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LEISHMANIASIS IN NORTHERN AND WESTERN AFRICA: A REVIEW

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Abstract

Leishmaniasis, one of the highly neglected diseases is currently a significant health problem in northern Africa with a rising concern in western Africa because of co-infection with the Human Immunodeficiency Virus (HIV). In this review, we present a summarized analysis of the epidemiology, infective species, parasites reservoirs, diagnosis, treatment and control measures of leishmaniasis in northern and western Africa region. In northern Africa, the disease is prevalent in Morocco, Algeria, Tunisia, Egypt and Libya. Comparatively, there are low prevalence rates of the disease in West African countries including Cameroon, Ghana, Burkina Faso, Niger, Mali, Nigeria and Senegal. In North Africa, visceral leishmaniasis (VL) is caused by *L. infantum* and transmitted by *Phlebotomus perniciosus* and *P. longicuspis*. On the other hand, cutaneous leishmaniasis (CL) is mainly caused by *L. major* and transmitted by *P. papatasi*, *P. duboscqi* and *P. pedifer* with *L. infantum* and *L. tropica* causing lower incidences of the disease. Notably, Algeria is one of the countries that constitute 90% of CL cases worldwide. In Western Africa; CL is caused by *L. major* while VL is caused by *L. donovani*. In these regions, zoonotic and anthroponotic cutaneous and visceral leishmaniasis is a health problem that should be addressed urgently.

Key words: Leishmaniasis; cutaneous leishmaniasis, visceral leishmaniasis

List of Abbreviations
CCL- Chronic Cutaneous Leishmaniasis
CL- Cutaneous Leishmaniasis
DCL- Diffuse Cutaneous Leishmaniasis
MCL- Mucocutaneous Leishmaniasis
MON- Mont-pellierzymodeme
SCL- Sporadic Cutaneous Leishmaniasis
VL- Visceral Leishmaniasis
WHO- World Health Organization
ZCL- Zoonotic Cutaneous Leishmaniasis

Introduction

Leishmaniasis is a zoonotic infection that is caused by obligate intracellular protozoa of the genus *Leishmania*. Natural transmission of *Leishmania* parasites is carried out by sandflies of the genus *Phlebotomus* (Old World) or *Lutzomyia* (New World) (Mandell et al. 2005). *Leishmania* parasites cause three forms of leishmaniases according to the localization of the parasites in mammalian tissues, notably visceral, cutaneous, and mucosal leishmaniasis. The outcome of infection depends on the species of *Leishmania* parasites and the host's immune responses (Roberts, 2006). Visceral leishmaniasis (VL) is the most severe form of leishmaniasis involving internal organs such as the spleen and liver and is fatal if left untreated (Mauel, 2002).

On a global scale, approximately 350 million people live in areas of active transmission of *Leishmania*, with 14 million people throughout Africa, Asia, Europe, and the Americas directly affected by the disease (WHO, 2006). The global burden of leishmaniasis has remained stable for some years, causing a morbidity and mortality loss of 2.4 million disability adjusted life-years (DALYs) and approximately 70,000 deaths, a significantly high rank among communicable diseases (Davies et al. 2003; Reithinger et al. 2007).

Leishmania and Human Immunodeficiency Virus (HIV) co-infection has surged as a major complication of leishmaniasis and has ignited calls for the recognition of leishmaniasis as an Acquired Immunodeficiency Syndrome (AIDS) defining illness (Singh, 2006). In Africa, particularly Ethiopia and Sudan, it is estimated that 70% of adults with VL also have HIV infection (Desjeux, 2001).

In West Africa, the disease has been reported in Niger (Stevene, 1911), Mali (Lefrou, 1948), Nigeria (Dyce-Shar, 1924), Senegal (Riou. and Advier, 1933), Cameroon, Burkina Faso, Mauritania, Gambia and Guinea (Boakye et al. 2006).

