Emergence of visceral leishmaniasis in Sri Lanka: a newly established health threat

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ABSTRACT

Background: Sri Lanka is a new focus of human cutaneous leishmaniasis caused by a genetic variant of usually visceralizing parasite *Leishmania donovani*. Over 3000 cases have been reported to our institution alone, during the past two decades. Recent emergence of visceral leishmaniasis is of concern.

Methods: Patients suspected of having visceral leishmaniasis ($n = 120$) fulfilling at least two of six criteria (fever > 2 weeks, weight loss, tiredness affecting daily functions, splenomegaly, hepatomegaly and anemia) were studied using clinic-epidemiological, immunological and haematological parameters. Seven cases (four progressive, treated (group A) and 3 non-progressive, potentially asymptomatic and observed (group B) were identified. Clinical cases were treated with systemic sodium stibogluconate or amphotericin B and all were followed up at the leishmaniasis clinic of University of Colombo for 3 years with one case followed up for 9 years.

Results: All treated cases responded well to anti leishmanial treatment. Relapses were not noticed. Clinical features subsided in all non-progressive cases and did not develop suggestive clinical features or change of laboratory parameters. Visceral leishmaniasis cases have been originated from different districts within the country. Majority had a travel history to identified local foci of cutaneous leishmaniasis.

Conclusion: Visceral leishmaniasis is recognized as an emerging health threat in Sri Lanka. At least a proportion of locally identified strains of *L. donovani* possess the ability to visceralize. Apparent anti leishmanial sensitivity is encouraging. Timely efforts in disease containment will be important in which accurate understanding of transmission characteristics, increased professional and community awareness, improved diagnostics and availability of appropriate treatment regimens.

KEYWORDS

Visceral leishmaniasis;
Leishmania donovani;
clinical; emerging; parasitic;
Asia

Introduction

Leishmaniasis, caused by multiple species of *Leishmania* draws global attention due to its high morbidity and/or mortality rates associated with different clinical forms [1]. WHO led VL elimination drive in Indian subcontinent has faced many difficulties [2] with timelines to achieve targets further extended [3–5]. Sri Lanka is a new focus of cutaneous leishmaniasis caused by a genetic variant of the visceralizing parasite *Leishmania donovani* [6–8]. Case distribution is widening with regionally different risk factors associated with the infection [9,10]. Only a single MCL and single VL case have been reported so far while CL remains as the main clinical picture up to this date [11–13]. Need for urgent action to contain the disease has been highlighted on several occasions [14]. Though considerable amount of work on CL has been carried out that include the description of its clinical profile [6,15,16] and diagnostic clinical markers [13]

with formulation of the National action plan for disease control [17], disease awareness and information on the burden of VL still remain poor. Case detection and management are considered the main control strategies for *L. donovani* control. Local information is necessary for evidence based case management and disease control in Sri Lanka. Epidemiological, clinical, immunological and haematological parameters of indigenous cases of VL were studied and followed up for a minimum period of 3 years.

Methodology

Passive case detection

Diagnostic facility for VL was established in University of Colombo in the Faculty of Medicine's Department of Parasitology (UCFM) in year 2004. Patients referred from major hospitals ($n = 120$) with ≥ 2 inclusion criteria

(fever >2 weeks, weight loss, tiredness affecting daily functions, splenomegaly, hepatomegaly and anemia), [18], were included in the study after informed consent.

Clinico-epidemiological data, blood (2 ml) and bone marrow (BM, 2 ml) were obtained from all patients. Light microscopy (LM) and culture on BM were done according to previously established protocols [19]. PCR was performed on BM of negative cases [20]. rK 39 assay was carried out on sera of all patients. Clinical, immunological (rk 39), hematological (Hb, TBC) and other laboratory parameters (liver enzymes, total proteins, USS) were monitored annually up to a minimum of 3 years and bi-annually for 9 years in 1 case.

Operational definitions

Likely place of infection acquisition (LPIA); Leishmaniasis prevalent areas (central CL database [2001–2015]) to which patient had mostly visited /stayed during the preceding three years of illness.

Incubation time between infection and clinical illness [Infection-illness time (IIT)]; approximate time from possible exposure date (last visit to LPIA) to clinically apparent illness calculated based on travel history.

