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Emergence or re-emergence of visceral leishmaniasis in areas of Somalia, north-eastern Kenya, and south-eastern Ethiopia in 2000–01

M. V. L. Marlet[1], D. K. Sang[2], K. Ritmeijer[1], R. O. Muga[2], J. Onsongo[2] and R. N. Davidson[1] *[1]Medecins Sans Frontieres–Holland, 40 Max Euweplein, P.O. Box 1001, 10014 EA Amsterdam, The Netherlands; [2]Ministry of Health, Nairobi, Kenya*

Abstract

Visceral leishmaniasis (VL) was known to be endemic in Somalia along the basins of the (Middle) Shebelle and (Lower) Juba rivers, and in Kenya in parts of the Rift Valley, on the border with Uganda and the Eastern Provinces. From May 2000 to August 2001, we diagnosed 904 patients with VL. The patients came from an area which spanned the Wajir and Mandera districts of north-eastern Kenya, southern Somalia, and south-eastern Ethiopia. Small numbers of patients were also seen in northern Somalia. These areas were either previously non-endemic for VL, or had only sporadic cases prior to the epidemic. We describe the features of the outbreak and review the history of VL in the region. Unusual rainfall patterns, malnutrition, and migration of a *Leishmania*-infected population seeking food and security may have contributed to this outbreak.

Keywords: visceral leishmaniasis, kala-azar, epidemiology, outbreak, East Africa

Introduction

The endemic areas for visceral leishmaniasis (VL; kala-azar) in Somalia, Kenya, and the south of Ethiopia, prior to March 2000, are shown as the hatched areas in Fig. 1. In March 2000, Medecins Sans Frontieres (MSF) noted a number of cases of VL among refugees from Somalia in refugee camps at Dadaab, in Garissa district, eastern Kenya (Fig. 1). Cases of VL were also detected and diagnosed in Mandera Hospital, where MSF had a feeding programme, and the Kenyan Ministry of Health reported increasing numbers of cases from Wajir Hospital. Boussery *et al.* (2001) reported an increase in the number of suspected cases in Kenya and those 34 cases are included in the number of treated patients in this report. The *gp63* polymerase chain reaction–restriction fragment length polymorphism typing of parasites isolated from patients in Dadaab refugee camp showed that the causative agent was *Leishmania donovani sensu lato* (Boussery *et al.*, 2001).

In July 2000, 5 blood samples from suspected VL cases were sent from Garbahaarrey, Gedo region, western Somalia, an area not previously known to be endemic for VL. All 5 patients had serological confirmation of VL (*Leishmania* direct agglutination test [DAT] titre $\geqslant$ 1:51 200). In August 2000, of 46 blood samples sent from Huddur, Bakool region, Somalia, 26 were DAT-positive at high titre ($\geqslant$ 1:51 200 and more) and 20 were negative ($<$ 1:400). Like Gedo region, Bakool region was not known to be endemic for VL.

Methods

In diagnosing this outbreak, we relied upon DAT and splenic aspiration. The DAT diagnosis was provided by MSF and was performed as reported elsewhere, using titres of $\geqslant$ 1:6400 as positive and of $<$ 1:400 as negative cut-off titres, exactly as in other MSF programmes (Boelaert *et al.*, 1999). The splenic aspirations were done by the Kenyan staff of the hospitals in Wajir and Mandera districts. Blood films were made of the aspirates, stained with reverse Field's stain and observed by direct microscopy ($\times$ 1000). Patients were seen at the health facilities—we could not do active case finding. The clinical case definition of VL was in accordance with WHO (1996): patients with fever for more than 1 month in combination with either splenomegaly or wasting, in whom malaria has been excluded. In cases meeting the case definition, VL was confirmed by a DAT titre $\geqslant$ 1:6400 and subsequent response to sodium stibogluconate treatment; or by demonstration of *Leishmania* amastigotes in spleen aspirates. The cut-off titre for the DAT was the same as MSF uses for VL cases in southern Sudan; no local value was determined. The Royal Institute of Tropical Medicine in Antwerp (Belgium) provided the DAT antigen.