In northern Africa, Algeria is one of the eight countries that constitute 90% of CL in the World (Desjeux, 2004). Leishmaniasis contributes significantly to the propagation of poverty, because treatment is expensive and hence either unaffordable or it imposes a substantial economic burden, including loss of wages (WHO, 2006. Current control measures against leishmaniasis rely on chemotherapy to alleviate disease and on vector control to reduce transmission. To date, there is no vaccine in routine use against leishmaniasis (Handman, 2001).

This review article is based on information from bibliographic research, PubMed and MEDLINE searches, review articles and papers in their reference lists on leishmaniasis with several key words such as ''leishmaniasis", "cutaneous", "diffuse cutaneous", "mucosal", ''mucocutaneous'' and "visceral leishmaniasis"; "kala azar" for recent clinical and basic science articles related to leishmaniasis in northern and western Africa.

Leishmaniasis in North African region

Zoonotic cutaneous leishmaniasis caused by *Leishmania major* and *L. tropica* is found in many countries of North Africa. Cutaneous leishmaniasis caused by *L. major* is distributed in a belt from Marrakech and Casablanca in Morocco through Algiers in Algeria, Tripoli in Libya to Cairo, Alexandria and to the Sinai in Egypt. On the other hand, CL due to *L. tropica* ("dry sore") is also distributed in a similar belt across North Africa from the Canary Islands to Egypt. It is widespread in the urban areas of these countries although *L. tropica* is becoming more common in rural highland villages. CL is an important health problem, with clinical manifestations varying from simple cutaneous to mucocutaneous and disseminated lesions. *Phlebotomus papatasi* (sandfly) is the proven vector of *L. major*, and rodents *Psammomys obesus* and *Meriones* spp. serve as animal reservoir hosts. Some countries of the region have endemic foci of zoonotic and anthroponotic cutaneous leishmaniasis, which could cause epidemics among non-immune populations if they are involved in the transmission cycle (Ben Salah et al. 1995).

Leishmaniasis in Morocco

Morocco lies in the Mediterranean region where leishmaniasis is prevalent. Both CL and VL have been reported. During the past 20 years, CL has emerged as a major public health threat in Morocco (Rhajaoui et al. 2007). Cutaneous leishmaniasis caused by *L. major* has been reported in Morocco since 1914 (Rioux et al. 1986). In this country, CL is caused by *L. major* or *L. tropica* while VL is caused by *L.*

infantum (Rhajaoui, et al., 2004; Boussaa et al., 2005). The dog was confirmed to be the main reservoir of *L. infantum* MON-1, while the reservoir of *L. infantum* MON-24, causative agent of both infantile VL and CL, has not yet been identified (Haralambous et al. 2007). Until recently, CL was largely confined to arid Saharan regions (Haralambous et al. 2007). The Ministry of Health in Morocco considers *L. tropica* as a significant health threat. In 2001, the Moroccan Ministry of Health (MMH) reported 2,028 CL cases caused by *L. major* and *L. tropica* (Haralambous et al. 2007). *Leishmania tropica* CL has been reported in Azilal, Essaouira, Taza, Fes, the province of Chichaoua, and central Morocco (Marty et al. 1989; Rhajaoui et al. 2004). The northern coastal regions of Morocco are endemic for human and canine VL. As in other VL-endemic regions surrounding the Mediterranean Sea, this disease is caused by *L. infantum* (Ashford, 2000). In Morocco, the only previous human CL case caused by *L. infantum* was reported in 1996, within an active focus of VL (Rioux et. al. 1986, 1996). Cutaneous leishmaniasis and VL overlap in many provinces of central Morocco; anthroponotic foci of *L. tropica* CL are found in Fes and Taza (Guessous-Idrissi et al 1997; Chiheb et al. 1999; Rhajaoui et al. 2004) not far from existing VL foci including Sidi Kacem. Furthermore, several cases of canine VL caused by *L. tropica* have been reported in regions where canine VL is caused by *L. infantum* (Rhajaoui et al. 2007).

