

Factors Associated With Nutritional Status among Children with Visceral Leishmaniasis (VL) Attending Kacheliba Subcounty Hospital, West Pokot County, Kenya

Ann Iruata Namulen^{1*} Prof. Simon Karanja² Dr. Yeri Kombe³

1. Institute of Tropical Medicine and Infectious diseases, Kenya Medical Research Institute PO box 54840-00200, Nairobi, Kenya.
2. School of Public Health, Jomo Kenyatta University of Agriculture and Technology
3. Centre for Public Health Research, Kenya Medical Research Institute

Abstract

Visceral Leishmaniasis (VL) causes considerable morbidity and mortality in Kenya. However, data on the factors associated with nutritional status among children aged 5-12 years at Kacheliba Sub County Hospital is nonexistent. This study sought to determine factors associated with the nutritional status among children aged 5-12 years with visceral leishmaniasis, in West Pokot County. A descriptive cross-sectional design was employed involving both quantitative and qualitative approaches to data collection. Two hundred and three children aged 5-12 years with VL were enrolled in the study. Data was analyzed using chi-square to test the association between the variables, odds ratio regression was used to test the multivariate relationships between various independent and dependent variables. WAZ and HAZ scores measures showed that malnutrition among these children there existed at 30.5%. Factors such as the guardian age, sex, monthly income, the length of illness, number of children in the household as well as level of education influenced the prevalence of malnutrition among these children. The study also established that knowledge, attitude and practices of these guardians also influenced prevalence of malnutrition among the children. The study concluded that there was interplay between malnutrition and VL among children and recommended that detailed surveys to establish the prevalence of malnutrition among communities where VL is rampant and coming up with initiatives of ensuring food security was necessary.

1. Introduction

Leishmaniasis is a group of diseases caused by the Leishmania parasite. It is still one of the most neglected diseases mainly affecting the poorest and impoverished group of the populations in Africa, Asia, and South America and, to some extent, Europe (World Health Organization, 2010). There are four types of Leishmaniasis; *Visceral Leishmaniasis* which is the most serious form and potentially fatal if untreated; *Cutaneous Leishmaniasis* which is the most common form which causes a sore at the bite site; diffuse *Cutaneous Leishmaniasis* which produces widespread skin lesions which resemble leprosy and is particularly difficult to treat and *Mucocutaneous Leishmaniasis* which commences with skin ulcers which spread causing tissue damage, to, (particularly), the nose and mouth (WHO, 2016). VL is the most devastating type among the group of Leishmaniasis. The term *kala azar* which means black (*kala*) fever (*azar*) in Hindi often is reserved for severe (advanced) cases of Visceral Leishmaniasis, although the terms *kala azar* and Visceral Leishmaniasis sometimes are used interchangeably. Visceral Leishmaniasis usually is caused by the species *L. donovani* and *L. infantum* (*L. chagasi*) and affects internal organs particularly, spleen, liver, and bone marrow. (Centers for Disease Control and Prevention, 2015). If untreated, severe cases of Visceral Leishmaniasis typically are fatal, either directly from the disease or indirectly from complications, such as secondary (myco) bacterial infection or hemorrhage (CDC, 2015).

2. Epidemiology of VL

Globally, of the estimated 1.5 to 2 million annual new cases of all forms of Leishmaniasis, the visceral form alone accounts for 0.5 million of the cases. It is estimated that 90% of the VL burden occurs in the poorest areas of Bangladesh, Brazil, Ethiopia, India, Nepal and Sudan (WHO 2009). Globally, over 200 million people are at risk of contracting VL (Boer & Davidson 2006).

In Africa, the estimated annual reported new VL cases in East Africa are 30,000 making the region the second highest foci for VL (WHO, 2010). In East Africa, VL is more closely related to the movement of seasonal migrant workers and refugees due to frequent civil unrest in most of these countries. The overlap in the epidemiology of HIV/AIDS and VL also greatly affects the latter (Desjeux 2014). This is due to the susceptibility of these groups when exposed to Leishmania-infected sand flies as they do not have natural immunity and rarely practice personal protective measures. Moreover, communities often have reduced immunity due to malnutrition and HIV/AIDS, which increases the susceptibility of individuals (WHO 2011).

