (chloride, potassium and sodium) were within normal limits. Microscopic examination of paraffin-embedded Haematoxylin and Eosin-stained skin sections prepared in October 2009 showed an atrophic superficial epidermis, epidermal micro-abscesses but no ulceration, diffuse cellular infiltration of the dermis with granuloma formation, lymphocytosis and heavy aggregation of Leishman-Donovan (LD) bodies in macrophages as well as their dispersal in the extracellular space (Figures 5A, B and C). No smear preparation or parasite culture was done. No leishmanin skin test, antileishmanial antibody detection or lymphoproliferation tests were performed. The Rapid Plasma Reagin (RPR), *Treponema pallidum* haemagglutination assay (TPHA) and hepatitis B surface antigen (HBsAG) had been reported as negative; no test was done for hepatitis C virus (HCV) antibodies. Stool and urinalysis results had been reported as normal.

The case was diagnosed as CL in advanced HIV/AIDS disease with presumed vulval herpes simplex and cryptosporidial gastroenteritis. The clinical staging was four with severe immunosuppression based on the CD4+ T helper cell count below 200/µl (WHO, 2005). She would be rehydrated, given antipyretics and perineal toilet, continue CPT and proceed with the preparation for ART initiation. Antileishmanial treatment would commence after the resuscitation.

