Loiasis: Report of Three Cases and Literature Review

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Loiasis is a tropical infection caused by the microfilarial nematode Loa loa. Increased numbers of cases of loiasis have been diagnosed outside of the endemic areas in Western and Central Africa because of widespread international travel. The author presents three cases of loiasis discovered at Howard University followed by a review of the literature.

Loiasis is a tropical infection caused by the microfilarial nematode Loa loa. Widespread international travel has increased cases of loiasis outside its endemic areas of Western and Central Africa. With an increase in international travel and work, the future will no doubt unveil increased cases of loiasis in native Americans and foreign visitors.[1,2] Three cases of loiasis in West African students studying in the United States are reported. These cases represent students who inhabited the endemic area for more than 20 years. The average length of time outside the endemic area was 43 months. These cases are presented to alert health care providers to the disease entity, its etiology, pathology, clinical manifestations, diagnosis, treatment, and prevention.

Case Reports

Case 1

In November 1977, an obese 30-year-old Nigerian man (R.A.) reported to the clinic with a whitish worm visible in his left eye. Emergency referral was made to the ophthalmologist for removal; however, the worm crept out of sight before completion of the consultation. The patient denied previous symptoms consistent with filarial infection. He had lived in the United States for five years. Physical examination revealed left gynecomastia but otherwise, the patient appeared normal. Tropical disease screening (stool and urine for ova and parasites) was negative. Thick smear of his peripheral blood revealed numerous microfilariae of Loa loa and numerous eosinophiles. Pre and post-treatment liver and renal functions were normal. He was treated with diethylcarbamazine citrite 6 mg/kg/day in three divided doses for 14 days. A post-treatment peripheral blood film was negative. Post-treatment eosinophile levels remained elevated at 9 percent. Interestingly enough, his pretreatment filarial indirect hemagglutination was 1:64, with 1:128 being a significant diagnostic titer; the Bentonite flocculation was negative, the diagnostic titer being 1:5.*

Case 2

In November 1977, a 23-year-old male Nigerian (S.I.) saw a whitish worm in his eye. At the time of medical consultation, the worm was no longer visible. He denied a history of previous tropical diseases, as well as specific symptoms of filariasis. He had been in this country approximately one year. His physical examination was unremarkable. A tropical disease screen, consisting of stool and urine for ova and parasites, was negative. His peripheral blood smears were positive for Loa loa and Acanthocheilonema perstans. A pretreatment eosinophile count was 10 percent. Pre and post-treatment liver and renal functions were normal. He was treated with diethylcarbamazine citrate 6 mg/kg/day in three divided doses for 21 days. His post-treatment eosinophile count was 5 percent. Post-treatment blood film was negative for Loa loa; however, A perstans persisted.

Case 3

A 27-year-old Nigerian man (N.A.) sought medical attention for hypertension follow-up. During the course of his hypertensive work-up, a differential white cell count revealed 12 percent eosinophilia. The eosinophilia prompted a tropical disease survey. He had had malaria at age 18; otherwise his medical history was negative for tropical diseases. He had been in this country approximately five years. Physical examination revealed blood pressure at 130/92, otherwise, unremarkable. His urine was negative for ova and parasites. His stool was positive for Trichuris trichiura. Thick smear of his peripheral blood was positive for Loa loa and A perstans. He was treated with diethylcarbamazine citrate 6 mg/kg/day in three divided doses for 14 days. Trichuria infestation was treated with thiabendazole 25 mg/kg/twice daily for three days. Pre and post-treatment liver and renal functions were normal. His post-treatment eosinophile count was 4 percent. Post-treatment blood film was negative for Loa loa; however, A perstans persisted. Post-treatment stool was negative for Trichuria.

Discussion

Manson first described Loa loa in 1891. Loiasis is distributed mainly among the coastal plains in West and Central Africa, from 8 degrees north to 5 degrees south of the Equator, and from the Gulf of Guinea eastward to the Great Lakes. The disease is also found between Southern Sudan and Zaire, between latitudes 4 and 6 degrees north and longitudes 27 and 31 degrees east.[1]

Etiology

The adult Loa loa worm is 30 mm or more in length with the female usually longer than the male. Both adults are enclosed within a sheath, somewhat V-shaped, with the concentration of nuclei in the tail. The presence of the sheath, and the finding of nuclei in the tail, are unique features of Loa loa and

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*The filarial titers were performed at the Center for Disease Control, Atlanta.

serve to differentiate Loa loa from other filariae.[3]

