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CONTENTS

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CASE REPORTS

Traveller's loiasis in Zimbabwe: a case report

L M BORDON*, M MAURICE**

SUMMARY

A case of loiasis diagnosed at Wankie Colliery Hospital is presented. The disease was suspected by the history of the patient and the presenting clinical signs, and it was confirmed by identification of *Microfilariae loa loa* in peripheral blood. The patient was successfully treated with a course of diethylcarbamazine.

The paper gives a brief account of the clinical aspects of loiasis and emphasizes the importance of the laboratory methods to differentiate microfilariae. The case is discussed against the background of important diseases in Zimbabwe.

INTRODUCTION

In loiasis, microfilariae (*Mf loa*) have a diurnal periodicity in peripheral blood and transmission is by day bloodsucking tabanid flies of the genus *Chrysops* which inhabit the canopies of the rain forest belt of West and Central Africa. Man is the only final host of this species, and becomes infected by vectors harbouring the infective stage of larvae loa loa. Vectors are infected by ingesting microfilariae during a blood meal on infected humans. Although in expatriates and sensitized patients microfilaraemia may be responsible for occasional serious reactions,[1] most clinical manifestations of the disease are related to the migratory habits of the adult worms through the subcutaneous tissues causing the so called Calabar or fugitive swellings, a type I immediate hyper-sensitivity host's reaction to the antigen materials elaborated by the adult worms, as evidenced by high levels of eosinophilia[2]

*Medical Department
Wankie Colliery Hospital
**Laboratory Section
Wankie Colliery Hospital
Correspondence to:
L M Bordon
Wankie Colliery Hospital
P O Box 92
Hwange
Zimbabwe

A frightening and painful clinical sign of loiasis is the migration of adult worms under the conjunctive from where they can be, at times, extracted surgically. According to the data-base obtained, this is the first report of traveller's loiasis in Zimbabwe in which the clinical diagnosis was confirmed by identification of *Mf loa*. The present case adds to the concern of imported diseases in the country.

CASE REPORT

A 24 year old Caucasian female patient presented to the hospital because of a swollen and sore right eye for the past four days. She also complained of on and off itching swelling over the palmar aspect of her right forearm, and of mild, rather erratic arthralgias. The transient swelling of the forearm had tended to recur more frequently during the last year. Four years ago, she had spent one month travelling in Equatorial Africa, and now, she had been working in the Binga area for two years.

Examination of the right eye revealed a painful palpebral swelling with oedema and congestion of both, the bulbar and the palpebral conjunctivae. There was excessive tearing, and she also referred to some blurring of the vision. A soft and painless swelling over the palmar aspect of her right forearm appeared to be in remission. A WBC revealed eosinophilia of 28 pc, and a sample of fresh blood taken at 14:00 hrs, treated by the concentration method in Wintrobe tube, and examined microscopically with a low power lens, showed an overload of motile microfilariae.

Identification of microfilariae was made according to the following steps: the patient was admitted into the hospital and the following day fresh blood was taken at 12:00 and 24:00 hrs respectively. For investigation of tail nuclei and distribution, the Quantitative Buffy Coat Blood Parasite Detection Method (QBC, Becton Dickinson registered trademark) was used. Fresh blood was drawn into a QBC haematocrit tube precoated internally with acridine orange stain and potassium oxalate.

Examination under ultraviolet microscopy showed three plus parasites per QBC field, with nuclei extending caudally to the tip of the tail, a distinctive feature of *Mf. loa* from *Mf. bancrofti*, which apart from having nocturnal periodicity, show no nuclei in the caudal space of the tail. For demonstration of microfilariae sheath, the Bohmers haematoxylin stain method was used. Microscopic examination showed the presence of sheathed microfilariae, a differential feature of *Mf. loa* from *Mf. perstans*, which apart from being non-periodic in peripheral blood, has no sheath.

It was of interest to discover that the nocturnal blood samples of this patient were also positive for *Mf. loa*. This indicated that microfilaraemia had lost their diurnal periodicity and had become non-periodic. The patient was initially covered with intravenous dexamethasone, 5 mg b.d. The following morning, an adult worm was noticed to move away from the outer corner of the bulbar conjunctivae of the right eye. The same afternoon, the contralateral eye also became swollen, which meant that in terms of hours, the worm had managed to shift its presence to the left eye. No attempt was made to extract the parasite.