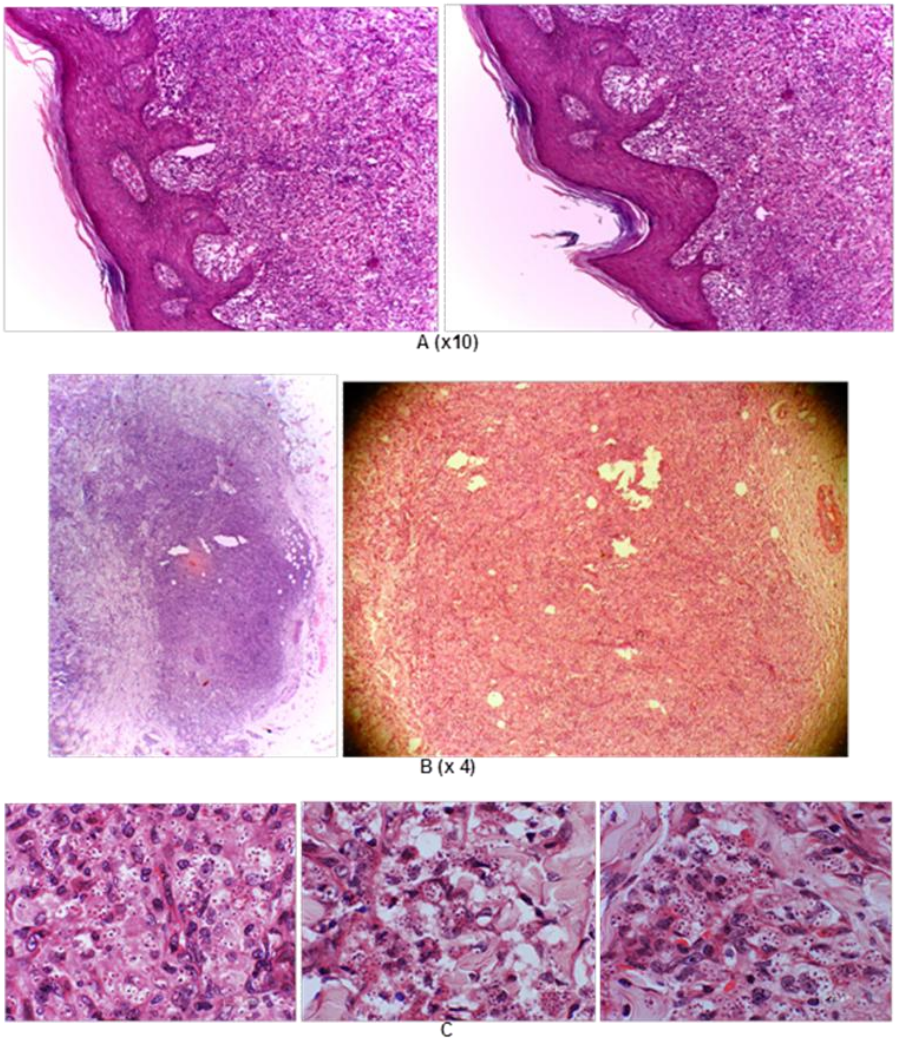


**Figure 5. A.** Atrophic superficial epidermis, micro-abscess formation in the epidermis and inflammatory cell infiltration of the papillary dermis; **B.** Granuloma formation in the reticular dermis; **C.** Lymphocytic infiltrate in the dermis, intracellular and extracellular *Leishmania.*

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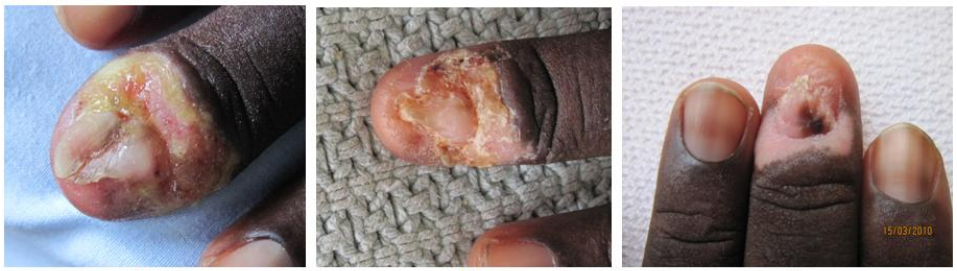
## Case treatment

On the Medical Ward the gastroenteritis eased and the perineal sores dried with warm saline soaks and topical acyclovir while the skin lesions turned grey. There was need for adequate nutrition, the options for antileishmanial treatments were limited and poor response and relapses were anticipated. Since 1998 medication has been provided by Non-Governmental Organisations to Amudat Hospital, the only leishmaniasis treatment centre in the country about 400 km to the north-east of Kampala through Mbale and Nakapiripirit Towns (Figure 1). The fist-line treatment for VL is meglumine antimoniate (Glucantime™) and second-line treatment is Amphotericin B. Generic sodium stibogluconate (sodium antimony gluconate) had just been registered with the National Drug Authority. A pentavalent antimony compound was not desired for the frail patient as adequate monitoring of liver, pancreatic, renal and cardiac function was not certain. Amphotericin B was the alternative, but owing to the high load of immunocompromised patients with life-threatening fungal infections (Kambugu et al., 2008) the assurance of an uninterrupted course was minimal. A donation of Amphotericin B (CIPLA Ltd. Goa, India) was eventually obtained from the Mildmay Centre, MNRTH gave free meals and a bed on the Private Ward and Kampala International Hospital pledged free board and treatment on subsequent hospitalisations. Due to a miscommunication, the patient left Hospital but was traced and re-admitted to the Private Ward on 14th January 2010. The facial lesion had developed an irregular grey scab, the finger lesion was ulcerated and inflamed, the plaques on the limbs were darker and more prominent, the perineal sores were healed and the mucosa were normal. There was no new lymphadenopathy, no liver or spleen enlargement and no parasitological evaluation was done on re-admission.

Management followed criteria for the treatment of VL in patients with risk of relapse (Médecins Sans Frontières-Amsterdam, 1999). Amphotericin B deoxycholate was given intravenously over 8 hours on alternate days at 1 mg/kg/day and concentrations less than 0.1 mg/ml for 15 doses, from 15th January to 12th February 2010. Oral fluids more than four litres and small yellow sweet bananas (ndiizi) rich in potassium were recommended daily. Apart from myalgia and lethargy after the first infusions, the recurrent side-effect was phlebitis at infusion sites. After two weeks of treatment all except the finger lesion had re-epithelialised, this was soaked in warm saline with good result (Figures 6A, B and C).