Address for correspondence: Michiel V. L. Marlet, Hofweg 6, 7231 BH Warnsveld, The Netherlands;
e-mail mvlmarlet@hetnet.nl

Results

Patients

Patients were mainly ethnic Somalis, who are from mostly nomadic tribes whose grazing land covers areas of Kenya, Somalia, and Ethiopia. All these areas are ecologically similar: altitude 200–550 m, dry savannah *Acacia* thorn bushes, *Balanites* trees, and abundant termite hills. Bakool region is exceptional with its sorghum and maize fields. Our preliminary entomological studies revealed the presence of potential vectors of VL, *Phlebotomus martini*, *P. vansomerenae* and *P. celiae* in Wajir district, and *P. martini* and *P. vansomerenae* in Bakool region. Of 941 clinically suspected cases assessed by DAT up to August 2001, 631 were serologically confirmed and 273 were confirmed parasitologically. From these 904 confirmed cases, we could retrieve 765 (85%) reliable clinical records. In the north-east of Kenya, 263 patients were treated in Wajir (all parasitologically confirmed), 54 in Mandera (all serologically confirmed), and 32 in the refugee camps of Dadaab (8 parasitologically confirmed, 24 serologically confirmed). In southern Somalia, 230 patients were treated in Bakool region and 179 in Gedo region; all were serologically confirmed. The areas where the patients probably became infected are shown in Fig. 1. Travel histories of these nomads could not reveal any specific foci of transmission—they came from every corner of an area of 20 000 km^2. A few of the patients presenting in Kenya and Somalia came from nearby regions in Ethiopia. In an MSF feeding centre in Denan in south-east Ethiopia, 7 cases were diagnosed (by DAT) and treated.

In north-east Kenya, most of the cases diagnosed in Wajir came from the area between Wajir and the Somali border. The cases presenting in Mandera came from the border area between Kenya and Ethiopia. The cases treated in Dadaab refugee camps came from the border area, between Kenya and Somalia and from Lower Juba region in Somalia. One case each was diagnosed in Nugaal and Mudug regions (centred on the towns of Galcaio and Garoowe, respectively), they had probably never left the area. One case was diagnosed in Somaliland, but had lived in Lower Juba region.

We raised awareness of VL in health centres of

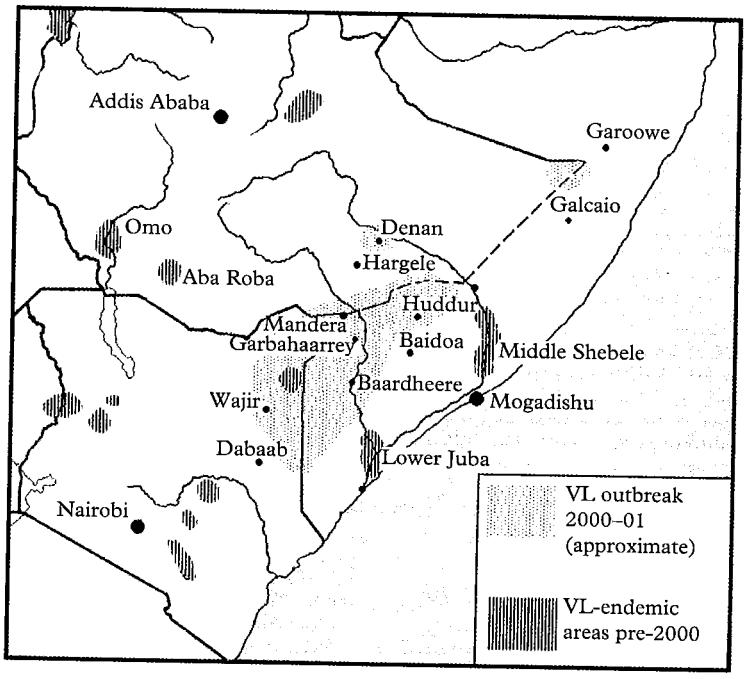

Fig. 1. Map of Kenya, Somalia, and parts of Ethiopia. The striped areas were known to be endemic for visceral leishmaniasis (VL) prior to 2000; the dotted areas are, approximately, the areas involved in the VL outbreak of 2000–01. The Gedo region centres around the town of Garbahaarrey and the Bakool region centres around the town of Huddur.

