Journal of
TRAVEL MEDICINE



ORIGINAL ARTICLES

# Acute Schistosomiasis in a Cluster of Travelers From Rwanda: Diagnostic Contribution of Schistosome DNA Detection in Serum Compared to Parasitology and Serology

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DOI: 10.1111/j.1708-8305.2011.00552.x

***Background.*** Diagnosis of acute schistosomiasis is often elusive in travelers. Serum schistosome DNA detection is a promising new diagnostic tool. Its performance is compared with current diagnostic procedures in a cluster of travelers recently infected in Rwanda.

***Methods.*** Recent infection with schistosomiasis was suspected in 13 Belgian children and adults, within 2 months after swimming in the Muhazi Lake, Rwanda. All were subjected to clinical examination, eosinophil count, feces parasite detection, schistosome antibody tests [enzyme-linked immunosorbent assay (ELISA) and hemagglutination inhibition assay (HAI)], and schistosome DNA detection in serum by real-time polymerase chain reaction.

***Results.*** All 13 patients, between 6 and 29 years old, had a high eosinophil count (median 2,120 $\mu L^{-1}$; range 1,150–14,270). Seven of nine persons exposed for the first time developed symptoms compatible with acute schistosomiasis. Eggs of *Schistosoma mansoni* were found in a concentrated feces sample of 9/13 (69%), with low egg counts (median 20 eggs per gram; range 10–120). Antischistosome antibodies (ELISA and/or HAI) were present in serum of 10/13 (77%) patients. Combining schistosome antibody tests and fecal microscopy demonstrated schistosomiasis in 11/13 (85%) patients. Schistosome-specific DNA was isolated in all 13 (100%) serum samples.

***Conclusion.*** In this cluster of travelers with acute schistosomiasis, schistosome DNA detection in serum was able to confirm infection in all exposed persons. It clearly outperformed antibody tests and microscopic parasite detection methods as a qualitative diagnostic test.

Schistosomiasis (or bilharziosis) is a tropical parasitic disease caused by blood-dwelling trematodes of the genus *Schistosoma*. Freshwater snails are the intermediate hosts, shedding cercariae infective to humans. Symptomatic acute schistosomiasis (AS), or Katayama syndrome, is a systemic hypersensitivity reaction directed against the maturing schistosomulae in the liver. AS is frequently reported in clusters of western travelers who have bathed in lakes and rivers in sub-Saharan Africa.[1–4] Diagnostic confirmation is often elusive in suspected AS as well as in asymptomatic infection. Primary infection may cause a range of nonspecific symptoms that are often overlooked, or may remain asymptomatic.[5,6] In cluster series, the prevalence of acute infection reported after exposure may be very high, sometimes exceeding 90%.[3,7,8] A raised eosinophil count is an early marker of infection.[9] Detection of serum antibodies against schistosome (adult worm and/or egg) antigen is currently the most sensitive standard test procedure to diagnose infection in travelers but often fails in the acute phase of the disease.[10–13]

Schistosomiasis due to *Schistosoma mansoni* is known to occur in Muhazi Lake, Rwanda, the site of infection of this cluster and a popular weekend destination for

The data of this paper have been presented at the annual TropNetEurop workshop, August 28th, 2010, Leiden, the Netherlands.

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local expats.[14] A nationwide survey on schistosomiasis recently conducted in Rwanda revealed a prevalence rate of 69.5% among primary school children from Rwesero on the west side of Lake Muhazi (Dr Eugene Ruberanziza, personal communication, 2009). In this study, we described the clinical and diagnostic features, and the treatment outcome of schistosomiasis among a cluster of 13 Belgian travelers recently exposed at the Rwesero section of the Muhazi Lake.

## Methods

AS was suspected in a group of 13 Belgian school children and adults, infected after swimming in Muhazi Lake, Rwanda during summer school holidays. A very high eosinophil count was seen in two children with fever and raised suspicion of AS. All 13 exposed persons were subsequently referred to our outpatient clinic. The children had traveled as a group on holiday to Rwanda, together with an adult monitor. They had stayed in a hostel at the north-western shore of Muhazi Lake, Rwesero district, and had been frequently swimming there for about 14 days.