On day three after admission, a 21 day course of diethylcarabamizine (DEC) was initiated. The initial dose was 1 mg/kg body weight daily, and it was gradually increased up to 600 mg daily in divided doses. As no side effects or allergic reaction to DEC were noticed, dexamethasone was discontinued on day three of the specific treatment, and free of the eye's symptoms, the patient was discharged on day seven on ambulatory treatment.

On day 16 of DEC treatment, blood examination was reported to be negative for microfilariae, and eosinophilia had dropped to 15 pc. Two months after completion of the treatment, eosinophilia was 8 pc and peripheral blood was free of microfilariae. Since then, the patent has remained well, with no clinical and laboratory evidence of the disease.

Further follow ups shall be necessary in order to determine the effectiveness of the treatment in the elimination of the adult worms.

DISCUSSION

A previous paper reported two cases of imported loiasis in Zimbabwe.[3] Although the authors recovered adult worms from the eyes of both patients, microfilaraemia were not found in either case. Primarily, microfilaraemia is the result of the successful mating of the adult worms in the host. On the other hand, the lack of both, microfilaraemia and manifestations showing the signs of adult worms, does not rule out filarial disease. Such is the case of the occult filariasis, usually manifested as Tropical Pulmonary Eosinophilia (TPE), a syndrome more frequently due to *W. bancrofti* or *Mf. perstans* infection.

A patient with more than seven years of follow up, initially presented with the clinical and radiological features of TPE, with an eosinophils count of 48 pc and non demonstrable microfilaraemia. A bone marrow biopsy showed a marked increase of eosinophil precursors with heavy deposits of reticulo-endothelial iron in a matrix of desorganised hyaline material, which in view of the long history of residence in the Hwange area strongly supported the diagnosis of occult *Perstans filariasis*.[4]

One of the authors of this paper (LMB) when he was practising in Argentina, saw the case of a patient from the north of that country with persistent serofibrinous pericardial effusion, high levels of eosinophilia and absence of microfilaraemia. Eventually, an open pericardial biopsy with mediastinal lymph node samplings disclosed the presence of *Mf. perstans* (personal observation).

All the above examples are consistent with that line of thought which states that microfilaraemia is controlled by a humoral immune response. In fact, higher levels of total serum IgE have been found in patients with occult filariasis, and a significant fraction of this IgE has been shown to be directed against microfilarial antigens[5] It is this humoral immune response by part of the host which explains the clearance of microfilariae from peripheral blood in such a way that this stage of the disease is rarely detectable. In the particular case of loiasis, the diganosis can be easily made by the epidemiological data of residence in an endemic area, by the presence of Calabar swellings and ocular manifestations, and by extraction and identification of the adult parasites from the conjunctivae of the patient.

A type of occult filariasis has also been described in Loa loa infection. Such is the case of European patients with a history of residence in endemic areas for some period of time. Predominance of allergic symptoms with episodes of angioedema, fever, arthralgias, high eosinophilia, absence of microfilaraemia and of skin and ocular manifestations together with elevated titres of IgE, have been reported from this group of patients. If untreated, an undetermined percentage of such patients may develop cardiomyopathy, most probably secondary to hypereosinophilia and to the immune hyper-responsiveness elicited by the infection.[6]

On the other hand, an overload of microphilariae in peripheral blood may indicate a poor or incomplete humoral immune response to microfilariae antigens. This could be the case of travellers who become exposed to the infection for a relatively short period of time. A tentative hypothesis to explain these cases, should postulate that a much longer exposure in endemic areas with the cumulative effects of repetitive infections is necessary to produce an effective humoral immune response to microfilariae antigens. In endemic areas, *Mf. loa* has diurnal periodicity. This is a mechanism under circadian control by temperature and adapted to the diurnal biting habits of the vectors.[7]

In traveller's loiasis, as the patient moves out from endemic areas, this pattern of periodicity gradually changes and microfilaraemia become non periodic. Such was the case of the patient presented here in whom *Mf. loa* was also detected in nocturnal blood samples.

In some African countries multiple infections with filarial parasites are not uncommon, e.g. *W. bancrofti*, *Mf. perstans*, *L. Loa* and *O. volvulus*; hence the need to investigate in the laboratory the possibility of this occurrence if subsequent treatment is going to be decided.