In Kenya the disease was first detected in Kenya in 1935 in the Northern frontier districts of Mandera and Wajir. It is endemic in arid and semi-arid regions of Wajir, Mandera, Turkana, Tharaka, Machakos, and Kitui; in West Pokot County alone, the prevalence is 30% (WHO 2011). The expected annual cases in Kenya average 600 annually. Case fatality rate of up to 7% is seen in outbreak situations and higher deaths are being observed in HIV-VL co-infections. The new trend risks causing a public health crisis in weak Africa economies like Kenya since the vaccine for the disease is non-existent.

2.1 Known factors causing VL

VL is known to be caused by infectious agents like *L. donovani* in Africa, Asia, and Europe. In South America the causative agents are *L. infantum* and *L. chagasi*. These agents have both human and dog reservoirs. The mode of transmission is through an insect vector, the adult female sand fly; *Phlebotomus* genus in Africa, Asia, Europe and *Lutzomyia* genus in the South America. Of all human infections, 80-90% is sub-clinical or asymptomatic due to the strong cell mediated immunity (Blackwell, Fakiola, Ibrahim, Jamieson *et al.*, 2009). Various factors make the human host susceptible to the disease, including malnutrition, HIV/AIDS, and socio economic factors such as poverty.

2.2 Diagnosis of VL

VL is diagnosed through laboratory investigations;

DNA in infected tissue (such as in bone marrow, liver, lymph node, or blood), through light-microscopic examination of stained specimens, culture techniques, or molecular methods

Serologic testing can provide supportive evidence for the diagnosis:

- i. Specific serologic tests: Direct Agglutination Test, ELISA
- ii. Skin test (Leishmanin Test) for survey of populations and follow-up after treatment.
- iii. Nonspecific detection of hypergammaglobulinaemia by formaldehyde (formol-gel) test or by electrophoresis

2.3 Management of VL

Treatment of Leishmaniasis depends on the type of Leishmania parasite and country.

Recommended treatment regimens for VL for the different geographic regions (WHO Expert Recommendations):

Type of Leishmania parasite	Region	Medication and dosage
<i>L. donovani</i>	East Africa	SSG 20mg/kg/day for 30 days
<i>L. donovani</i>	India	Miltefosine 2.5/kg/day PO for 28 days
<i>L. infantum</i>	Spain, Portugal	Liposomal Amphotericin 3-5 Mg/kg/day in 3-6 infusions
<i>L. infantum</i>	Brazil	Liposomal Amphotericin 3-5 Mg/kg/day in 3-6 infusions

Supportive therapy includes nutritional support because 30% of VL patients in Ethiopia present with malnutrition (MOH 2006). Therapeutic feeding, balanced diet and vitamin supplementation are required in the management of VL patients. Secondary infections, including gastroenteritis, respiratory tract and skin infections, are common and need proper treatment (WHO 2010).

2.4 Prevention and Control of VL

Early case detection and treatment is the most important strategy for the control of VL, especially in areas where transmission is anthroponotic (WHO 2010)..

Improved knowledge on Leishmaniasis, availability of diagnostic and drug supplies, implementation of vector control strategies, and existence of functional surveillance systems would contribute to the control programme. Availability of health care workers trained in Leishmaniasis to improve access to the Leishmaniasis services and help the health workers to identify cases as early as possible before the disease becomes advanced and complicated (Chappuis *et al.*, 2007).

Personal protection measures like use of insecticide treated nets, vector control through indoor residual spraying, health education (WHO 2010).

2.5 Diagnosis of Malnutrition

The WHO recommends the use of the three most common anthropometric indices to assess the children's nutritional status these indices are weight-for-height, height-for-age and weight-for-age

2.6 Management of Malnutrition

The diet of a moderate acutely malnourished individual should comprise of well balanced meals containing proteins, carbohydrates, minerals and vitamins. They should also receive supplementary feeds to supplement their diet.

These supplements should provide 1000 to 1200 Kcals, of which 30% is supplied by fats and has 35 to 45g protein. Examples of supplementary feeds available locally include: Fortified Blended Flours (e.g. Fast Food and Corn Soy Blend) Ready to Use Supplementary Food (RUSF e.g. Plumpy Supplementary) Liquid Nutrition Supplements.

A typical food ration for such individuals should comprise of a cereal (e.g. maize meal), pulses (e.g. beans, lentils), oil, vegetables and at times sugar. Moderate acutely malnourished children with diarrhea should be given extra food and extra fluids to avoid deterioration of their nutritional status. Nutrition education and counseling is quite crucial in the management of moderate acute malnutrition.