Results

Case grouping and trends analysis

A total of 120 patients (1–84 years age range, male: female ratio 48:52%) were recruited. Majority of the referrals (>70%) was from less-CL prevalent Western province in Sri Lanka (central data base, UCFM). Non-specific clinical features of VL were present in different proportions in the study population, with fever being the commonest (50%), (Table 1). *L. donovani* local infection was confirmed in 7/120 by travel history and BM positivity by investigations. Each of these case were analyzed in detail and described below.

There were two males and five females (24–53 years age) in the laboratory-confirmed VL group. Four (4/7) patients progressed clinically and were treated with anti leishmanials, while two of them tested positive for rK39. Other 3 cases were non progressive and were observed. None of the laboratory-confirmed patients had VL as a possibility in the initial differential diagnosis.

Table 1. Prevalence of different clinical manifestations considered for VL screening in the study group (n = 120).

Clinical/laboratory feature	Number	Percentage
Fever	60	50.0
Hepatomegaly	25	20.8
Splenomegaly	19	15.8
Pallor	18	15.0
Lymphadenopathy	8	6.6
Reduced Hb levels	8	6.6
Jaundice	6	5.0
Loss of weight	6	5.0

Patients presented from different locations (Figure 1) while two of them had insignificant overseas travel history (Case 2 to Northern India after appearance of clinical features and Case 7 to Southern India before and after illness). All except one either had a travel history to a main local CL focus or they were at least residents from a CL prevalent district (Figure 2). Approximate incubation time was variable (3 months- to many years), (Table 2). Approximate time taken from first appearance/suspicion of one or more clinical features to the point of laboratory diagnosis was highly variable (3 months–6 years).

Group A: cases progressing to full blown clinical disease

Case 1

A 37 year old female housewife from North-Central province (location C3, Figure 2) presented with progressive abdominal discomfort for 1 year and fever for 3 months. There was no loss of appetite, or abdominal pain. She was apparently healthy looking and active, but was obviously pale, emaciated, afebrile, abdomen was markedly distended, had marked hepato-splenomegaly and axillary lymphadenopathy. There was no jaundice, or skin lesions suggestive of post kala-azar dermal leishmaniasis (PKDL).

Patient was first investigated for malaria and hematological disorders. Failing this, she was referred for 'exclusion of leishmaniasis'. LM of BM and culture were positive for *Leishmania* sp. and serum was positive for anti rK 39 antibodies. PCR was not available at that time. High ESR (141 mm 1st hour), low haemoglobin (7 g/dl), elevated liver enzymes (AST 88.8u/l, ALT 28 u/l) and low total blood cell counts (3.1×10^6 /l) were detected. She was treated with IV sodium stibo-gluconate (SSG) 70 mg daily for a period of 28 days. On discharge, she was afebrile and feeling well. Two months later, Hb and white cell counts improved (10.6 g/dl, 3.87×10^6 /l respectively). Three months later, both SGPT and SGOT levels showed a reduction (52 [8–39] u/l, 37 [3–44] u/l).

Follow up: She was apparently healthy at the end of first year and rK 39 was negative. Hb and liver enzyme levels were normal after two years following treatment. Body weight increased by 2 kg. rK 39 remained negative 4 years later. She was followed up for a period of 9 years. Clinical and laboratory profiles remain normal and patient remains apparently healthy without any hospital admissions.

Travel history: Patient is from location C3, a CL prevalent area and had travelled to multiple places in Southern Sri Lanka (location B) preceding the clinical disease.

Case 2

A 50-year-old female from Colombo district (location A1, Figure 2) presented with loss of appetite, progressive tiredness and progressive weight loss (approx. 11 kg)

Figure 1. Temporal distribution of residences and LPIA of cases of VL in Sri Lanka over space. Source: The Authors.

*Negligible visits; visits either following illness point, visits to non-case reported areas visits after onset of clinical features, **LPA; Likely place of infection acquisition

over 24 months. Examination revealed pallor, emaciation, splenomegaly and a hyper-pigmented papular rash over the exposed body areas. There was no fever (at the time of examination), jaundice or lymphadenopathy. Her full blood count report showed pancytopenia. Detailed history revealed splenomegaly by USS 6 years back during

routine medical checkups, but no interventions done as there were no other clinical symptoms.