A B C

**Figures 6.** The healing stages of a digit.

Monitoring showed improving general condition and appetite, increasing body weight, serum albumin and haemoglobin levels while the CD4+ cell count reduced to 1 /µl. The weekly blood smears were negative for malaria parasites, the blood electrolytes remained normal however, the GGT levels remained elevated (88 U/l) and a serum amylase could not be done. Two attempts to start ART failed due to severe vomiting. At discharge the haemoglobin, serum albumin and body weight had increased from 6.7 to 8.5 g/dl, 28.8 to 42.4 g/l and 30 to 35 kg, respectively, and the CD4+ cell count was 5/µl. All lesions had formed depigmented scars, the skin was lighter and smoother (Figure 4C) and the hair firmer and more abundant. A repeat biopsy of the arm scar showed fibrosis, no granulomata or LD bodies; no other parasitological evaluation was performed. She was released to the AIDS care facility on 15th February 2010 to be reviewed monthly at MNRTH for 12 months.

## Case follow-up

At follow-up evaluation one month after discharge, the patient had started highly active antiretroviral therapy (HAART) on 23rd February 2010 and was recovering from malaria. She vomited occasionally, was pale but not jaundiced and had lost 2 kg of body weight. All lesions were healed, there was no new lymphadenopathy, no abdominal tenderness, liver or spleen enlargement and chest findings were normal. There was anaemia, leukopenia and lymphopenia (8.4 g/dl, 1.6 x $10^3$/µL and 0.4 x $10^3$/µL), abnormal liver function tests (ALT 51.4 U/L, AST 84.6 U/L, GGT 292.5 U/L) but normal renal function. She returned to the AIDS care facility with a request for a serum amylase test and a repeat abdominal scan. On 6th April 2010 she was admitted at Entebbe Grade B Government Hospital, with fever, vomiting and dehydration. Four days later her caretakers requested to take her back to Masaka where she died on 14th April 2010.

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**Parasite species identification**

Attempts to identify the parasite species did not yielded results right away. Non-stained unbuffered 10% formalin-fixed paraffin-embedded (FFPE) skin sections were examined at institutions abroad using the polymerase chain reaction (PCR) with probes for *L. major*, *L. tropica* and *L. infantum*. The parasite species was eventually identified as *L. major*.

## DISCUSSION

In Uganda cases of leishmaniasis are increasingly reported from previously unknown areas. For decades, the southernmost tip of the Karamoja sub-Region was known as endemic for VL (McKinnon, 1962; van den Bogaart, 2012). Currently, over 1 million inhabitants of that sub-Region are additionally at risk of the cutaneous disease that was long described in Sudan and Kenya (Kirk, 1938; Manson-Bahr, 1955). The occurrence of CL in central Uganda implies that more millions are at risk and raises questions about an animal reservoir and the source of infection.

Because healing is usually spontaneous in immunocompetent individuals, CL escapes health records and gives little information on population exposure. This patient was referred by an HIV/AIDS care facility to MNRTH due to the unremitting gastroenteritis. Other patients probably present at Skin and Venereal Diseases Clinics and Leprosy treatment centres but clinical suspicion of CL tends to be low in non-endemic areas (Bari and Rahman, 2006). Detection depended on the patient's improved health-seeking behaviour and diagnosis associated with opportunistic infections in HIV and AIDS (Uganda Ministry of Health, 2005), and described a typical trend of events in our setting. Due to chances of transmitting blood-borne infectious organisms, the slit-skin procedure usually performed by the laboratory technician to detect *Mycobacterium leprae* is less encouraged, making microbiological diagnosis of CL less probable. A biopsy is readily performed by the clinician and it allows histological evaluation of features suggestive of CL (Safaei et al., 2002). However, parasites are less easily detected in tissue sections (Abdalla et al., 1973) and the number is very low in chronic CL (Laskay et al., 1995). Touch preparation and aspiration are performed more for cytopathological than microbiological analysis, and parasite culture is not feasible in most Health Units. Overall, case detection would depend largely on clinical suspicion of non-healing skin lesions.