The overlapping distribution of parasite species, causing diseases with similar clinical pictures, demonstrates the need for additional epidemiologic and ecologic studies of CL in conjunction with species identification. This is especially important as opposed to traditional methods of determining infection from patient history and microscopy (Rhajaoui et al. 2007). In Morocco, local physicians and healthcare administrators often do not realize that different species of *Leishmania* require differential treatments, which can result in a failure to diagnose more serious disease. A simple, sensitive polymerase chain reaction (PCR) test could easily reduce such risk. One of the emerging threats is recurrent failure of local treatments (paromomycin and intralesional sodium stibogluconate) against *L. tropica* (Blum et al. 2004).

Leishmaniasis in Libya

Visceral leishmaniasis is an important public health problem in Libya, but until 1992, its exact prevalence was unknown (Mehabresh and El-Mauhoub, 1992). In addition, the cases of VL in Libya that have been reported for over 80 years were all from the northern coastal areas near Tripoli and the Green Mountain area. However, since 1985, there have been new cases of the disease from the southern part of Libya in the Saharan and sub-Saharan areas, an area 250 km to the south-west of Sabha. This southern area has recently undergone much agricultural organization with increasing water supply and other environmental changes, which may be partially responsible for the establishment of these new foci. Twenty patients with hepatosplenomegaly and fever were referred from that area to the El-Fateh Children's Hospital in Benghazi for investigation. All had the clinical features and laboratory data indicative of the Mediterranean type of the disease. All were treated with sodium stibogluconate (10 mg kg^{-1} day^{-1}), and responded well to this regime (Mehabresh, 1994).

A review of the records of 21 children treated at El-Fatah Children's Hospital, Benghazi between March 1982 and May 1990 demonstrated that the commonest presenting features were fever, abdominal distension, and anorexia with weight loss, hepatosplenomegaly and pallor. The consistent laboratory findings were aneamia with reticulocytosis and normal serum iron, neutropenia, thrombocytopenia, high erythrocyte sedimentation rate (ESR) and hyperglobulinaemia. The bone marrow was positive for *L. donovani* in 86% of cases and the indirect haemagglutination test was positive in all patients. Bronchopneumonia was the most common complication and responded rapidly to antibiotics. All patients were treated with sodium stibogluconate 10 mg/kg/day. There were no major side-effects or complications of drug therapy. The relative paucity of cases and their late presentation may reflect a lack of awareness of the occurrence of visceral leishmaniasis by doctors in the community (Mehabresh and El-Mauhoub, 1992).

Cutaneous leishmaniasis is widespread in the north-western region of the Libyan Arab Jamahiriya (El-Buni et al. 2000). The first two cases of CL were recorded in 1930 (Onorato, 1931). Another 40 cases were recorded in Nalut near the Tunisian border (Kadiki and Ashraf, 1971). Several CL cases have been recorded west and south-west of Tripoli among residents and new settlers in towns, villages and agricultural projects (El-Buni et al. 2000).

In the Yafran area of Libya, *P. papatasi* was the most common sandfly species found, followed by *P. sergenti*. Wild rodents including *Meriones libycus, M. shawi* and *Gerbillus gerbillus* have been found in the studied area and are considered possible animal reservoirs (El-Buni et al. 2000).

Leishmaniasis in Tunisia

Various forms of leishmaniasis are recurring in Tunisia, posing numerous problems concerning their aetiology and their clinical and epidemiological aspects which vary according to region. The respective role and importance of the animal hosts as well as that of the vectors involved is not yet clear, especially where, in recent years, important ecological changes especially construction of hydroelectric dams have taken place.

