Short Report

Haematuria in coastal Kenya is associated with *Schistosoma haematobium* but not *Wuchereria bancrofti* infection

C. Njeri Wamae[1] and Patrick J. Lammie[2] [1]*Kenya Medical Research Institute (KEMRI), Centre for Microbiology Research, P.O. Box 54840, Nairobi, Kenya;* [2]*Division of Parasitic Diseases, 4770 Buford Highway, F-13, Centers for Disease Control and Prevention (CDC), Atlanta, Georgia 30341, USA*

Keywords: filariasis, schistosomiasis, *Wuchereria bancrofti*, *Schistosoma haematobium*, haematuria, prevalence, Kenya

Understanding the level of morbidity associated with filarial infections has assumed greater importance as part of efforts to provide better estimates of the global disease burden (WORLD BANK, 1993; WHO, 1994; MICHAEL et al., 1996). Historically, persons with circulating *Wuchereria bancrofti* microfilariae with no outward evidence of lymphatic dysfunction have been considered to be asymptomatic. However, more recent studies have provided evidence of subclinical renal and lymphatic abnormalities in these persons (DREYER et al., 1992; FREEDMAN et al., 1994, 1995).

each community was made. Subsequently, 200 participants were randomly selected from each community, each person in the 100 households was given a number, and 200 numbers were drawn at random from a pool of correspondingly numbered tags. After obtaining informed consent, venous blood samples (2–4 mL) were collected between 22:00 and 24:00 to detect any circulating microfilariae and to collect serum. Serum samples were examined for circulating filarial antigen by Og4C3 enzyme-linked immunosorbent assay, according to the manufacturer's instructions (TropBio, Townsville, Australia). Urine specimens were collected between 10:00 and 12:00 and checked for haematuria and proteinuria using a urine test strip (Chemstrips, Boehringer Mannheim Diagnostics, Indianapolis, Indiana, USA). Subsequently, 10 mL of each urine sample were filtered through polycarbonate Nuclepore® filters, 25 mm diameter and 12·0 μm pore size, and examined for *S. haematobium* ova. Group differences in the prevalence of *S. haematobium*, *W. bancrofti*, haematuria and proteinuria were analysed using the χ^2 test.

The overall age-specific prevalences of *W. bancrofti* microfilaraemia and antigenaemia, *S. haematobium*, haematuria and proteinuria in both communities are shown in the Table. As previously reported, *W. bancrofti* microfilarial prevalence was significantly higher in Kilore than in Mvumoni (WAMAE et al., 1997). The prevalence of filarial antigenaemia increased with age in both Mvumoni and Kilore and was higher in Kilore (48·9%) than in Mvumoni (20·5%) ($P<0·001$). In contrast, the prevalence of *S. haematobium*, haematuria and proteinuria decreased with age and there was no significant difference in the prevalence of *S. haematobium* be-

Table. Age-specific prevalence of *Wuchereria bancrofti* microfilaraemia and antigenaemia, and *Schistosoma haematobium* infection, haematuria and proteinuria

Community and age group (years)	Prevalences (%)[a] Microfilaraemia	Og4C3 antigen	*S. haematobium* ova	Haematuria	Proteinuria
Mvumoni					
≤20	4·7(85)	16·2(99)	74·7(95)	58·2(98)	48·9(98)
>20	8·5(59)	27·4(62)	35·4(48)	38·0(50)	22·0(50)
Overall	6·3(144)	20·5(161)	61·5(143)	51·4(148)	39·9(148)
Kilore					
≤20	14·5(55)	38·3(60)	81·0(79)	74·4(78)	61·5(78)
>20	30·0(70)	57·5(73)	53·6(69)	53·6(69)	37·7(69)
Overall	24·0(125)	48·9(133)	69·6(148)	64·6(147)	50·3(147)

[a]The numbers of subjects in each group are given in parentheses.