There were two types of skin lesions, the larger hyperaemic granular-surfaced patches and the smaller pale smooth-surfaced plaques, all of which may have resulted from multiple insect bites rather than parasite dissemination. Lesions were distinct from the perineal sores that healed before antileishmanial treatment and that were particularly noted due to the possibility of genital acquisition or transmission of *Leishmania* (Symmers, 1960). It is noteworthy that in contrast to *L. tropica*, *L. major* which was identified in this case does not normally cause human VL (WHO, 2010a). However, parasite dissemination was a possibility due to the profound immunosuppression, failure of spontaneous healing after ten months and heavy parasitosis evident on histology. The lack of a generalised lymphadenopathy or liver or spleen enlargement spoke against VL while laboratory findings indicated multiple organ involvement. The anaemia, neutropenia and lymphopenia suggested bone marrow malfunction but this recovered to a degree after antileishmanial therapy. The hypoalbuminaemia, alternating AST and ALT and persistently elevated GGT implied a hepatocellular process, not severe enough to cause jaundice. Failure of liver function to normalise implicated alternative protozoal, viral and chemical causes of hepatic, biliary or pancreatic disease. Though not supported by laboratory or radiological findings, clinical symptoms suggested a pancreatitis possibly present since the initial admission to MNRTH, that may have been HIV-related (Rizzardi et al., 1997), consequent to cryptosporidial enteritis (Hawkins et al., 1987), associated with herpes simplex viral infection (Konstantinou, 2009) or induced or exacerbated by HAART (Kaurich, 2008).

Of particular interest was the source of infection. The equine leishmaniasis reported at Entebbe (Richardson, 1926) was probably presumed to be due to *L. major* on ecological criteria. The Ssesse Islands, fifteen minutes by motor boat and four hours by wooden boat from the mainland are inhabited by fishing communities. Due to the relative seclusion and increased economic activity, they attract people from all over Uganda. The occurrence of CL in a migrant of barely two years highlights the vulnerability of non-immune persons who move to areas where *Leishmania* are transmitted. Because the patient had not travelled outside Uganda suggested an indigenous infection and the presence of active skin lesions indicated active transmission at the time. Infection may have been contracted on Serinya or neighbouring Buvu, Kibi, Banda and Kitobo Islands where they felled trees or even at mainland Entebbe, Abayitababiri or Luzira where she briefly stayed. Alternatively, the parasite may have been introduced from neighbouring Kenya or Sudan where *L. major* is known (Sang et al., 1993c; El Safi et al., 1991). Lastly, there was a possibility of *L. aethiopica* since transmission could occur on the Uganda side of Mt. Elgon at heights that are uninhabited.

Parasite identification was essential since *L. aethiopica, L. major*, *L. tropica, L. infantum* and *L. donovani* cause CL in the Old World (WHO, 2010a). The species can be identified by isoenzyme analysis of cultured promastigotes (Kreutzer and Kristensen, 1980) or PCR (Laskay et al., 1995) but the methods are not readily available. Analysis of FFPE tissue sections was done in three institutions abroad before a positive result was obtained. In our health institutions, clinical samples are preserved in formol-saline consisting of 4% formaldehyde in 0.9% sodium chloride, delivery of the specimen to the pathologist being recommended within 48 hours. However for molecular analysis, use of neutral buffered formalin is recommended to avoid protein-protein cross-links, DNA damage and suboptimal PCR amplification (Lehman et al., 2001; Wu et

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al., 2002) and for nucleic acid extraction, fixation need not exceed one day (Inoue et al., 1996).

This case report agrees with the WHO map showing *L. major* as endemic on the mainland north of the Ssesse Islands. It is not clear where the equine leishmaniasis was acquired from earlier however, zoonotic transmission would give a low incidence of human disease that is not recognised by immunocompetent persons whose lesions heal spontaneously. This human case may be consequent to many that are missed or misdiagnosed, thus the training of health workers is essential. Suspicion of skin and mucosal lesions should be accompanied by a detailed history of the person's movement and adequate knowledge of the geography of the disease is essential. Surveys should be conducted to detect other cases on the Islands, determine where human infection takes place and to identify the vectors and animal reservoir. Because infected persons endanger the community and the potential animal reservoir, awareness should be created in the general population to enable early diagnosis and plans should be made to protect humans against the vector.