Key nutrition education and counseling topics in the management of moderate acute malnutrition include:

Optimal complimentary feeding, feeding during sickness of the malnourished individual, hygiene and sanitation, de-worming, importance of monthly growth monitoring for children below 5 years, preparation, utilization, preservation and storage of supplemental feeds. In the management of moderate malnutrition, it is also crucial for effective medical management of prevailing clinical/medical conditions as they may be the root to the prevailing nutritional status of the individual.

In the management of moderate acute malnutrition in children below 5 years of age, supplemental feeds are given for a period of 8 consecutive weeks in which period they should have improved to normal nutritional status, and extra 4 weeks as follow up to ensure that they have completely recovered. In total, the moderate acute malnutrition management program should take 12 weeks.

There are such hindrances to the success of these programs as missing follow-up appointments, insufficient feeds among other socio-economic factors.

2.7 Interplay of VL and malnutrition

Nutritional health disorders play a role in the expression of clinically overt VL disease in the endemic areas. The nutritional status of the host may be impaired by the increased nutrient demands due to VL infection. (Hailu, 2014). There is a relationship between nutritional status of a host and infectious disease-related mortality and morbidity.

There is a vicious cycle involved between nutrition and VL, while nutrition increases or decreases the susceptibility to VL; VL also causes reduction in food intake (Scrimshaw, 2011.)

In response to infection, the immune system first executes innate and then subsequently acquired host defense functions of high diversity.

Both processes involve activation and propagation of immune cells and synthesis of an array of molecules requiring DNA replication, RNA expression, and protein synthesis and secretion, and therefore consume additional anabolic energy. Mediators of inflammation further increase the catabolic response (Moret & Schmid-Hempel, 2000). The nutritional status of the host critically determines the outcome and course of VL. Almost all nutrients in the diet play a crucial role in maintaining an optimal immune response, so that deficient and excessive intakes can have negative consequences on immune status and susceptibility to a variety of pathogens, including parasites of the *Leishmania* genus (Field, Johnson, & Schley, 2002; Malafaia, 2007).

The nutritional status of individuals infected with *Leishmania* ssp. has a significant role in the clinical evolution of VL, especially in children under 5 years. In the case of tropical disease, data obtained on Leishmaniasis in endemic regions of Brazil show that 67% of cases of cutaneous Leishmaniasis (CL) occur in younger people, and even more striking, 85% of cases of VL occur in children. Thus, among humans, the majority of the clinical cases appear to occur in younger individuals.

Studies have demonstrated that the high susceptibility to infection, in this group of risk, can be explained by the typical immunologic immaturity of this group, associated to PEM, very common in endemic areas of VL (Pearson *et al.*, 2011).

Protein Energy Malnutrition is a major determinant of both progression and severity of VL and greatly increases the case-fatality rate. Malnutrition, in any of its forms, contributes for more than 50% of deaths among children under 5 years in those countries (Malafaia, 2007).

Mortality rates of severely malnourished children treated as patients have been unchanged for the last five decades.

In developing countries, an estimated 50 million children under 5 years old are malnourished, and those who are severely malnourished, presenting a severe illness leading to hospitalization, face a case-fatality rate exceeding 20%. Epidemiologic studies have documented an increased risk for VL in the malnourished host (Maciel *et al.*, 2008). However, the impact of Protein Energy Malnutrition (PEM) specifically on immune response against infection with *Leishmania* is not totally understood yet and the nutritional status is many times neglected.

In Latin America, VL is still a disease of childhood with 60% of the cases occurring in children under 10 years old, an age group that has shown several other morbidities such as diarrhea and PEM contributes to the development of VL. These conditions frequently found in Leishmanial endemic areas, especially in North Eastern Brazil where the disease-endemic area has reached peri-urban locations.

PEM and VL provoke disastrous consequences for the growth, development and survival of children, mainly those with age between 1 and 4 years old PEM in individuals with VL has been associated with increased risk of in-hospital morbidity and mortality and increased period of stay, cost and use of healthcare resources (Bruna *et al.*, 2008).

Depending on the severity and duration of illness, VL patients may present with mild to moderate levels of malnutrition and approximately 30% of all VL patients are severely malnourished (MOH, 2006).