She had pancytopenia (white cells, $1.1 \times 10^9/l$, platelet count; $42 \times 10^9/l$, severe normocytic normochromic anemia (hemoglobin 4.5 g/dl), non-elevated C-reactive protein (5.8 mg/dl) and normal liver enzyme levels (ALT

Figure 2. LPIA and residence distribution of VL in comparison to incidence of CL (2005–2013). Source: The Authors.

Table 2. Temporal distribution of infections across LPIAs of patients.

21 g/dl, AST 10 g/dl). USS abdomen further confirmed massive splenomegaly.

BM was positive for *Leishmania spp.* by both microscopy and culture. Repeated rK 39 assay remained negative. Diagnosis of VL was established. She was treated with liposomal amphotericin (brand Fungizone) 3 mg per kg body weight for 14 days (1 cycle) into 3 cycles. On completion of treatment, fever and body rashes subsided. Six months later she continued to have hepato-splenomegaly, anemia and pancytopenia. Histology of spleen revealed a marginal zone lymphoma. Splenectomy was done. Following splenectomy she became completely asymptomatic and her blood counts normalized. As it was a low grade lymphoma with no other lymphadenopathy it was decided to observe her without any chemotherapy.

Follow up: One year following splenectomy, HB, full blood count, platelets and serum protein profiles tested normal with raised liver enzymes. After 3 years, she remained asymptomatic, has gained weight (5 kg), and haematological investigations (FBC, rK39) were normal. Six months later she presented with generalized lymphadenopathy and constitutional symptoms. Relapse of lymphoma with high grade transformation to a diffuse large B cell lymphoma was diagnosed and succumbed shortly thereafter.

Travel history: She is from location A1 and frequently visited to her original home at location B2, and location B5 on pilgrimage (Figure 2) for >3 decades and had multiple visits to many places in Northern disease foci (location C, Figure 2). She had a single visit to VL endemic states of Northern India (3 weeks) and Nepal (4 days) 2 years prior to the current hospital admission.

Case 3

A 24 year old male soldier from Colombo (location A2) presented with intermittent fever and frontal headache of 1 month. There was myalgia, arthralgia, fatigue, wasting and loss of appetite. He was ill-looking, wasted, febrile and weighted 59 kg. There was no pallor, hepato-splenomegaly, jaundice, lymphadenopathy or skin lesions suggestive of PKDL.

He was investigated for malaria, TB, HIV, JE and other systemic disorders by urine full report, liver function tests, ESR, CSF, blood cultures and serum creatinine levels. He was empirically treated with IV antibiotics and antivirals, without improvement. Later on, BM examinations revealed *Leishmania sp.* parasites by light microscopy and culture and chronic inflammatory process by histology. Blood was negative for rK 39 assay.

Liver enzymes were elevated (ALT 88.3 u/l, [10–40], AST 62.7 u/l, [13–31]). Total blood cell count (9.1×10^3

with 57.9% neutrophils, 21.7% lymphocytes and 11.6% eosinophils), platelet count ($310 \times 10^3/\text{ul}$) and Hb (3.4 g/dl) were normal. He was treated with IV sodium stibogluconate (SSG) 800 mg, daily for 26 days. On completion of the treatment, Hb remained at 13.1 g/dl, liver enzymes showed a reduction (SGOT 33 u/l, SGPT 49 u/l). WBC count was 6980 /ml. He was given oral miltefosine 100 mg daily for another 24 days.

Follow up: Post treatment (nearly 2 months after starting anti-leishmanial drugs) liver enzyme and full blood count were normal, rK 39 remained negative. Weight improved gradually over a year. After 3 years, he remained asymptomatic (Table 3) and haematological investigations, USS abdomen were normal. rK 39 done annually remained negative. Patient remains asymptomatic and healthy without any hospital admissions and carries out military physical training activities as usual up to date.

Travel history: He spent most of his time in Northern Sri Lanka (location C1, C2, Figure 2) as a soldier for the past 9 years. He travelled to his home in location B3 almost monthly.

Case 4

A 41 year old male (location F) presented with high nocturnal fever of 3 weeks. There was loss of appetite, tiredness, myalgia and 5 kg weight reduction over 2 months prior to admission. He was ill looking, febrile and spleen was moderately enlarged. Pallor, jaundice, hepatomegaly, lymphadenopathy or skin lesions suggestive of PKDL were not detected.