## Authors' contributions

ES treated the patient, organised medical supplies, sent samples for analysis and prepared the manuscript. ED arranged the hospital abode, additional medication, nursing services and inpatient feeding. JA arranged the out-of-hospital care and counselling services. RO examined the tissue samples and made the diagnosis of cutaneous leishmaniasis.


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**Competing interests:** None


## Ethical approval

The patient gave informed written consent for treatment on admission at Mulago National Referral and Teaching Hospital in Kampala. The Director General of Health Services, Uganda Ministry of Health gave permission to publish this case report.

## REFERENCES


Abdalla RE, Ali M AI, Wasfi El Hassan AM (1973). Cutaneous leishmaniasis in the Sudan. Trans. R. Soc. Trop. Med. Hyg. 67(4): 549 – 559.

Andersen OS (1964). Dermal leishmaniasis in a patient with leprosy in western Tanganyika. East Afr. Med. J. 41(10): 471

Bari A, Rahman S (2006). Correlation of clinical, histopathological and microbiological findings in 60 cases of cutaneous leishmaniasis. Indian J. Dermatol. Venereol. Leprol. 72: 28 – 32.

Bennett SCJ (1935). Equine cutaneous leishmaniasis: Treatment with berberine sulphate. J. Comp. Pathol. Ther. 48: 241 – 243.

Chance ML, Schnur LF, Thomas SC, Peters W (1978). The biochemical and serological taxonomy of *Leishmania* from the Aethiopian zoogeographical region of Africa. Ann. Trop. Med. Parasitol. 72(6): 533 – 542.

Christopherson JB (1914). On a case of naso-oral leishmaniasis (corresponding to the description of Espundia) and on a case of oriental sore, both originating in the Anglo-Egyptian Sudan. Ann. Trop. Med. Parasitol. 8: 485 – 496.

Clyde DF (1964). Leishmaniasis in Tanganyika. East Afr. Med. J. 41(10): 472

Elamin EM, Guizani I, Guerbouj S, Gramiccia M, El Hassan AM, Di Muccio T, Taha MA, Mukhtar MM (2008). Identification of *Leishmania donovani* as a cause of cutaneous leishmaniasis in Sudan. Trans. Roy. Soc. Trop. Med. Hyg. 102(1): 54 – 57.

El-Safi SH, Peters W, El-Toam B, El-Kadarow A, Evans DA (1991). Studies on the leishmaniases in the Sudan. 2. Clinical and parasitological studies on cutaneous leishmaniasis. Trans. R. Soc. Trop. Med. Hyg. 85(4): 457 – 464.

Gigase P, Moens F, van Emelen J, van Marck E, van Mullem J (1978). Autochthonous visceral leishmaniasis in Zaire. Ann. Soc. B. Méd. Trop. 58: 235 – 240.

Hawkins SP, Thomas RP, Teasdale P (1987). Acute pancreatitis: a new finding in cryptosporidium enteritis. Br. Med. J. (Clin Res Ed). 294(6570): 483 – 484.

Hoogstraal H, Dietlein DR (1963). Leishmaniasis in the Sudan Republic 9. Ecological relationships of sandfly species and *Leishmania* infection. Am. J. Trop. Med. Hyg. 12: 165 – 174.

Hoogstraal H, van Peenen PFD, Reid TP, Dietlein DR (1963). Leishmaniasis in the Sudan Republic 10. Natural infection in rodents. Am. J. Trop. Med. Hyg. 12: 175 – 178.

Inoue T, Nabeshima K, Kataoka H, Koono M (1996). Feasibility of archival non-buffered formalin-fixed and paraffin-embedded tissues for PCR amplification: An analysis of resected gastric carcinoma. Pathology International 46(12): 997 – 1004.

Kadaro AY, Ghalib HW, Ali MS, Eltoum I, Ismail A, Gaafar A, Kemp M, Kordofani AAY, Reed SG, El-Hassan AM, Kharazmi A, Hag-Ali M, Mustafa MD (1993). Prevalence of cutaneous leishmaniasis along the Nile River North of Khartoum (Sudan) in the aftermath of an epidemic in 1985. Am. J. Trop. Med. Hyg. 48(1): 44 – 49.