Three clinico-epidemiological forms of cutaneous leishmaniasis exist in Tunisia: zoonotic cutaneous leishmaniasis (ZCL; epidemic in the centre and the southwest); sporadic cutaneous leishmaniasis (SCL; found in the north); and chronic cutaneous leishmaniasis (CCL; originally described from Tataouine, in the southeast) (Kallel et al. 2005). Zoonotic CL is caused by *L. major*, SCL by *L. infantum and* CCL by *L. tropica* (Kharfi et al. 2003).

Recent studies by Ghrab et al. (2006) on vectors of leishmaniasis showed the dominance of subgenus *Larroussius* species in northern foci, *P. papatasi* in south-western foci and their co-dominance in the centre of the country in accordance with the distribution of *L. infantum* and *L. major* in Tunisia. These studies indicated that in *L. infantum* areas, the dominant species were respectively: *P. perfiliewi* in the cutaneous leishmaniasis site of the humid bioclimatic stage, *P. perniciosus* in the cutaneous and visceral leishmaniasis foci of semi-arid and arid bioclimatic stages and *P. longicuspis* in the visceral leishmaniasis focus of Saharan bioclimate.

In the ZCL foci, *P. papatasi* was a dominant species. In the well-known south-eastern foci of cutaneous leishmaniasis due to *L. killicki*, *P. sergenti* was a dominant species with *P. perniciosus* while in the central emerging foci of *L. killicki*; *P. perniciosus* was a dominant species in some sites whereas it was very rare in others. In these sites, the subgenus *Paraphlebotomus* was always present with a higher abundance of *P. alexandri* than *P. sergenti*. Rodents *Psammomys obesus* and *Meriones spp.* serve as animal reservoir hosts of *L. major* (Ghrab et al. 2006).

Zoonotic CL in Tunisia is characterized by clinical polymorphism (Masmoudi et al. 2007). Besides the classical forms (ulcerated and crusted form), other clinical forms seen in Tunisia are the lupoid, loco regional spreading (sporotrichoid form, satellite papules). Some atypical forms can be found which are due to variation of host immune responses and to the strain of the parasites involved.

The different forms of CL in Tunisia are treated with the same protocol (Chargui et al. 2005). In addition, a study on the efficiency of a polymerase chain reaction (PCR) method in establishing the diagnosis of CL in Tunisian patients supported the incorporation of PCR into diagnostic strategies for CL in the country (Chargui et al. 2005). The first three documented cases of anthroponotic cutaneous leishmaniasis due to *Leishmania killicki* were recently reported from locations outside the original focus of Tataouine in southeast Tunisia. Three strains were isolated from three patients from Gafsa, Sidi Bouzid and Seliana indicating an extension of this parasite's range towards the centre and the north of Tunisia (Haouas et al. 2005).

Whereas mucocutaneous leishmaniasis endemic in Central and South America are due to *L. braziliensis* and provokes mutilating and disfiguring lesions, the form in Tunisia is caused by *L. major*, is dermotropic and is characterized by the absence of mutilating lesions. In addition, MCL in the New World differs from that in Tunisia in that it is refractory to treatment while that in Tunisia responds excellently to treatment (Kharfi et al. 2003). A randomized, placebo-controlled trial in Tunisia treating CL with paromomycin ointment did not indicate any difference between treatment and control groups suggesting that paromomycin ointment should not be used in the present formulation as a treatment for ZCL in Tunisia (Ben Salah et al. 1995).

According to Kamarianakis (Kamarianakis et al. 2007), there are several reasons behind the increased ZCL incidence in the region. The majority of them depend on human activities such as environment modifications, resettlement of non-immune populations or development of agro-industrial projects, military activities, and urbanization and so on. Environmental modification, such as construction of dams, can change the temperature and humidity of the soil and vegetation, which may result in changes in the composition and density of sandfly species as well as changes in populations of rodent species. The formation of new settlements with non-immune populations facilitates the outbreak of leishmaniasis. For example, the outbreak of ZCL in the central and southern governorates of Tunisia in 1982-83 occurred following the construction of the Sidi Saad Dam (Kamarianakis et al. 2007). On the other hand, the destruction of *Psammomys obesus* burrows by deep ploughing, removal of chenopods and planting of trees

in a 2-3 kilometer zone surrounding human settlements has resulted in a significant reduction of the incidence of cutaneous leishmaniasis among the local human population (Kamarianakis et al. 2007).