CT scan of abdomen confirmed splenomegaly and absence of lymphadenopathy. There was pancytopenia (WBC $1.83 \times 10^9/\text{l}$), low hemoglobin (6.1 g/l), high ESR (147 mm 1st hour), high CRP (105 mg/l) and elevated liver enzymes (ALT 89 [$1.83 \times 10^9/\text{l}$], AST 65 [u/l]). He was negative for malaria and TB and was empirically treated with IV antibiotics without success. Peripheral blood smear was suggestive of a hairy cell leukaemia which was later confirmed by immune-phenotyping by flow cytometry. Histology of the bone marrow biopsy was consistent with Hairy-cell leukaemia and the microscopy and culture were positive for *Leishmania* sp. rK 39 assay was negative. He was treated with IV sodium stibogluconate (SSG) for 28 days. On completion of the treatment, Hb remained at 8.1 mg/dl, liver enzymes showed a reduction. WBC count was 3.4/ml. He was treated with 7 days of intravenous cladribine as for Hairy-cell leukaemia and IV sodium stibogluconate (SSG) for 28 days. Following treatment, Hb remained at 8.1 mg/dl, liver enzymes showed a reduction. WBC count was 3.4/ml.

Follow up: At post treatment 6 months, patient remained asymptomatic and rK 39 negative with no evidence of hairy cell leukaemia. This patient had been followed up for three years up to this date. At the end of three years, laboratory parameters were within normal range and patient remains asymptomatic (Table 4). USS abdomen is not available.

Table 3. Clinical profile and trends of clinically progressive and treated cases of VL.

Follow ups	Pre treatment				Post Treatment				First year				Second year				Latest follow up*			
	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
Case no.	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
Noticeable weight loss	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Fatigue	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
LOA	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Fever	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Pallor	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Hepatomegaly	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Splenomegaly	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Weight gain	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Dark skin/skin rashes	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓

*9 years in case 1, 3 years in cases 2–4.

Table 4. Laboratory profile and trends of clinically progressive and treated individuals.

Follow up visits		Pre treatment				Post treatment				End of 12 months				End of 24 months				Last follow up*			
Case no.		1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
Lymph node biopsy		+				-				-											
Bone marrow biopsy		+	+	+	+					-											
rK39		+	-	+	-					-	-	-	-	-	-	-	-	-	-	-	-
Hemoglobin g/dl																					
Liver	ALT																				
	AST																				
WBC count 10 ⁹ /l																					

Travel history: He is from location F (Figure 2) who stayed at location B4 and travelled in the same district for employment purposes 1 year prior to the illness. He also had a visit to location D, one month prior to the onset of illness.

Group B: locally acquired infections with non-progressive (potentially asymptomatic) clinical profile

Case 5

A 53 year old female from non- endemic, location A2 (Figure 2) presented with PUO for 2 months. No myalgia, tiredness or emaciation was noticed. On examination, she was not ill looking, not pale or icteric. There was no hepato-splenomegaly. Hemoglobin and USS abdomen were normal. There was mild thrombocytopenia. Salmonella agglutination test was positive. BM showed reactive changes of chronic infection and inflammation. Culture of BM revealed presence of *Leishmania* sp. amastigotes. rK 39 assay was negative.

She was treated as a case of typhoid fever and no anti-leishmanials were given. Follow up annually and at 1.5 and 3 years revealed no clinical VL, hematological parameters and USS abdomen was normal. rK 39 assay was negative. Patient remains apparently healthy.

Travel history: She had frequent visits to her previous home in CL reported areas in Southern Sri Lanka (location Bs) and single visits each to locations C 1 and G during the preceding 12 months.

Case 6. Seventy-five year old female from location A1 presented with fever, tiredness and lethargy of 2 weeks duration. There was no pallor, jaundice or hepato- splenomegaly. Blood cell counts were normal. Microscopy and culture of BM was positive for *Leishmania* sp. rK39 assay was negative. Meanwhile she was empirically treated with antibiotics and fever settled. rK 39 was negative, FBC and USS abdomen were normal at 1 year follow up. At completion of three years she remains clinically normal. Laboratory profile (Hb, blood cell counts, USS abdomen) is also normal.