We studied the epidemiology of *W. bancrofti* infection in 2 adjacent communities in Kwale District, Kenya, in an effort to understand the relationship between transmission intensity and antifilarial antibody levels (WAMAE et al., in press). To investigate the influence of other parasitic infections on the relationship between filarial infection and antifilarial responses, we also examined subjects for intestinal helminths and for *Schistosoma haematobium*, which is prevalent in this region of Kenya (SHIMADA et al., 1987; KING et al., 1988). This provided us with an opportunity to analyse the relative contributions of *W. bancrofti* and *S. haematobium* to haematuria in an area where their distributions overlap.

The study was conducted in Muhaka Mvumoni and Muhaka Kilore, 2 of the 13 communities that constitute Muhaka, Kwale District. After registration of all members of each household, aged one year and above, a simple random sample selection of 100 households from

tween the 2 communities. Age-specific prevalence of proteinuria paralleled that of haematuria. The prevalence of haematuria was significantly higher in Kilore (64·6%) than in Mvumoni (51·4%) ($P=0·019$). Although haematuria prevalence was higher in Kilore, the community with the higher prevalence of *W. bancrofti*, there was no association between age-specific prevalence of *W. bancrofti* infection (either microfilaraemia or antigenaemia) and haematuria in univariate or multivariate analyses. In contrast, *S. haematobium* infection was significantly associated with haematuria ($P<0·001$). Thus, unlike *S. haematobium*, *W. bancrofti* does not appear to be associated with gross haematuria in this setting. It is possible that we failed to observe haematuria in some microfilaraemic persons because we did not count erythrocytes in urine specimens, as did DREYER and co-workers (1992). Although the dipsticks we employed are capable of detecting 5000–10 000 erythrocytes/mL of urine, it is difficult to compare these numbers to Addis counts based on total numbers of erythrocytes in a urine sample collected over a period of time. It seems likely, however, that Addis counts are a more sensitive means of detecting erythrocytes (RACE, 1980). Thus, we cannot rule out the possible contribution of microfilariae or immune complexes elicited by

circulating filarial antigens as a cause of subclinical renal damage. Nonetheless, we found no evidence that co-infection with *W. bancrofti* exacerbated haematuria in persons infected with *S. haematobium* in this setting.

References
Dreyer, G., Ottesen, E. A., Galdino, E., Andrade, L., Rocha, A., Medeiros, Z., Moura, I., Casimiro, I., Beliz, F. & Coutinho, A. (1992). Renal abnormalities in microfilaremic patients with Bancroftian filariasis. *American Journal of Tropical Medicine and Hygiene*, 46, 745–751.
Freedman, D. O., de Almeida Filho, P. J., Besh, S., Maia e Silva, M. C., Braga, C. & Maciel, A. (1994). Lymphoscintigraphic analysis of lymphatic abnormalities in symptomatic and asymptomatic human filariasis. *Journal of Infectious Diseases*, 170, 927–933.
Freedman, D. O., de Almeida Filho, P. J., Besh, S., Maia e Silva, M. C., Braga, C., Maciel, A. & Furtado, A.F. (1995). Abnormal lymphatic function in presymptomatic and asymptomatic human filariasis. *Journal of Infectious Diseases*, 171, 997–1001.
King, C. H., Lombardi, G., Lombardi, C., Greenblatt, R., Hodder, S., Kinyanjui, H., Ouma, J., Odiambo, O., Bryan, P. J., Muruka, J., Magak, P., Weinert, D., Mackay, W., Ransohoff, D., Houser, H., Koech, D., Arap Siongok, T. K. & Mahmoud, A. A. F. (1988). Chemotherapy-based control of schistosomiasis haematobia. I. Metrifonate versus praziquantel in control of intensity and prevalence of infection. *American Journal of Tropical Medicine and Hygiene*, 39, 295–305.
Michael, E., Bundy, D. A. P. & Grenfell, B. T. (1996). Re-assessing the global prevalence and distribution of lymphatic filariasis. *Parasitology*, 112, 409–428.
Race, G. J. (editor) (1980). *Laboratory Medicine*. Hagerstown: Harper & Row.
Shimada, M., Hirata, M., Ouma, J. H., Wambayi, E., Thiong'o, F.W. & Aoki,Y. (1987). Epidemiological study of *Schistosoma haematobium* infection in the coastal area of Kenya. Parasitological baseline data in the pilot area, Mwachinga. *Japanese Journal of Tropical Medicine and Hygiene*, 15, 173–184.
Wamae, C. N., Gatika, S. M., Roberts, J. M. & Lammie, P. J. (in press). *Wuchereria bancrofti* in Kwale District, Coastal Kenya: patterns of focal distribution of infection, clinical manifestations and antifilarial IgG responsiveness. *Parasitology*.
WHO (1994). *Lymphatic Filariasis Infection and Disease: Control Strategies. Report of a WHO Consultative Meeting held at the Universiti Sains Malaysia, Penang, Malaysia*. Geneva: World Health Organization.
World Bank (1993). *World Development Report 1993. Investing in Health*. New York: Oxford University Press.