Untreated VL is invariably fatal as the duration of illness, malnutrition, human immunodeficiency virus (HIV) co-infection and the presence of other co-morbidities affect the case fatality rate (Collin, Davidson, Ritmeijer *et al.*, 2014)

3.0 Materials and Methods

3.1 Study site

This study was undertaken at Kacheliba Sub - County Hospital in West Pokot County. This is one of the level 4 hospitals under the Ministry of Health and is located in Karon, Suam location, Kacheliba division in Kacheliba Constituency. It is an operational hospital with a capacity of 52 beds. Referrals are from 5 locations and 18 sub-locations serving about 193,625 people. Some of the health care services offered include Antiretroviral Therapy, Curative In-patient services, family planning, HIV testing and counseling, home based care and immunization.

3.2 study design

A descriptive cross sectional design as employed applying both quantitative and qualitative approaches.

3.3 Study population

The study population in this study was children 5-12years with VL in the Hospital.

3.4 Sample size determination

This study used a formula to arrive at a sample size of 203. The sample for a large population is determined using the formula given as;

p= prevalence of poor nutritional status in children with VL from previous studies=30%

Cochran (1977) formula:

$$n = \frac{z^2 pq}{d^2}$$

n = desired sample size when population is greater than 10,000

z= standard normal deviation at at 95% confidence level (= 1.96)

q= 1-p

d= degree of accuracy desired set at 0.05

N= the target population

$$= \frac{1.96^2 \times 0.3 \times 0.7}{0.05^2} = 322$$

$$n = \frac{n}{1 + \frac{n}{N}}$$
$$= \frac{322}{1 + \frac{322}{550}}$$
$$= 203$$

Therefore the number of participants in the study was 203.

3.5 Sampling method

Systematic random sampling technique was used for this study. Children aged 5-12years with VL participated in the study. The skip interval was determined as follows;

N=550

n= 203

N/n = 550/203 =2.7

A random number 2 was selected from 1 to 3

Sampling was started from number 2 and every 3rd child was selected.

3.5.1 Inclusion criteria

The following inclusion criteria were used for this study:

- Children aged 5-12yrs with VL attending Kacheliba Sub-County Hospital.
- Participants willing to participate in the study

- Consent form from the guardians of the children with VL

3.5.2 Exclusion criteria

The exclusion criteria for this study were;

- Children below 5 and above 12 years.
- Guardians of the children who will not give consent for their children to participate in the study.

3.6 Sampling frame

The sample frame for this study consisted of all children registered in the Kacheliba - Sub county hospital kala azar register. A list of computer generated random numbers was used to select the total sample size.

3.8 Data collection method

Semi-structured questionnaires were administered to the guardians of the children with VL. The respondents were required to select answers from the choices given in the structured questions. Nutritional forms were used to capture the nutritional information. Nutritional status of the study children was determined by use of the anthropometry method:

- measure height, weight, and age
- Evaluate using the three nutritional indicators of height for age (HAZ), weight for age (WAZ) and weight for height (WHZ).

3.7 Data management and analysis

Both quantitative and qualitative approaches were used for data analysis. Quantitative data from the questionnaire was coded and entered into the computer for computation of descriptive statistics. The Statistical Package for Social Sciences (SPSS version 20) was used to run descriptive statistics such as frequency and percentages so as to present the quantitative data in form of tables and graphs based on the major research questions. The qualitative data generated from open ended questions was categorized in themes in accordance with research objectives and reported in narrative form along with quantitative presentation. The qualitative data was used to reinforce the quantitative data.

Descriptive statistics was used to investigate and describe the clinical manifestations of VL and its correlation with anthropometric measures.

Inferential statistics such as chi-square and odd ratio regression were used. While chi-square tested the association between the variables, odds ratio was used to test the multivariate relationships between various independent and dependent variables.

The odds ratio regression will be as follows;

Odds of Y (malnutrition=1/normal=0/given X) = $a + b_jX + e$

Where the dependent variable is prevalence of malnutrition measure by a dummy, where malnutrition=1 and normal =0.

e^{b_j} = the odds ratio calculated as the exponent (2.718718) raised to the power of beta

X= the independent variable

e=error term

4 Results

4.1 Prevalence of malnutrition

Based on these two measures the overall prevalence of malnutrition was assessed.

The study findings revealed that 30.5% of the children were malnourished while 69.5% of these children were normal.