Travel history: She had a visit 1 year ago to leishmaniasis endemic site in Northern India and multiple visits to location B4, last visit being 6 months prior to illness.

Case 7

Female of 28 years from location A2 presented with history of fever, chills and rigors over one month. There was no myalgia, pallor, skin rashes or clinical hepato-splenomegaly. Initial differential diagnosis including melioidosis, rickettsia, dengue, TB and mycoplasma, hepatitis and HIV infections were excluded. Full blood count, Hb, ESR, CRP, Urine culture, blood culture, stool culture and CT scan of chest were normal. Empirical antibiotics were started. BM cultures revealed *Leishmania sp.* Infection. USS abdomen revealed mild hepato-splenomegaly. rK 39 assay was negative.

Patient responded to antibiotic therapy and kept under close observation. At 3 year follow up patient was apparently healthy, had gone through a normal pregnancy and delivery, blood cell counts and USS abdomen revealed no abnormalities. rK 39 assay results not available.

Travel history: She is a resident in location A2 during proceeding 1 year and in Central province (location E2) since birth. She had travelled to Madras and Chennai in India 3 years ago. These areas had no published cases of leishmaniasis to the knowledge of authors. She had travelled to location E (CL case reported area in Central highlands, Sri Lanka) frequently for past several years.

Cutaneous manifestations

None of the above cases had a past history suggestive of CL skin lesions or manifestations suggestive of PKDL. Only one patient (case 2) had a macular, generalized skin rash which resolved spontaneously during anti-leishmanial therapy.

Concurrent illnesses

Hairy cell leukemia, typhoid, lymphoma and diabetes were identified as co illnesses in 4 patients. However it was not clear whether these conditions proceeded or followed leishmanial infections.

Discussion

Local emergence of VL is evident at various temporal and spatial points within an existing and expanding local CL picture. True case burden may be higher than reported and VL emergence may be older than believed. Detected cases provide a projection on clinical, laboratory and epidemiological features of local VL, calling for urgent multi-sectoral action.

VL was not considered as the first diagnosis in any of these cases described above probably due to the diverse clinical presentations (Table 3), indicating the

need for improved awareness among clinicians. Since VL is asymptomatic during early stages, subsequent clinical features are not pathognomonic and other non-leishmanial laboratory parameters have variable trends (Table 4), high degree of suspicion is critical for early case detection in minimizing morbidity and mortality. In spite of some drawbacks, VL diagnosis was established within 3 months of current hospital consultations in all cases and anti-leishmanial therapy was completed within the next approx. 3 months. However, time taken from the appearance of one/ more of the clinical features to the time of laboratory confirmation was highly variable. Probable incubation period also varied from a minimum of 3 months to several years among the cases, indicating the necessity to carefully examine one's records on residence and travel over a lengthy period, if VL is suspected.

None of the cases fulfilled the full VL diagnostic profile (fever for over 2 weeks, splenomegaly and positive rK 39 test). Fever was a common presentation. Infections resulted in either acute features within a short period (fever, fatigue and rapid weight loss) or less apparent chronic features (splenomegaly, anemia, and pancytopenia). Fever, weight reduction and fatigue seem to be frequently associated features. VL can be easily misdiagnosed as other febrile illnesses such as malaria and enteric fever. Co-infections may mask the VL picture, further complicating timely diagnosis. Co-existing skin abnormalities as seen in one case may be suggestive of a PKDL like picture. Acute features seem to subside along with the specific treatment while chronic features tend to remain for a longer period in these patients. Clinical parameters seem to normalize within 24 months and without recurrence or PKDL. Laboratory confirmed infections with non-progressive clinical disease point towards the important question such as whether they are self-resolving minor leishmanial infections or whether concurrent accidental detection of still sub-clinical or asymptomatic infection (during investigations for another illness which share similar clinical features with VL).

Sero-conversion in local CL is being currently investigated [21]. Negative rK 39 results of 2/4 local VL cases indicate the lack of sensitivity/reliability of this assay in Sri Lankan patients, indicating the possible antigenic differences. Lack of sensitive and standard assays in VL detection been reported [22]. Current finding stresses the need for more locally appropriate serological tools. Sero-positivity was demonstrated in endemic but apparently healthy inhabitants (rK 39 assay positive [$n = 4$] but apparently healthy and from location B5, data unpublished) indicating possible sero-conversion without disease, self-resolving or asymptomatic VL.