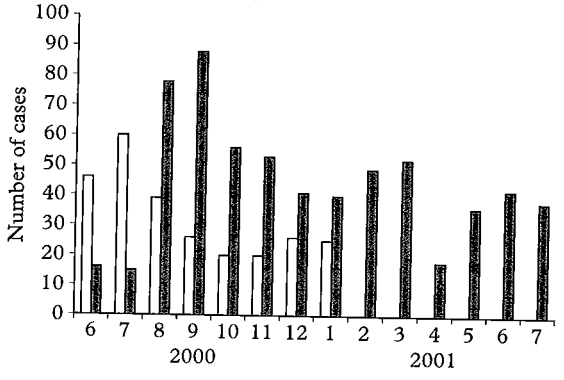

Fig. 2. Number of visceral leishmaniasis patients from north-eastern Kenya, southern Somalia, and south-eastern Ethiopia, diagnosed per month from 1 June 2000 to 31 July 2001. Key: white bars, cases treated in Wajir district, north-eastern Kenya; dark bars, patients treated in other centres. From February onwards no data were received from Wajir district.

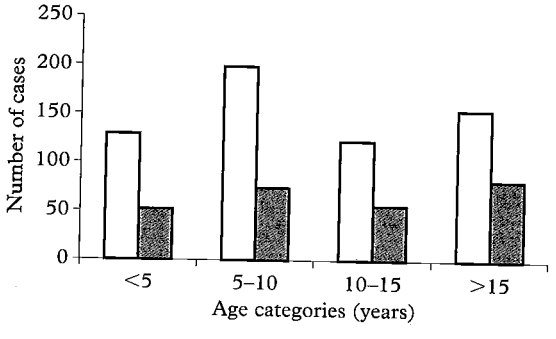

Fig. 3. Age and gender distribution of visceral leishmaniasis patients from north-eastern Kenya, southern Somalia, and south-eastern Ethiopia, diagnosed from May 2000 to August 2001. Key: white bars, males; dark bars, females.

Middle Shebelle region, a region known to be endemic area for VL, but no cases were reported to us from this region.

From February 2001 onwards, we stopped receiving data on patients from Wajir Hospital, when MSF-Belgium stopped its support programme to that hospital. In June and July 2000, there were very few patients from Somalia probably because there was no awareness of VL. In April 2001, few cases were presented in Somalia, probably because the rains made the farmers of Bakool region busy on their fields. We did not see a clear epidemic curve, possibly because the variation in duration of symptoms was wide (Fig. 2).

Clinical features

The male:female ratio of the DAT-positive and spleen aspirate-positive cases was 2.3:1. The age and gender distribution is shown in Fig. 3.

A few patients had symptoms for 1 month, the majority between 6 months and 2 years, and, especially in Somalia, some people claimed to be sick for up to 4 years. In the treatment centre of Huddur, Bakool region, the main treatment centre in Somalia where there were no laboratory facilities, the average duration of symptoms was 11 months. Many of the patients had gross abdominal distention due to splenomegaly. The clinical features were entirely typical of VL; starting with fever, splenic discomfort, poor appetite, weight loss, and wasting. Anaemia was a common feature, requiring blood transfusion in 10% of the VL cases in Wajir Hospital. Epistaxis was seen in 137 of 765 (18%) of cases. Intermittent respiratory infections were common. Whilst some patients had a daily fever, mostly in the afternoon or evening, the majority had no objective fever most of the time. In the treatment centre of Bakool region in Somalia we confirmed that many patients had only intercurrent fever. For example, in a boy aged 12 years, fever was confirmed on only 2 occasions in a 14-d observation period, yet his splenic aspirate contained numerous *Leishmania* amastigotes.