Loa loa has been demonstrated in monkeys inhabiting the rain forests of the Cameroons. Duke, however, concludes that there is no practical importance of the monkey as a reservoir for human loiasis.[4]

Pathology

Loa loa is transmitted by species of Chrysops, a mangrove fly. The fly ingests the microfilaria in a blood meal from an infected victim. Infective maturity is reached in about two weeks, at which time the larva is capable of transmission into another host. The infective larva migrates to the labella of the fly, and is deposited near and enters the puncture wound of the fly. The microfilaria migrates to the intradermal tissues where it reaches sexual maturity in about one year. The sexually matured worm is capable of reproducing microfilariae, and/or penetrating the microcirculation. Newly produced microfilariae likewise penetrate the circulation.[5] Adult worms are known to have first appeared as long as 17 years after the patient left the endemic area.

Clinical Manifestations

Calabar swellings are frequent clinical symptoms of loiasis. These nonpitting swellings classically occur over the eyes and/or dorsum of hands and feet. Calabar swelling is thought to be due to the following:

1. Migration of the worm
2. The liberation of toxic products by the parasites
3. The liberation of microfilariae
4. Allergic reactions by the host

The adult worm frequently migrates and surfaces within the subcutaneous tissues. Generally, the migration of the worm causes no local symptoms; however, occasional symptoms of itching, prickling, and creeping sensations, transient edematous swellings (calabar swellings), and conjunctivitis do occur.

It is suggested that heat stimulates migration and attraction to the skin's surface. Although the worms appear to have greatest predilection for the eyes, they do surface under the skin of the back, scalp, the lingual fraenum, the loose skin of the penis, and the skin of the fingers and breast.[3] Other clinical entities in patients with documented loiasis include peripheral neuritis,[6] endomyocardial fibrosis,[7] retinopathy,[8] fatal meningitis with microfilariae in the cerebrospinal fluid,[9] and glomerulonephritis.[10]

Loa loa worm is also known to migrate freely in body cavities and the mesentery. In 1977, Callihan et al[11] reported an incidental finding of a Loa loa worm in a cervicovaginal smear.

Diagnosis

Identification of the adult Loa loa worm in the subcutaneous tissue or of the microfilaria Loa in the peripheral blood is diagnostic. Loa loa exhibits diurnal periodicity. The embryos appear in large numbers in the peripheral blood during the day and disappear at night. Blood samples should therefore be taken about midday. Occasionally, the adult worm manifests itself, but examination of the peripheral blood reveals no microfilariae. This absence of microfiliriae in the blood may be the results of infection with only adult male nematodes, or of immunologic suppression of microfilariae production.[2]

Additional confirmatory evidence is obtained by hemagglutination, flocculation-complement fixation, and intradermal skin test. Infection is usually accompanied by increased eosinophiles.

Treatment

Diethylcarbamazine 2 mg/kg three times daily for 21 days is effective in killing the microfilariae and adult worms. Occasionally, a repeated dose, three to four weeks apart, is required to produce cure.

Serious Herxheimer-like reactions may occur, especially during the first week of drug therapy. To minimize such side effects, begin with 50 mg on the first day, increasing by 50 mg daily to a total of 2 mg/kg/three times daily. An antihistamine is also recommended during the first week of therapy. Steroids are indicated when fullblown allergic reactions occur.[12]

Prevention

Control and eradication of the Chrysop has proved less than satisfactory.

Duke developed a method for testing diethylcarbamazine as a prophylactic against Loa loa. His experiment was conducted on four volunteers. He concluded that 5 mg/kg daily for three consecutive days, once a month (200 mg for an average adult person), is likely to give complete prophylaxis against Loa loa.[13]

Conclusions

None of the patients experienced drug-related side effects or toxicity. To minimize the side effects of diethylcarbamazine citrate, the drug was started at 50 mg/day, and increased by 50 mg doses to the final dose of 6 mg/kg/day in divided doses. An antihistamine was also given during the first five days of therapy.

The three patients had microfilaria of Loa loa. Additionally, two of the three patients had A perstans. One patient with A perstans was treated for 14 days, the other patient was treated for 21 days. A perstans persisted in both patients following therapy. The persistence of A perstans coincides with reported cases of relative drug resistance of this filaria. Despite the persistence of A perstans, the eosinophile count fell by less than one half in both treated patients. The persistent eosinophilia in Case 1 is undetermined at present; it could have been the manifestation of an allergic reaction to diethylcarbamazine and/or the persistence of another parasitic infestation.

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