Local infections responded well to the anti-leishmanials with no apparent evidence of recurrences, though it might be too early to make generalizable conclusions based on small samples. Neighboring India reports

resistance to first line anti-leishmanials and often uses second line drugs such as Amphotericin B. Expensive and toxic anti-leishmanial usage may cause a huge economic burden and serious side effects on patients. Sri Lankans are frequent travelers to VL endemic Bihar state in India and African countries for religious and employment purposes respectively. Management protocols and treatment response patterns of VL show great variability within different global endemic settings. Local anti-leishmanial therapy has followed multiple options so far [23]. Current global trend is to treat clinical illness and closely follow up asymptomatic cases, owing to the toxicity and complexity of available anti-leishmanials [24]. Evidence based revisit to local protocols may be necessary in future. All cases are likely to have acquired infection locally, although overseas travel history was present in two cases. Local infections appear to be responsive to first line antimonials which is encouraging when there is *L. donovani* associated drug resistance in other settings [25].

Residences and LPIAs may not be the same. Majority point towards a history of travel to/stay in known CL areas in Southern or Northern Sri Lanka (*central leishmaniasis database of University of Colombo*, 15). Two independent foci of disease transmission in North and South [9] and case clustering [10] were suggested by previous studies. None of the patients were residents of these areas except two, one who also spent most of his time in Northern Sri Lanka over the past 9 years and another whose residence was from location C. No other cases of VL have been reported from these foci, though the low index of suspicion among clinicians might be a contributing factor.

Spontaneous remission of VL has been reported at extremely rare situations [26]. In this backdrop, high degree of clinical suspicion and investigations are necessary to detect any possible cases, which might act as a parasite reservoir to the community.

Establishment of VL will result in considerable socio-economic ill effects on the country in addition to the increased mortality and high health cost in management. Wide prevalence of incriminated vector species (*P. argentipes*), free movements and possible intermingling of non-immune and immune populations, silent nature of initial infections, poor awareness on visceral leishmaniasis and scarcity of scientific information may viciously facilitate delayed intervention, and silent disease progression. With increased air travel, particularly to neighboring country India there is a risk of imported leishmaniasis too. Local transmission cycle is likely to be receptive to the foreign parasite strains which can facilitate disease spread.

Timely case detection and management are the recommended methods in control of *L. donovani* causing VL [2]. Though only a proportion of CL cases are offered pre-treatment laboratory confirmation, due to practical difficulties in the local setting, suspected VL always

require pre-treatment confirmation. CL in Sri Lanka is already known to be the result of *L. donovani* infection. A recent study on a single VL case identified *L. donovani* MON 37 as the causative organism of VL in Sri Lanka [27]. It is possible that some of the CL infections acquire visceralizing ability or there is strain variation within *L. donovani* in Sri Lanka enabling some of the strains to visceralize. Latter possibility is more likely as a long term follow up study carried out by some of us has failed to demonstrate any possibility for visceralization of initial CL infections at least during first 4 years following detection [28]. Origin of the local *L. donovani* is uncertain. Phylogenetic tree developed in the first local study on *Leishmania* parasite genetics demonstrated a distinct positioning of local isolates and their close relatedness to Indian/Nepal/Bangladesh group within *L. donovani* complex (8).

Conclusions

There is an on-going program to eliminate leishmaniasis from the Indian subcontinent [18] in which Sri Lanka, is not a part of. Raising professional and public awareness will improve the case detection. Community-based interventions are likely to help in the combat of the local disease spread.

Authors' contributions

YDS (MBBS, PhD) developed the concept, study design, conducted the project, designed and wrote the manuscript. PK (MBBS, MD) carried out clinical evaluations, data collection and commented on manuscript. LG (MBBS, MD) carried out haematological procedures and commented on manuscript. NK (MBBS, PhD) guided HVD in all steps. All authors commented and agreed on final manuscript.

Ethics approval

Ethical clearance was obtained from the Ethics Review Committee of Faculty of Medicine, University of Colombo, Sri Lanka.

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Disclosure statement

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