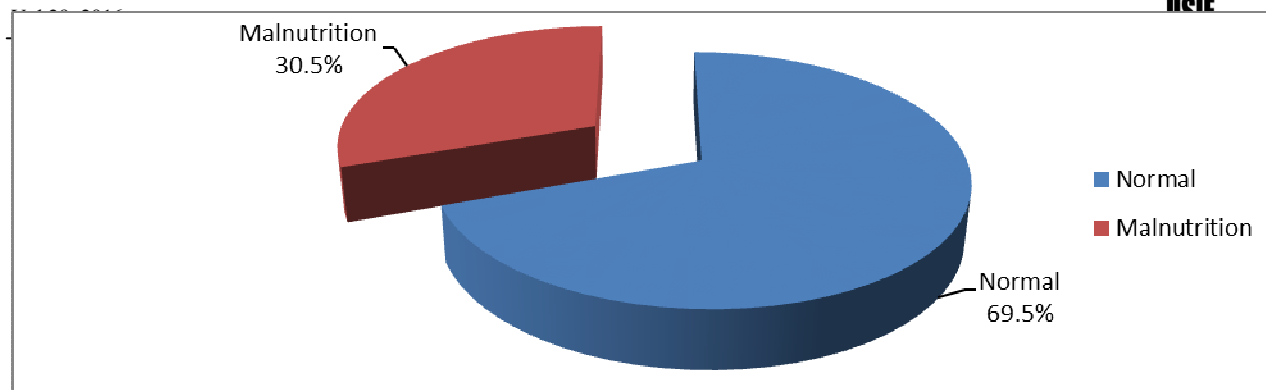


Figure 4.9 Prevalence of Malnutrition

4.1.1 Relationship between Social Demographics Characteristics and Prevalence of malnutrition among children with VL

The study sought to determine if there was any relationship between the following socio-demographic characteristics (Guardian sex, their Age, marital status, level education, religious affiliation and length of a child's sickness); these were the independent variables with prevalence of malnutrition-the dependent variable using cross tabulation and regression analysis model. The findings of these analyses are presented in table 4.4

The prevalence of malnutrition of the children was analysed individually and rated as either normal or malnourished, thereafter all the normal and malnutrition cases were added to give the total number of children with having malnutrition and those without from each category.

The study findings revealed that the percentage of malnourished children was 25.2% where the guardian was a male and 38.75% where the guardian was a female. The Chi-square statistic, however, showed a significant relationship between the guardian sex and prevalence of malnutrition ($\chi^2=4.193$ and $p= 0.041$) at 0.05 level of significance.

Marital status was cross tabulated against prevalence of malnutrition; the percentage of children with malnutrition was higher where the guardian was unmarried as compared to when they were married (29.89% vs. 44.44%). However, there was no statistically significant association between marital status and prevalence of malnutrition ($\chi^2=0.858$ and $p= 0.354$) at 0.05 level of significance.

Guardian age was cross tabulated against prevalence of malnutrition; the percentage of children with malnutrition was higher where the guardian was below 30years at 100% followed by ages 51-60 years at 68.96% as compared to when they are aged between 41-50 years, 31-40 years and above 60 years respectively. The study found a statistically significant association between guardian age and prevalence of malnutrition ($\chi^2=56.433$ and $p= 0.000$) at 0.05 level of significance.

Length of a child's sickness was cross tabulated against prevalence of malnutrition. A longer period of sickness was associated with higher prevalence of malnutrition as compared to shorter periods (100%, 64.00% vs. 22.81%). The study found a statistically significant association between the length of child's sickness and prevalence of malnutrition ($\chi^2=33.934$ and $p= 0.000$) at 0.05 level of significance.

The guardian highest level of education was cross tabulated against prevalence of malnutrition. A lower level of education was associated with higher prevalence of malnutrition as compared to higher education levels (40.39% vs. 3.03%).

The study found a statistically significant association between the level of education of the guardian and prevalence of malnutrition ($\chi^2=27.038$ and $p= 0.000$) at 0.05 level of significance.