All 13 exposed persons were subjected to a standard clinical and a diagnostic workup according to current practice in our outpatient clinic. Workup includes absolute eosinophil count, schistosome antibody detection, and feces parasitology as detailed below. Exposure to diagnosis (EtD) was defined as the time lapse between first exposure and the date of diagnostic workup. In symptomatic patients, the incubation period (EtS: exposure to symptoms) was defined as the time lapse between first day of exposure until the earliest appearance of symptoms associated with AS. Symptomatic AS was defined as a raised eosinophil count ($>1{,}000\ \mu L^{-1}$) associated with at least one of the following symptoms appearing within 3 months from primary exposure to schistosomiasis: urticaria, angio-edema, fever $>38^\circ C$, diarrhea, abdominal pain, and cough.

A single fecal sample was processed for microscopic detection of ova and parasites using the ether sedimentation technique adapted from Laughlin and Spitz.[15] Two methods were used for antibody testing in a serum sample: an in-house enzyme-linked immunosorbent assay (ELISA) using a *S mansoni* egg antigen extract mixed with *S mansoni* adult worm extract imported from Egypt, and an indirect hemagglutination inhibition assay (HAI), using a *S mansoni* adult worm extract (commercial test, Fumouze SA, France), with titration and cut-off set at 1/80 (positive at $\geq 1/160$).

In addition, a 2-mL serum aliquot preserved at $-60^\circ C$ was sent to the Bernhard-Nocht Institute, Hamburg for DNA extraction and subsequent detection of cell-free schistosome DNA by a real-time polymerase chain reaction (PCR) assay according to the method described by Wichmann in 2009.[16] Meanwhile this method has been adapted to suit smaller volumes (2 mL) of serum instead of 20 mL of plasma, without losing sensitivity. To perform a real-time PCR with sufficient specificity, a 121-bp tandem repeat sequence, identified by Hamburger in 1991, that consitutes about 12% of the *S mansoni* genome and is highly specific for all human schistosomes, was chosen as the real-time PCR target gene.[17] Together with the patients' test samples, a quantification standard plasmid (positive control), containing the nucleotides 39 to 79 bp from the 121-bp tandem repeat sequence, and an internal negative control were run, as described by Panning.[18] Plasmids were purified and subsequently quantified by spectrophotometry. Dilutions of the standard plasmid were used as a quantification reference in real-time PCR. The cycle threshold value (Ct value) corresponds with the number of PCR cycles needed to attain the threshold level of log-based fluorescence. A test was considered positive when the threshold was attained within 45 PCR cycles (Ct value <45). A lower Ct value corresponds with a higher amount of template DNA copies in the serum sample.

In this cluster series, the specificity of the PCR assay for human schistosomes has not been assessed. This was already done by Wichmann in his original assay setup where he used plasma from 30 blood donors and 35 patients examined for other conditions to be tested by large-volume plasma extraction and CFPD real-time PCR.[16] None yielded positive results. We therefore felt it unnecessary to have this experiment rerun.

All patients were treated with a single dose of praziquantel at 40 mg/kg. Patients were instructed to take a single dose of methylpredisolone (0.5 mg/kg) in case fever developed soon after praziquantel treatment, and to contact the attending physician at our outpatient department for further advice regarding the duration of this treatment. Patients were seen again 5 weeks thereafter for evaluation of cure and a second single dose of praziquantel to be given to all, consistent with the established clinical practice at the outpatient clinic.

For data analysis, a nonparametric test (Mann–Whitney/Wilcoxon) was used to compare continuous variables (including Ct values), and the Fisher exact test to compare proportions.

Consistent with the in-house ethical guidelines for noninterventional studies, patients' consent (or their adult guardians' consent) was formally obtained to perform an additional diagnostic test on a preserved serum sample already used for antibody testing. The outcome of PCR test results did not interfere with the diagnosis, standard treatment, and follow-up procedure of schistosomiasis at the outpatient clinic. All patients were already assumed to be infected with schistosomiasis on the basis of exposure and raised eosinophil count.