Short Report

Second case of ocular parastrongyliasis from Sri Lanka

D. Wariyapola[1], Nalin Goonesinghe[1], T. H. Hsu Priyamanna[1], Charith Fonseka[2], M. M. Ismail[3], W. Abeyewickreme[3] and A. S. Dissanaike[4]*   [1]*Department of Ophthalmology, Sri Jayewardenapura Hospital, Talapathpitiya, Nugegoda, Sri Lanka; [2]Eye Hospital, Colombo, Sri Lanka; [3]Department of Parasitology, Faculty of Medicine, Kynsey Road, Colombo, Sri Lanka; [4]28 Welikadewatte, Nawala Road, Rajagiriya, Sri Lanka*

Introduction
When the rodent metastrongyle lungworm *Parastrongylus cantonensis* was first reported in rats and bandicoots in Ceylon (now Sri Lanka) it was predicted that human infection with this parasite was possible (ALICATA, 1966; DISSANAIKE, 1968). ALICATA (1966) showed that 64% of bandicoots (*Bandicota malabarica*) were infected and suggested that the widespread and abundant giant African snail *Achatina fulica* was a likely intermediate host.

DURETTE-DESSET *et al.* (1993) reported the first authentic case of ocular infection with this parasite in Sri Lanka from the anterior chamber of the right eye of a 30 years old male. The finding of a second emphasizes the fact that this infection is, surprisingly, not so rare, especially in a country where the intermediate host snails and slugs are rarely if ever eaten.

Case report
A 30 years old male from Makola, Kiribathgoda in the Colombo district complained of loss of vision in the right eye of 2 d duration. Four weeks earlier, he had suffered fever, headache, arthralgia, myalgia and generalized malaise. He had been treated with diethylcarbamazine and prednisolone and his symptoms subsided. Ocular examination on admission (25 June 1997) revealed visual acuity of 6/60 in the right eye and 6/6 in the left.

Fundal examination of the affected eye showed an actively motile, creamy-white roundworm in the subretinal space, with retinal oedema. Several tracks due to the movement of the worm were also seen (Fig. 1A). Five days later the worm had moved into the vitreous (Fig. 1B).

A trans-pars-planar posterior vitrectomy was performed by one of us (C.F.) on 10 July 1997, under general anaesthesia, and the worm was found in the subhyaloid space of the vitreous. A posterior vitreal detachment was induced using suction with the vitreal probe and an opening was made in the posterior face. Thereafter an intra-ocular foreign body forceps was used to extract the intact live worm.

No neurological sign or symptom or evidence of meningeal irritation was noted. The white blood cell count was 9600 with 24% eosinophils and the erythrocyte sedimentation rate (ESR) was 62 mm (in the first hour). On discharge from hospital (13 July 1997) the patient's vision was unchanged but the eosinophil count and ESR had returned to normal.

Four weeks after the operation, vision in the right eye remained at 6/60.

The worm (Fig. 2) showed the characteristic reddish streak of blood in the intestinal tract. It died a few hours after removal and was fixed in 70% alcohol and exam-

Author for correspondence; fax: +94 1 699284.