The religious affiliation of the guardian was cross tabulated against prevalence of malnutrition. The percentage of malnourished children was 32.61% where the guardian was a Christian, 55.00% when a traditionalist and 23.08% when the guardian had no religious affiliation. The Chi-square statistic, however, showed no significant relationship between the guardian religious affiliation and prevalence of malnutrition ($\chi^2=8.215$ and $p= 0.016$) at 0.05 level of significance

Table 4.4 Cross Tabulation Analysis between Social Demographic Characteristics and Prevalence of Malnutrition among children with VL

Nutritional Factors	Normal	Malnourished	Malnourished (%)	χ^2	p-value
Guardian sex					
Male (n=123)	92	31	25.2	4.193	0.041
Female (n=80)	49	31	38.75		
Total (n=203)					
Marital status					
Married (n=194)	136	58	29.89	0.858	0.354
Unmarried (n=9)	5	4	44.44		
Total (n=203)					
Length of child's sickness					
Less than a year (n=171)	132	39	22.81	33.934	0.000
2-3years (n=7)	0	7	100.0		
Over 3years (n=25)	9	16	64.00		
Total (n=203)					
Level of education					
Never attended school (n=151)	90	61	40.39	27.038	0.000
Primary (n=34)	33	1	3.03		
Secondary (n=18)	18	0	0.0		
University/college (n=0)					
Others (n=0)					
Total (n=203)					
Religion					
Muslim (n=0)				8.215	0.016
Christian (n=92)	62	30	32.61		
Traditionalist (n=20)	9	11	55.00		
No religious affiliation (n=91)	70	21	23.08		
Total (n=203)					

4. Conclusion

Based on the study findings, the study concluded that cases of malnutrition among children with VL visiting this Health Centre existed. This was based on the finding that 30.5% of the children were malnourished. The study also concluded that there was interplay between malnutrition and VL among children. This was based on the indication by the respondents that the cause of weight loss was associated with Kala a zar. The study also concludes that the length of child's sickness was a very critical factor that led to loss of child's appetite and most of the time vomiting which was a hindrance to feeding the children. Long periods of sickness would also imply that the child was constantly deprived of crucial nutrients that were normally used up in fighting the disease and thus reduced their immunity. Sicknesses would also hinder the metabolism and absorption of food which could lead to malnutrition. The study also concluded that a minimum of education up to the primary school level would go a long way in reducing the prevalence of malnutrition in this area. This implies that some basic information on the interplay between VL and malnutrition is crucial. The study also concludes that taking a child to the hospital when they fall sick could help in the management of the disease since they are more likely to get treatment and basic information on how to manage the condition.

References

- Abubakar, A., Uriyo, J., Msuya, S. E., Swai, M., Stray-Pedersen, B. (2012). "Prevalence and risk factors for poor nutritional status among children in the Kilimanjaro region of Tanzania". *Int J Environ Res Public Health*, 9, 3506–3518.
- Alvar, J., Bashaye, S., Argaw, D., Cruz, I., Aparicio, P., Kassa, A., Orfanos, G., Parreno, F., Babaniyi, O., Gudeta, N., et al. (2007). "Kalaazar outbreak in Libo Kemkem, Ethiopia: epidemiologic and parasitologic assessment". *Am J Trop Med Hyg*, 77(2):275–282.
- Anshuman, M., Vijay, K. P., Sanjana, M., Kamlesh, G., Madhukar, R., & Shyam, S. (2009) "Risk, Factors for Visceral Leishmaniasis in India: Case-Control Study. 4th world congress on leishmaniasis, Page 228
- Anstead, G. M., Chandrasekar, B., Zhao, W., Yang, J., Perez, L. E., & Melby, P. C. (2001). "Malnutrition alters the innate immune response and increases early visceralization following *Leishmania donovani* infection". *Infect Immun*, 69: 4709–4718.
- Badaro, R., Jones, T. C., Lorenco, R., et al. (1986). "A prospective study of Visceral Leishmaniasis in an endemic area of Brazil". *J Infect Dis*, 154, 639–649.
- Bern, C., Maguire, J. H., & Alvar, J. (2008). "Complexities of assessing the disease burden attributable to leishmaniasis". *PLoS Negl Trop Dis*, 2: e313.
- De Onis, M., Onyango, A., Borghi, E., Siyam, A., Blossner, M., et al. (2012). "Worldwide implementation of the WHO Child Growth Standards". *Public Health Nutr*, 15: 1603–1610.
- Collin, S., Davidson, R., Ritmeijer, K., Keus, K., Melaku, Y., et al. (2004). "Conflict and kalaazar: determinants of adverse outcomes of kalaazar among patients in southern Sudan". *Clin Infect Dis*, 38, 612–619.