## Results

In total, 13 patients (median age 12, ranging from 6 to 29 y) had been exposed to schistosomiasis when repeatedly swimming in the Muhazi Lake for up to 14 days, and presented at a mean time lapse of 78 days (range 54–96 d) from the first day of exposure to the

diagnostic workup at our outpatient clinic (Table 2). Of these 13 patients, 4, all asymptomatic, had also been exposed at the same site 2 years prior, and were unaware of having been possibly infected thereafter. The remaining nine patients had been exposed for the first time. Of these, seven developed symptoms compatible with AS. Symptoms appeared at a median period of 55 days (range 25–93 d) from the first day of exposure, and at a median of 6 days (range 3–28 d, $n = 6$) before the clinical diagnosis was suspected. Reported symptoms included angio-edema (5), urticaria (1), fever (2), cough (4), abdominal pain (4), and diarrhea (3) (Table 1).

Biological markers and test results are compiled in Table 2. All 13 patients had a significantly elevated eosinophil count (median 2,120 $\mu L^{-1}$; range 1,150–14,270). Eggs of *S mansoni* were found in a concentrated feces sample in 9/13 (69%) patients, all with low egg counts (median 20 eggs per gram; range 10–120). At least one anti-schistosome antibody test (ELISA and/or HAI) was positive in 10/13 (77%) patients. When combined with fecal microscopy results, schistosomiasis was demonstrated in 11/13 (85%) patients.

Schistosome-specific DNA was detected in serum by real-time PCR in all 13/13 (100%) exposed persons within the preset maximum of 45 cycles (median Ct value of 30; range 27–36).

Five weeks after the first treatment with praziquantel, 12/13 patients presented at a post-treatment visit. Eosinophil count was significantly lower (median 835 $\mu L^{-1}$; range 290–1,960 vs median 2,120 $\mu L^{-1}$; range 1,150–14,270; $n = 12$, $p < 0.001$) and egg count was negative in all five patients who submitted a sample and in whose feces eggs were detected before treatment. Anti-schistosome antibodies were still undetectable in 3/12 (25%) follow-up samples, while schistosome DNA remained detectable in all 12/12 (100%) cases tested at slightly lower Ct values, although the difference was not statistically significant (median 28.5; range 23–35 vs median 30; range 27–36; $n = 12$, $p =$ ns) (Table 2).

**Table 1** Clinical and diagnostic features of schistosomiasis in a cluster of patients recently exposed to *S mansoni* infection

| *N* | Exposure | Symptoms | Onset | Fever | Cough | Angio-edema | Urticaria | Abdominal pain | Diarrhea |
|---|---|---|---|---|---|---|---|---|---|
| 1 | Primary | + | 5 | + | + | − | − | + | + |
| 2 | Primary | + | 14 | + | + | + | + | + | + |
| 3 | Primary | + | 7 | − | − | + | − | + | − |
| 4 | Primary | + | 28 | − | + | + | − | + | − |
| 5 | Primary | + | 5 | − | − | + | − | + | + |
| 6 | Primary | + | 3 | − | + | − | − | − | − |
| 7 | Primary | + | n/a | − | − | + | − | − | − |
| 8 | Primary | − | | | | | | | |
| 9 | Primary | − | | | | | | | |
| 10 | Prior | − | | | | | | | |
| 11 | Prior | − | | | | | | | |
| 12 | Prior | − | | | | | | | |
| 13 | Prior | − | | | | | | | |

onset: symptom onset prior to diagnosis (days); primary: recently exposed for the first time; prior: recently exposed but also exposed 2 years prior; n/a: not available.

**Table 2** Evolution of diagnostic parameters of schistosomiasis before and at 5 weeks after treatment with praziquantel