Two clinically typical and DAT-confirmed VL patients had post-kala-azar dermal leishmaniasis (PKDL)

before treatment, which healed along with VL during treatment. A third case developed PKDL immediately after his treatment. The PKDL resolved spontaneously following treatment for tuberculosis.

Treatment

Patients in Kenya were treated with sodium stibogluconate (Pentostam®, GlaxoSmithKline, Greenford, UK) 20 mg Sb/kg per day for 30 d, with a maximum dose of 900 mg Sb (Kenya Ministry of Health protocol). Patients in Somalia, were treated with sodium stibogluconate B.P. (International Dispensary Association, Amsterdam, The Netherlands) 20 mg/kg per day for 30 d, with no maximum dose (WHO and MSF protocols). Of 904 VL patients, 765 were treated, 349 in Kenya, 409 in Somalia, and 7 in Ethiopia. Of the 139 patients in whom we could not obtain confirmation of treatment, insufficient data were available in 113 cases, 16 died before treatment was started, and 10 failed to return to get their positive DAT result. Most cases responded well, clinically, to treatment. Discharge criteria were defined as: no fever in the last week of treatment, regression of spleen size, and improved general well-being. No routine parasitological test of cure was done. In only 1 centre, Wajir, test of cure was done if discharge criteria were not met. In Somalia, no aspirations were done due to a lack of skilled personnel and facilities. Thirty-four patients (4.5%) had their treatment prolonged by 2 weeks because they failed to meet the discharge criterion of no fever. Eighty-four (11%) patients died during treatment, varying from 7% to 20% among centres. We have no way of estimating the number of patients who died before they presented to health facilities.

Discussion

This outbreak indicates the emergence or re-emergence of VL in the areas described above. Cases of VL were reported from Wajir and Mandera districts of Kenya as far back as 1935 (Ashford & Bettini, 1987). Subsequently an outbreak occurred in a battalion of servicemen, patrolling the northern part of Kenya during the Second World War (Anderson, 1943; Cole *et al.*, 1942; Cole, 1944). In living memory (from community elders), cases suggestive of VL appeared to have occurred in Wajir and Mandera districts in the early 1930s and in the early 1950s. The latter coincided with a major epidemic in the nearby Kitui district, Eastern Province (Fendall, 1952). Other cases occurred in the early and mid-1980s, similarly coinciding with cases in Kitui district (D. K. Sang, unpublished data).

Visceral leishmaniasis was first reported from Somalia by Penso (1934), followed by a series of cases in 1955 (Moise, 1955) and 1963 from Middle Shebelle region (Barrufa, 1965). Cahill *et al.* (1967) and Shiddo *et al.* (1995) found the Middle Shebelle region endemic with younger age groups mostly affected. Later, in 1995 and 1996, MSF-Belgium reported 39 cases from the hospital in Kismayu, Lower Juba (M. Moncada, personal communication). During this period 1 case was reported from Baidoa (Woolhead, 1995). Up to 1984, no VL cases were reported from south-east Ethiopia (Ayele & Ali, 1984). Lindtjorn (1987) reported 1 VL case who may have been infected in the Dawa river valley in the south of Ethiopia.

The greatest risk of infection with *L. donovani* in the Aba Roba focus in southern Ethiopia, is throughout the wet season, from February to May and from September to October (Gebre-Michael & Lane, 1996). Southgate (1977) reported that in the Kitui district in eastern Kenya the monthly incidence of VL cases rises 6 months after the rainy season and the peak density of *Phlebotomus* spp. For the Aba Roba focus *P. martini* is incriminated as the principal vector and *P. celiae* is regarded as a secondary vector for VL (Gebre-Michael & Lane, 1996). The vector status of *P. celiae* still has to be confirmed. Both species are associated in Aba Roba with termite hills. The sandfly fauna of Wajir district contains *P. martini*, *P. vansomerenae*, and *P. celiae*, and is also termite hill-associated (D. K. Sang, unpublished data). The VL-affected areas in north-east Kenya experienced an unusually long wet season in 1997 and 1998, which might have had an impact on the sandfly population. In average years, there are 2 months with >70 mm rainfall and an annual average of 297 mm in Wajir district. In 1997 there were 5 months with >70 mm rainfall and the annual total was almost 1100 mm (Kenyan Meteorological Institute, Nairobi). This may have had an impact on vegetation and vector density in the following years.