Leishmaniasis in Algeria

As in other countries of Africa where leishmaniasis is prevalent, both VL and CL have been reported in Algeria (Harrat et al. 1996). The Grande Kabylie region of Algeria appears to be the most important focus of VL in North Africa and the Mediterranean region (Dedet et al. 1977). In Algeria, the incidence of hospital diagnosed VL has increased dramatically in recent years (Regional Disease Vector Ecology Profile, North Africa, 2000). About 500 cases were recorded between 1965 and 1974. Between 1985 and 1990, a resurgence of VL was observed with 5 human cases per 100 000 annual prevalence and a 6 per cent death rate in the region (Harrat et al. 1996). During this period, there were 1,122 cases, the majority of which were in the region of Tisi-Ouzzou in the Greater Kabylia. This increase was attributed to the cessation of insecticide spraying as part of the malaria control program. A small number of cases have been identified in Algiers (Regional Disease Vector Ecology Profile, North Africa, 2000).

Two forms of CL are endemic in Algeria. Zoonotic CL caused by *L. major* is widespread in the steppe regions of the northern Sahara while sporadic CL caused by *L. infantum* occurs in the north along the coastline (Benikhlef et al. 2004). The main causative agent for the sporadic form is *L. infantum* zymodeme MON-24. It has been isolated from the sandfly vector *P. perfiliewi* and the main reservoirs are dogs. Recently, *L. infantum* MON-24 was first isolated from a dog (Benikhlef et al. 2004).

In Algiers, the number of cases of canine leishmaniasis has been shown to be on the rise with a frequency of 35%. Human leishmaniasis was also shown to be on the rise, with 22 cases of VL and 40 cases of CL being noted in the period between 1990-1997 (Harrat and Belkaid, 2003).

Leishmaniasis in Egypt

Both VL and CL occur in Egypt although the prevalence is relatively low (Mohareb et al. 1996). Visceral leishmaniasis due to *L. infantum* was not confirmed in Egypt until 1983. It is primarily a disease of rural populations, but in some areas urban transmission exists. The disease has been found near Alexandria, in El Agamy (Awadalla et al. 1987). On the other hand, VL due to *L. donovani* is less frequent and is usually imported from Sudan (Mohareb et al. 1996). The proven or suspected vectors of *L. infantum* are *P. ariasi*, *P. longicuspis*, *P. pernicious*, *P. perfiliewi* and *P. langeroni* (Regional Disease Vector Ecology Profile, North Africa, 2000).

In Egypt, CL has primarily been identified in northern Sinai (Mansour et al. 1987) and was attributed to *L. major*, according to isoenzyme analysis (Mansour et al. 1989). A longitudinal epidemiologic study of CL transmission conducted between July 1989 and June 1991 in a 1,200-sq km sector of the north eastern Sinai Desert revealed that *L. major* was the only *Leishmania* species isolated from human, sand fly, and wild rodent (*Gerbillus pyramidum*) (Fryauff et al. 1993).

There are few endemic ZCL cases in the Sinai and Suez Canal Governates in Egypt. A survey of new cases of human ZCL among outpatients complaining of skin disorders revealed 16 cases out of 100 individuals examined. None of the 50 normal individuals had latent infections. Isolates from 3 individuals were identified as *L. major* Zymodeme London 70 (Hamadto et al. 2003).

Leishmaniasis in West Africa

Visceral leishmaniasis, CL and MCL have been reported in West Africa (Boakye et al. 2006). The first case of leishmaniasis in the region was reported in Niger in 1911 (Stevene, 1911), followed by other reports from Nigeria (Dyce-Shar, 1924), Senegal (Riou and Advier, 1933) and Mali (Lefrou, 1948). Other countries that have reported cases in the past include Cameroon, Burkina Faso, Mauritania, Gambia and Guinea (Boakye et al. 2006).