| | | | Eosinophils (per microliter) | | ELISA | | HAI | | DNA-PCR | | EPG | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *N* | EtD | EtS | First | FU | First | FU | First | FU | First | FU | First | FU |
| 1 | 62 | 57 | 5,400 | 570 | + | + | 320 | 160 | 28 | 28 | 30 | − |
| 2 | 54 | 33 | 2,090 | 470 | − | + | 320 | 160 | 27 | 26 | 120 | n/a |
| 3 | 54 | 47 | 2,640 | 290 | − | − | − | − | 30 | 28 | 10 | − |
| 4 | 90 | 62 | 14,150 | 1,960 | + | + | 640 | 160 | 29 | 29 | 40 | − |
| 5 | 62 | 57 | 1,150 | 1,210 | − | − | − | − | 35 | 35 | − | n/a |
| 6 | 96 | 93 | 2,860 | n/a | + | n/a | − | n/a | 31 | n/a | 20 | n/a |
| 7 | 54 | 25 | 14,270 | 870 | + | + | 320 | 160 | 30 | 34 | 60 | − |
| 8 | 96 | n/a | 11,120 | 740 | + | + | 160 | 160 | 29 | 23 | 10 | n/a |
| 9 | 96 | n/a | 1,960 | 1,120 | + | + | 320 | 160 | 32 | 30 | − | − |
| 10 | 62 | n/a | 1,290 | 390 | − | − | − | − | 36 | 33 | − | − |
| 11 | 96 | n/a | 1,210 | 800 | + | + | − | − | 32 | 31 | 10 | n/a |
| 12 | 96 | n/a | 2,120 | 1,890 | + | + | − | 320 | 27 | 28 | − | n/a |
| 13 | 96 | n/a | 1,700 | 1,280 | + | + | 320 | 640 | 35 | 28 | 10 | − |

First: first visit of diagnosis; FU: follow-up visit at 35 days post-treatment; EtD: first exposure to diagnosis (in days); EtS: first exposure to symptoms (in days); ELISA: schistosome ELISA qualitative antibody test; HAI: schistosome indirect hemagglutination inhibition test, in 1/titer; DNA-PCR: schistosome DNA detection by PCR, in cycle threshold (Ct) values; EPG: eggs per gram feces; n/a: not available; +: positive; −: negative.

Following treatment with the first single dose of praziquantel, three of the nine patients with primary infection (all three with symptoms of AS before treatment) developed high grade fever (above 38.5°C). Fever subsided promptly after administration of a single dose of 16 mg methylprednisolone given the next day, and did not reappear thereafter. Two patients had only mild and transient abdominal pain that did not require additional treatment. None of the patients experienced any symptoms after the second dose of praziquantel given at the follow-up visit 5 weeks later.

## Discussion

AS is a frequent diagnosis in travelers and expatriates from non-endemic countries recently exposed to cercariae of human schistosomes. Symptoms may be discreet and comprise an urticarial rash and/or angioedema, medium grade fever, a non-productive cough, abdominal pain, and diarrhea.[5,10,19–22]

In most patients of this cluster, symptoms were mild and had already resolved before treatment was given. In practice, AS is usually not recognized by primary health care providers who are not familiar with tropical pathology. When the first symptoms appear, eosinophilia may not yet be raised.[10] As illustrated with this cluster, eosinophilia will however increase rapidly in the course of the following days to levels rarely seen in other parasitic diseases. Diagnosis is thus likely when at least one of the above symptoms appears in association with a clearly raised eosinophil count and with a primary exposure to schistosomiasis up to 90 days prior, pending confirmation of schistosome infection.[1,23,24] In the early disease stage, diagnosis cannot be reliably confirmed by antibody tests or parasite detection methods.[6,25] However, by the time patients are referred to a travel clinic, evidence of schistosomiasis is found in most, mainly by serum antibody detection and/or ova detection in feces or urine.[6,10,23,25] The current techniques for the laboratory diagnosis of AS have some shortcomings. Antibody production against adult worm and egg antigens starts only after schistosomules in the liver have been matured and after oviposition has started around the perirectal or perivesical venous plexus. This occurs at the earliest 6 weeks after infection, when symptoms may have largely subsided. Serological techniques used in clinical practice do not distinguish active infection from past exposure nor provide reliable information on parasite burden, and are not species-specific. Most routine techniques detect IgG, IgM, or IgE against soluble worm antigen (SWA) or soluble egg antigen (SEA) by ELISA, HAI or immunofluorescence. When combining assays using different sets of antigens in parasitologically confirmed infection, sensitivity may exceed 90% while retaining specificity at over 97%, but is less in AS.[10,11,26,27] In this cluster, seroconversion failed to happen in three patients during the follow-up period, one parasitologically confirmed and two with symptomatic AS. Whether this is due to early treatment, a low parasite burden, or host immune response factors is unclear.