Malnutrition is an important risk factor for developing clinical VL after infection with *L. infantum* (Badaro *et al.*, 1986), and there are indications (Ali, 1997) that malnutrition could be a risk factor for developing VL after infection with *L. donovani* in East Africa. There is also some evidence that asymptomatic people infected with *L. donovani* who are leishmanin skin test-positive, can convert to being leishmanin skin test-negative and can develop overt VL (Ali & Ashford, 1993). The years of drought following the excessive rains of 1997 caused malnutrition in this region. Unpublished nutritional surveys in 1999 and 2000 in Wajir district from OXFAM and Save the Children Fund (UK), and from Gedo and Bakool regions by the Food Security and Assessment Unit for Somalia showed increased acute malnutrition rates. Malnutrition may, historically, have caused earlier epidemics of VL. We compared the reports of VL in Wajir district with the rainfall data of Wajir station. The cases reported in the 1930s may have coincided with the droughts of 1934 (99 mm of rain), and the cases reported in the 1950s could be related to the droughts of 1949 (72 mm of rain) and 1955 (110 mm of rain)—it is unclear how large these outbreaks of VL were. The cases reported in the early 1980s followed the drought of 1980 (108 mm of rain)—the numbers of patients in this VL outbreak were thought to be small (D. K. Sang, personal observation). The 2000 outbreak may have been caused by a combination of a prolonged wet season in 1997 and 1998, followed by drought and malnutrition in 2000.

In addition to these factors, the migration of infected people seeking food and security may have contributed to the geographical extent of the outbreak. In Bakool region, VL seems have been endemic for some time—the disease is locally known as '*dedabsi*' meaning big spleen. It appears to affect mostly children in this farming community, and a survey (A. Berg, unpublished data) showed that many families in Bakool lost members due to a disease suggestive of VL. In 1998, part of the population of Bakool fled to Gedo region because of drought and insecurity in Bakool (E. Musch, unpublished data). These internally displaced people remained in Gedo and those with VL or PKDL might have introduced *L. donovani s.l.* in to an area with a competent sandfly vector. We have not yet been able to investigate the sandflies of Gedo.

Before this outbreak, there had been sporadic VL cases and small and localized outbreaks occurred in the eastern provinces of Kenya. In this area the disease probably became epidemic in a non-immune population living in an area of normally low endemicity. A similar situation probably occurred in Gedo region. In Lower Juba region the situation was unclear, as there is little information from this area. The influx of cases from Lower Juba seeking care in Dadaab refugee camp in Kenya may indicate that an outbreak of VL probably also occurred in Lower Juba. We cannot make conclusions about the situation in the south east of Ethiopia, but noted that all VL cases were children, which might indicate prior endemicity of the disease.

In conclusion, a combination of climatic and demo-

graphic changes seems to have resulted in the emergence, or re-emergence, of VL in this area.

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References

Ali, A. (1997). Visceral leishmaniasis in Southern Ethiopia. II. Nutritional risk factors. *Ethiopian Journal of Health Development*, **11**, 140–144.

Ali, A. & Ashford, R. W. (1993). Visceral leishmaniasis in Ethiopia. II. Annual leishmanin transformation in a population. Is positive leishmanin reaction a life-long phenomenon? *Annals of Tropical Medicine and Parasitology*, **87**, 163–167.

Anderson, T. F. (1943). Kala azar in the East African Forces. *East African Medical Journal*, **20**, 172–175.