In the West African region, CL is caused by *L. major* and has been reported from reservoir hosts, vectors and human patients from The Gambia, Senegal, Burkina Faso and Mali (Dedet et al. 1981, 1982; Izri et al. 1989; Harrat et al. 1998). Cutaneous leishmaniasis is proposed to be endemic in a belt running from Mauritania, Gambia and Senegal in the west to Nigeria and Cameroon in the east. Although the CL belt mentioned cuts across the northern part of Ghana, the disease has not been reported in the country until

recently, in 1999, when some chronic ulcers diagnosed as cutaneous leishmaniasis were observed in the Ho District of the Volta Region (Boakye et al. 2006). Recently, the first definitive evidence of human CL resulting from *L. major* infections in Ghana was reported in Ho District (Fryauff et al. 2006). On the other hand in Ouagadougou, the capital city of Burkina Faso, 1,845 people were diagnosed with CL between 1996 and 1999 (Traoré et al. 2001).

Cases of MCL are reported to be rare in West Africa (Boakye et al. 2006). However, two cases of cutaneous leishmaniasis with mucous membrane involvement were reported in Senegal (Strobel et al. 1978). Like in other parts of Africa and the Old World, the causative agent of VL in West Africa is *L. donovani* (Bryceson, 1996). The disease however, is uncommon. Visceral leishmaniasis cases have been reported from Togo (De Campos et al. 1979), Burkina Faso (Andre et al. 1978) and the Gambia (Conteh and Desjeux, 1983; Greenwood et al. 1984).

It has been observed that all the *L. major* strains isolated from Senegal were identical to those from Central Asia (Dedet et al. 1982). However, the zymodeme found in Burkina Faso was different from that from Mali and Senegal (Izri et al. 1989). Most other reports including those from Ghana have indicated the presence of amastigotes in human samples but have not identified the specific *Leishmania* parasites (Boakye et al. 2006).

In West Africa, the reservoir hosts for *L. major* have been identified only from Senegal and The Gambia (Dedet et al. 1981; Dedet et al. 1982; Blanchot et al. 1984). The rodents *Mastomys erythroleucus, Tatera gambiana, Arvicanthis niloticus* and *Mastomys erythroleucus* and the dog are the reservoir hosts in the Gambia (Boakye et al. 2006).

Leishmaniasis in Cameroon

Both VL and CL have been reported in Cameroon. Cutaneous leishmaniasis prevails mainly in the northern part of the country, in the area of Mokolo, areas bordering Chad (including N'Djamena), and the eastern parts of the country whereas VL is more frequent in the area of Kousseri, in the north-east (Donji et al. 2001). Cutaneous leishmaniasis has been prevalent in Cameroon since 1930 (Hervé, 1937). Since then, there has been an upsurge in the number of CL cases that have been recorded (Donji et al. 2001). On the other hand, VL was first reported by Djibrilla et al. in 1979. However, few studies have focused on VL in Morocco, and the disease is still very little known even by medical staff and the final diagnosis is established only late and sometimes patients die without specific treatment. Between October 1987 and January 1988, a study conducted in the Kousseri region of the country found that out of 120 individuals who were sampled, 46 of them exhibited symptoms of leishmaniasis and 9 were confirmed positive for VL based on parasitological and serological diagnosis (Kaptue et al. 1992).