Kambugu A, Meya DB, Rhein J, O'Brien M, Janoff EN, Ronald AR, Kamya MR, Mayanja-Kizza H, Sande MA, Bohjanen PR, Boulware DR (2008). Outcomes of cryptococcal meningitis in Uganda before and after the availability of HAART. Clin. Infect. Dis. 46(11): 1694 – 1701.

Kaurich T (2008). Drug-induced acute pancreatitis. Proc. (Bayl. Univ. Med. Cent.) 21(1): 77 – 81.

Kirk R, Drew CB (1938). Preliminary notes on dermal leishmaniasis in the Anglo-Egyptian Sudan. Trans. R. Soc. Trop. Med. Hyg. 32(2): 265 – 270.

Kirk R (1938). Primary cutaneous sore in a case of kala-azar. Trans. R. Soc. Trop. Med. Hyg., 32(2): 271 – 272.

Konstantinou GN, Liatsos CN, Patelaros EG, Karagiannis SS, Karnesis LI, Mavrogiannis CC (2009). Acute pancreatitis associated with herpes simplex virus infection: report of a case and review of the literature. Eur. J. Gastroenterol. Hepatol. 21: 114 – 116.



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Kreutzer RD, Kristensen HA (1980). Characterisation of *Leishmania* species by isoenzyme electrophoresis. Am. J. Trop. Med. Hyg. 29(2): 199 – 208.
Kungu A, Mutinga MJ, Ngoka JM (1972). Cutaneous leishmaniasis in Kenya. East Afr. Med. J. 49(3): 458 – 151.
Laskay T, Mikó TL, Negesse Y, Solbach W, Röllinghoff M, Frommel D (1995). Detection of cutaneous *Leishmania* infection in paraffin-embedded skin biopsies using the polymerase chain reaction. Trans. R. Soc. Trop. Med. Hyg. 89(3): 273 – 275.
Lehmann U, Kreipe H, 2001. Real-time PCR analysis of DNA and RNA extracted from formalin-fixed and paraffin-embedded Biopsies. Methods 25(4): 409 – 418.
Lemma A, Foster WA, Gemetchu T, Preston PM, Bryceson ADM, Minter DM (1969). Studies on leishmaniasis in Ethiopia. I. Preliminary investigation in the epidemiology of cutaneous leishmaniasis in the highlands. Ann. Trop. Med. Parasit. 63(4): 455 – 472.
Manson-Bahr PEC (1955). A primary skin lesion in visceral leishmaniasis. Nature 175(4453): 433 – 434.
McKinnon JA (1962). Kala-azar in the upper Rift Valley of Kenya. Part I Background and discovery of the disease. J. Trop. Med. Hyg. 65(3): 51 – 63.
Mebrahtu Y, Lawyer P, Githure J, Kager P, Leeuwenburg J, Perkins P, Oster C, Hendricks LD (1988). Indigenous human cutaneous leishmaniasis caused by *Leishmania tropica* in Kenya. Am. J. Trop. Med. Hyg. 39(3): 267 – 273.
Mebrahtu YB, Van Eys G, Guizani I, Lawyer PG, Pamba H, Koech D, Roberts C, Perkins PV, Were JB, Hendricks LD (1993). Human cutaneous leishmaniasis caused by *Leishmania donovani s.l.* in Kenya. Trans. R. Soc. Trop. Med. Hyg. 87(5): 598 – 601.
Médecins Sans Frontières-Amsterdam (1999). Manual for the diagnosis and treatment of visceral leishmaniasis (kala-azar) under field conditions. Public Health Department.
Muigai R, Githure JI, Gachichi GS, Were JBO, Leeuwenburg J, Perkins PV (1987). Cutaneous leishmaniasis caused by *Leishmania major* in Baringo District, Kenya. Trans. R. Soc. Trop. Med. Hyg. 81(4): 600 – 602.
Mutinga MJ, Mngola EN (1974). Alternate treatment of cutaneous leishmaniasis. East Afr. Med. J. 51(6): 68 – 78.