In our context, *Mf. loa* should be distinguished from *Mf. bancrofti* and *Mf. perstans*, these last two forms of human filariasis being the only ones endemic in Zimbabwe.[8,9,10] Basically, microfilariae are differentiated by their periodicity, shape and size, but mainly, by the nuclear structure and distribution in the caudal space of the tail, and by the presence or absence of a sheath.[11,12] The value of Acridine Orange stain in the identification of the tail nuclear structure of microfilariae has already been emphasized by Goldsmid *et al.*[13] Currently, the QBC Blood Parasite Detection Method[14] facilitates the differentiation of those microfilariae whose nuclei extend caudally to the tip of the tail (*Mf. loa* and *Mf. perstans*) from those in which the tip off the tail is free of nuclei (*Mf. bancrofti*). In addition, a detailed examination under the ultraviolet microscope will show the cephalic space, nerve ring and excretory pore of microfilaria.

If doubts remain in relation to the presence or the absence of a sheath in microfilaria, a blood film stained with Haejatoxylin or Giemsa must be examined under ordinary microscopy. Haematoxylin stain will reveal both, the sheaths of *Mf. loa* and *Mf. bancrofti*, a differential feature from *Mf. perstans* which is unsheathed. On the other hand, Giemsa stain will reveal the sheath of *Mf. bancrofti* but will not stain the sheath of *Mf. loa* which will appears an unsheathed parasite leading to confusion with *Mf. perstans*.[15]

Unusual presentations of Loa loa infection include, among others, meningoencephalitis with micrfilariae in the CSF, endomyocardial fibrosis and acute arthritis with joint effusion. A series of female patients with suspected malignant breast lumps turned out on mammography to be vermiform calcifications of dead Loa loa filariae.[16] Of greater concern, is a recent report of Loa loa orchitis which can be considered as evidence to establish a link between filarial disease and some cases of male infertility in the tropics.[17]

For the most part, loiasis has a benign course, and the only manifestations noticed by the patients are the disturbing appearance of Calabar swellings and the transit of adult parasites under the conjunctivae. If these cases are accompanied by positive microfilaraemia DEC treatment is generally recommended. There is no doubt that this drug is highly effective in the eradication of microphilariae, the immature worms, and sometimes against the adult worms.

Severe reactions have been reported in patients with heavy microfilaraemia treated with DEC. They include, febrile and allergic states, purpura, and even brain and meninges capillary blockage by dying microfilariae which may lead to a severe, and sometimes lethal meningoencephalitis[2,7,8] In these instances, DEC treatment should be commenced with caution under intravenous steroid coverage, and the initial dose tailored in relation to the load of microfilariae.

Usually, it is recommended starting with no more than 1 mg per kg body weight of DEC daily in three divided doses. According to the clinical response, DEC should be increased gradually up to 200 mg, three times daily.[18] The average course of 21 days is highly effective in eradicating microfilariae and killing or immobilizing the adult worms.

In presenting this case, the vulnerability of the background to 'traveller's or 'imported' disease is also presented, just to illustrate that this phenomenon is not restricted to Western, developed countries. Diseases know no boundaries, and the Hwange area represents a vulnerable place for the introduction of diseases in Zimbabwe. It is a crossroad for both, migrant labour from neighbouring and overseas countries, and at the same time, a place of transit for regional and international tourists. Recorded at our hospital there are many examples of imported disease, such as most cases of chloroquine resistant malaria, some cases of amoebic colitis, typhoid and paratyphoid fevers, and shigellosis. In an interim survey (unpublished data) seven out of 48 expatriates working in the area of Hwange between 1986 and 1991, proved to be Hepatitis B Antigen carriers. A case of sparganosis is also recorded in a patient from Tanzania, and another one of 'Lung flukes' from Nigeria.

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tion upon the liver was necessary in order to decompress the intrahepatic biliary duct system. The case is discussed against the background of congenital cysts of the liver with especial reference to symptomatic polycystic liver disease. A review of the recent literature on the subject aims to summarise the current trends on diagnosis and treatment.

INTRODUCTION

Congenital cysts of the liver have been described as a rare cause of morbidity.[1] They can be solitary or multiple, may vary in size and location, but usually they are asymptomatic and compatible with normal liver function throughout life.

Symptomatic cysts which require abdominal exploration may be difficult to diagnose on clinical grounds only. In the past, they used to be surgical findings discovered at laparotomy or at necropsy.[2] While polycystic liver disease (PLD) shares the general behaviour of congenital hepatic cysts, in 50 pc of the cases it has the distinctive feature of forming part of the spectrum of adult polycystic disease (APD), an autosomal dominant inherited disturbance which by the fifth decade of life finds kidney clinical expression in