In Cameroon, *L. major* transmitted by *P. duboscqi* has been identified as the causative agent of CL while VL is caused by *L. donovani* (Donji et al. 2001). However, several gaps still exist about the epidemiology of leishmaniases in Cameroon; the prevalence of the disease in the population of endemic foci, identifying new foci probably in the southern forest, the repertoire of these parasitic species, vectors, animal reservoirs, natural factors and contributors to the spread of the disease. These data are deemed essential in planning a program against this emerging public health problem. Their availability will allow a better appreciation of risk of outbreaks and thereby limit the impact of the disease on the health of the population. Such studies ought to be accompanied by training of medical staff and paramedics on the different diagnostic methods (clinical, parasitological, immunological serum) and therapeutic especially in VL (Donji et al. 2001).

Leishmaniasis in Ghana

Since 1999, an increasing number of suspected CL cases have been reported from the southern Volta Region of Ghana in a moist semi-deciduous forest ecosystem where such skin lesions had not been previously reported. The assumption of CL was based subjectively on local microscopy, which identified *Leishmania* amastigotes in skin lesion biopsies taken from a cluster of local patients. Between 1999 and 2002, the Ghana Health Service recorded 2,426 suspected cases of CL in the Ho, Hohoe, and Kpando Districts (2,348, 2, and 76 cases, respectively). In 2003, the number of suspected cases rose to 6,450 (6,185, 174, and 91 in the same respective districts) with 116 villages affected (Fryauff et al. 2006). A limited survey of towns in the Ho district during 2002 identified suspected CL lesions in 12.2–32.3% of local school children.

Microscopic dissection and examination of 722 individual female sand flies captured in light traps during an epidemiological survey in the Ho District revealed no *Leishmania* infections. Although the study considered *P. duboscqi* as the primary suspected vector of CL, the species was near the least abundant of 17 different sand fly species that were collected and comprised only 0.1% of the total catch. One other suspect sand fly species, *P. rodhaini*, was captured for the first time in Ghana, but at similarly low numbers (0.3%), insufficient to yield a profile of seasonal abundance (Fryauff et al. 2006).

Leishmaniasis in Burkina Faso

Burkina Faso is a western African country with a high prevalence of both HIV and leishmaniasis infections (Niamba et al. 2007). Cutaneous leishmaniasis is caused by *L. major* MON-26 and clinical symptoms include papules, nodules, ulcers or papulo-ulcerative lesions (Heid, 1999). To date, no VL cases have been reported in Burkina Faso. Diffuse cutaneous forms of leishmaniasis are uncommon in Burkina Faso. However, a DCL case masquerading as leprosy in the context of HIV co-infection was recently reported (Niamba et al. 2007).

Cutaneous leishmaniasis in Senegal

A case of infiltrated cutaneous leishmaniasis and sporothricoid due to *L. major* MON-74 has been reported in Senegal (Thierno et al. 2001). In addition, DCL has been reported in this country (Develoux et al. 1996).

Control of leishmaniasis in Northern and western Africa

Leishmaniasis is one of the most neglected tropical diseases, in terms of the few tools available for control and the lack of clear criteria for methods of control (WHO, 2006). The main control strategy of leishmaniasis is case finding and treatment plus, when feasible, vector control and in zoonotic foci, animal reservoir control (Neouimine, 1996; WHO, 2004). So far, there is no effective vaccination against human leishmaniasis, and control of this disease relies primarily on chemotherapy (Handman, 2001). Pentavalent antimony has long been the pillar of anti-*Leishmania* chemotherapy, and although resistance to this class of drug has not been reported in Africa, the situation in northeast India has reached epidemic proportions (Croft et al. 2006). However, the main shortcoming arises from the fact that the pentavalent antimonials are costly, toxic, and require long duration of treatment and hospitalization. In addition, second-line drugs (pentamidine and amphotericin B) are limited by toxicity and availability, and newer formulations of amphotericin B are not affordable (Guerin et al. 2002). The high cost of drugs results in interruption of treatment with the possibility of development of chronic debilitating forms of the disease (Desjeux, 1992). Paromomycin ointment has been tried for the treatment of cutaneous leishmaniasis in Tunisia although results suggest that the formulation of the ointment needs further improvement before this method can be recommended for field practice (Ben Salah et al. 1995). International and voluntary organizations greatly support national control programmes with antileishmanial drugs, especially in emergencies (Neouimine, 1996).