In most travelers and migrants, established schistosomiasis infection is predominantly asymptomatic and with a low parasite burden, so that eggs are often not found in excreta.[28,29]

Nevertheless schistosome eggs were detected in feces of nearly all (6/7) symptomatic patients of this cluster. This may be due to low average age of patients, as well as to the relatively long average time lapse between exposure and diagnosis.[30]

There is thus a need for a more sensitive qualitative diagnostic test that confirms schistosomiasis infection at an earlier stage. Diagnosis based on schistosome DNA detection by PCR both in excreta and in serum has shown promising results.[31–33] Most assays target parasite DNA sequences common to all human schistosome species. Assays using species-specific probes are less sensitive.[34] A real-time PCR assay to detect schistosome DNA in plasma was found to be 100% sensitive in parasite proven established infection, and showed superior diagnostic sensitivity compared with serology in AS.[16] In Wichmann's series of eight patients with AS, schistosome DNA could be demonstrated in 10 mL plasma from all, at an average of 40 days after exposure, and at an average of 14 days after symptom onset, while schistosome EIA antibody detection was still negative in three of eight patients. This was also the case in the present cluster, but then the time lapse between first exposure and diagnosis was considerably longer (mean 78 d). This suggests that schistosome DNA detection in serum appears to be superior to egg detection and serum antibody assays as a qualitative marker of early symptomatic infection, and that a 2 mL serum sample may contain enough schistosome DNA to be amplified, at least when infected with *S mansoni*. Actually the number of copies per genome of the 121-bp tandem repeat sequence target gene may vary considerably between human schistosome species.[17] To be clinically useful in a post-travel setting, where only limited amounts of blood are routinely taken, its sensitivity should also be assessed in acute urinary schistosomiasis (*Schistosoma hematobium*) using an equally small serum sample. Furthermore, the minimum time lapse after infection needed to detect parasite DNA by this method has not yet been determined.

The amount of schistosome DNA copies in serum did not diminish significantly, despite a very clear drop in the mean eosinophil count and the halting of egg excretion 5 weeks after initial praziquantel treatment. This is in line with results of animal studies, and probably results from the continued release of cell-free DNA from degrading schistosomes, from persisting schistosomes still immature at the time when the initial praziquantel treatment was given.[16,25] Clearance of this circulating cell-free DNA is apparently a very slow process. In Wichmann's series of patients with AS, schistosome DNA was still detectable in most patients even up to 15 months after treatment. Only by then the

plasma DNA concentration had substantially declined. Schistosome DNA detection in serum obviously fails as an early quantitative marker of therapeutic success, in contrast with PCR assays on fecal samples.[31]

It is tempting to assume that the number of cycles required to attain parasite DNA detection in a blood sample by a real-time PCR assay is a reliable marker of parasite burden. However, there is insufficient evidence supporting that thesis, and there is considerable interpersonal variation even when exposure is similar. This study was not designed to address this issue.

## Conclusion

In this cluster, symptoms compatible with AS were seen in primary infection only. Diagnosis of active schistosomiasis infection was confirmed in all cases by schistosome DNA detection in serum, which clearly outperforms other current direct and indirect diagnostic methods. It is particularly helpful to confirm diagnosis of schistosomiasis in its early stage. It is yet unclear to what extent schistosome PCR in serum can be used as a very early qualitative marker of infection, and as a quantitative marker of parasite burden.

## Declaration of Interests

The authors state they have no conflicts of interest to declare.

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This is a public warning to be aware of the dangers of chronic schistosomiasis in Senegal (West Africa). In this area, along the River Sénégal (Richard-Toll, Northern Senegal), schistosomiasis (due to Schistosoma haematobium) is endemic and the complications of chronic schistosomiasis are common in the Senegalese. Travelers are exposed as well when they bath in endemic areas. In contrast to local residents and immigrants settled in Western countries, western travelers more often present with acute schistosomiasis rather than chronic forms of the disease. In addition clusters of acute schistosomiasis are regularly described in travelers (see the articles by Ruth N. Chunge and Jan Clerinx as well as the accompanying Editorial by Stéphane Jauréguiberry).

**Credit**: Eric Caumes