Ashford, R. & Bettini, S. (1987). Ecology and epidemiology: Old World. In: *The Leishmaniases in Biology and Medicine*, vol. 1. Peters, W. & Killick-Kendrick, R. (editors). Orlando: Academic Press, pp. 366–424.

Ayele, T. & Ali, A. (1984). The distribution of visceral leishmaniasis in Ethiopia. *American Journal of Tropical Medicine and Hygiene*, **33**, 548–552.

Badaro, R., Jones, T. C., Lorenco, R., Cerf, B. J., Sampiao, D., Carvalho, E. M., Rocha, H., Teixeira, R. & Johnson, W. D. Jr (1986). A prospective study of visceral leishmaniasis in an endemic area of Brasil. *Journal of Infectious Diseases*, **154**, 639–649.

Baruffa, G. (1965). Kala azar among the nomads of the middle Webi Shabeli regions. First report of a confirmed endemic source of kala azar in Somalia. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **59**, 205–208.

Boelaert, M., El Safi, S., Jacquet, D., De Muynck, A., Van der Stuyft, P. & Le Ray, D. (1999). Operational validation of the direct agglutination test for diagnosis of visceral leishmaniasis. *American Journal of Tropical Medicine and Hygiene*, **60**, 129–134.

Boussery, G., Boelaert, M., Van Peteghem, J., Ejikon, P. & Henckaerts, K. (2001). Visceral leishmaniasis (kala azar) outbreak in Somali refugees and Kenyan shepherds, Kenya. *Emerging Infectious Diseases*, **7**, supplement 3, 603–604.

Cahill, K. M., Mazzoni, P. L. & Hassan, A. (1967). A leishmanin survey in Giohar, Somalia. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **61**, 340–342

Cole, A. C. E. (1944). Kala azar in East Africa. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **37**, 409–435.

Cole. A. C. E., Cosgrove, P. C. & Robinson, G. (1942). A preliminary report of kala azar in a battalion of Kings African Rifles. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **36**, 25–45.

Fendall, N. R. E. (1952). Kala azar in East Africa with particular reference to Kenya and Kamba Country. Part IV. Clinical Studies. *Journal of Tropical Medicine and Hygiene*, **55**, 245–256.

Gebre-Michael, T. & Lane, R. P. (1996). The roles of *Phlebotomus martini* and *P. celiae* (Diptera: Phlebotominae) as vectors of visceral leishmaniasis in the Aba Roba focus, southern Ethiopia. *Medical and Veterinary Entomology*, **10**, 53–62.

Lindtjorn, B. (1987). Visceral leishmaniasis in the Dawa valley, south Ethiopia. *Ethiopian Medical Journal*, **25**, 211–212.

Moise, R. (1955). A proposito dei casi di kala azar finora segnalati. *Annali di Medicina Navale e Tropicale*, **68**, 481–501.

Penso, G. (1934). Il kala azar nella Somalia Italiana. *Bollettini e Atti di Ricerca Accademia Medica Roma*, **60**, 292–293.

Shiddo, S. A., Aden Mohamed, A., Akuffo, H. O., Mohamud, K. A., Herzi, A. A., Haji Mohamed, H., Huldt, G., Nilsson, L. A., Ouchterlony, O. & Thorstensson, R. (1995). Visceral leishmaniasis in Somalia: prevalence of markers of infection and disease manifestations in a village in an endemic area. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **89**, 361–365.

Southgate, B. A. (1977). The structure of foci of visceral leishmaniasis in north-eastern Kenya. *Colloques Internationaux du Centre Nationale de la Recherche Scentifique*, **239**, 241–247.

Woolhead, A. (1995). A recent case of visceral leishmaniasis in Somalia. *Annals of Tropical Medicine and Parasitology*, **89**, 687–688.

WHO (1996). *Manual on Visceral Leishmaniasis Control*. Geneva: World Health Organization, WHO/LEISH/96.40.

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