Demonstration of parasites in stained smears of tissue aspirates from the spleen, bone marrow, or lymph node is the gold standard for diagnosis of VL with splenic aspirate and bone marrow being more superior to lymph node (Hailu et al. 2005). However, difficulties in obtaining and examining tissues mean that serological methods based on the detection of specific humoral antibodies are increasingly being used. The direct agglutination test (DAT), in which stained parasites are agglutinated by serum antibodies, is popular in Africa, but variation between batches and the high cost of commercially available antigen are limiting factors (Davies et al. 2003). In the Indian subcontinent, but less so in Europe and Africa, a rapid strip test is used to detect antibody to rK39 is both sensitive and specific. Weak responses in some patients, persistence of antibodies after cure, and presence of antibodies in some healthy individuals are inherent limitations with antibody based diagnostics. Techniques based on PCR are potentially highly sensitive and specific, but they need to be adapted for field use in terms of cost and user skills (Davies et al. 2003).

On vector control, sandflies are still very sensitive to insecticides (WHO, 2004). A recent study in Sudan indicates a potentially strong reduction in VL incidence following a community distribution of Insecticide Treated Nets (Ritmeijer et al. 2007). Further studies indicated that the use of impregnated bed

nets and going to bed early could provide a high degree of personal protection against this VL (Elnaiem et al. 1999). However, the potential use of bed nets for sandfly control in northern and western Africa is complicated by the fact that during the hot season it is considered too hot to sleep under the fine-mesh nets, which cut down ventilation (Ritmeijer et al. 2007). Furthermore, during the dry season people prefer to sleep outdoors and may be reluctant to use nets due to the daily routine to set up the nets before going to bed. In these resource restrained countries, vector control by spraying houses with insecticide is not sustainable due to logistic constraints and high cost.

Similarly, animal reservoir control through environmental management is expensive and difficult to implement; the efficacy of dog culling is questionable. In spite of this, in Tunisia, the destruction of *Psammomys obesus* burrows by deep ploughing, removal of chenopods and planting of trees in a 2-3 kilometre zone surrounding human settlements has resulted in a significant reduction of the incidence of cutaneous leishmaniasis among the local human population (Kamhawi, 1993). This is worsened by the lack of well-trained technical personnel and weak delivery systems, political and financial commitments are low and the level of implementation is frequently low (WHO, 2004).

Due to the apparent neglect of leishmaniasis in West Africa, there is no organized control effort in this region except during outbreaks when chemotherapeutic treatment is administered to those showing the disease (Boakye et al. 2006). In spite of the documented effectiveness of vector control with insecticides, and its potential in reducing disease incidence this has not been practiced in West Africa. In view of this, intensified collaboration between countries is essential in order to establish surveillance sites, map foci and prevalence on the basis of epidemiological assessments, train technical staff, investigate treatment failures, and set up computerized systems for data collection and analysis (WHO, 2007). The most pressing research needs for leishmaniasis control are the search for alternative and cheap medicines for oral, parenteral or topical administration in shorter treatment cycles, and identification of mechanisms to facilitate access to existing control measures including health sector reform in some developing countries (WHO, 2007).

Conclusions

Although VL is relatively rare in West Africa as compared to the CL, the disease is spreading to previously non-endemic areas and there is need for integrated control strategies to curb this wave. Of particular concern is the emerging *Leishmania*-HIV co-infection in West Africa which complicates treatment of the disease. Research into novel control measures is limited and this is compounded by the limited availability of data on prevalence of leishmaniasis. Consequently, there is need to establish a surveillance system to monitor the spread of the disease.

The number of leishmaniasis cases in Tunisia is increasing mostly because of human activities such as environment modifications, resettlement of non-immune populations or development of agro-industrial projects, military activities, and urbanization.

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