Mutinga MJ (1975a). The animal reservoir of cutaneous leishmaniasis on Mount Elgon, Kenya. East Afr. Med. J. 52(3): 142 – 151.
Mutinga MJ (1975b). *Phlebotomus* fauna of Mount Elgon, Kenya in the cutaneous leishmaniasis focus of Mount Elgon, Kenya. East Afr. Med. J. 52(6): 340 – 347.
Pharoah PDP, Ponnighaus JM, Chavula D, Lucas SB (1993). Two cases of cutaneous leishmaniasis in Malawi. Trans. R. Soc. Trop. Med. Hyg. 87(6): 668 – 670.
Prévot H, Parmentier R, Bounameaux Y (1968). Un cas de Leishmaniose viscéral au Congo. Ann. Soc. B. Méd. Trop. 48(4): 421 – 427.
Richardson UF (1926). A probable case of equine leishmaniasis. Trans. R. Soc. Trop. Med. Hyg. 19(7): 411
Rizzardi G P, Tambussi G, Lazzarin A (1997). Acute pancreatitis during primary HIV-1 infection. N. Engl. J. Med. 336(25): 1836 – 1837.
Safaei A, Motazedian MH, Vasei M (2002).Polymerase chain reaction for diagnosis of cutaneous leishmaniasis in histologically positive, suspicious and negative skin biopsies. Dermatol. 205(1): 18 – 24.
Sang DK, Okelo GBA, Ndegwa CW, Ashford RW (1993). New foci of cutaneous leishmaniasis in central Kenya and the Rift Valley. Trans. R. Soc. Trop. Med. Hyg. 87(6): 629 – 632.
Sang DK, Njeru WK, Ashford RW (1994). A zoonotic focus of cutaneous leishmaniasis due to *Leishmania tropica* at Utut, Rift Valley Province, Kenya. Trans. R. Soc. Trop. Med. Hyg. 88(1): 35 – 37.
Symmers WStC (1960). Leishmaniasis acquired by contagion: A case of marital infection in Britain. Lancet 1: 127 – 132.
Uganda Ministry of Health (2005). Training manual for the management of opportunistic infections in HIV/AIDS for operational level health workers. http://www.aim.jsi.com/Docs/hiv_mgt_opp_infections_training_manual.pdf. STD/AIDS Control Programme [accessed on 30th November 2010].
van den Bogaart E, Berkhout MMZ, Adams ER, Mens PF, Sentongo E, Mbulamberi DB. Straetemans M, Schallig HDFH, Chappuis F (2012). Prevalence, features and risk factors for malaria co-infections amongst visceral leishmaniasis patients from Amudat Hospital, Uganda. PLoS Negl. Trop. Dis. 6(4): e1617. doi:10.1371/journal.pntd.0001617
Wenyon CM (1926). Laboratory Meeting on 19th November 1925. Trans. R. Soc. Trop. Med. Hyg. 19(7): 281
Wu L, Patten N, Yamashiro CT, Chui B (2002). Extraction and amplification of DNA from formalin-fixed paraffin-embedded tissues. Appl. Immunohistochem. Mol. Morph. 10(3): 269 – 274.
WHO African Region (2005). Interim WHO clinical staging of HIV/AIDS and HIV/AIDS case definitions for surveillance. WHO/HIV/2005.02
WHO (2008). Report of the consultative meeting on cutaneous leishmaniasis. WHO Headquarters, 30 April - 2 May 2007, WHO/HTM/NTD/IDM/2008.7
WHO (2010a). Control of the leishmaniases. WHO Tech. Rep. Ser. 949: 1-186
WHO (2010b). Essential leishmaniasis maps: Cutaneous leishmaniasis. http://www.who.int/entity/leishmaniasis /leishmaniasis_maps/en [accessed on 30th November 2010].
Wykoff DE, Barnley DR, Winn MM (1969). Studies on kala-azar in Uganda - entomological observations. East Afr. Med. J. 46(4): 204 – 207.

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