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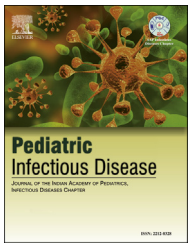

Original Article

Schistosoma haematobium and *Plasmodium falciparum* single and concomitant infections; any association with hematologic abnormalities?

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ABSTRACT

Aim: To assess the association between single infection and co-infection status of the two parasites with hematologic profiles in school children.
Methods: A cross-sectional epidemiological survey was carried out on a total of 202 school children between ages 6–18 years (mean age 11.5 ± 2.6 years). Urine and blood samples were collected by standard methods for concurrent microscopic diagnosis of *Schistosoma haematobium* and *Plasmodium falciparum* infections respectively. The following hematologic parameters; hematocrit, hemoglobin, neutrophils, leukocytes, lymphocytes and eosinophils were determined.
Results: The prevalence of single infection was 52.0% and 59.9% for *S. haematobium* and *P. falciparum* respectively, while 28.2% individuals were infected with the two parasites. The prevalence of abnormal hematologic profiles in the subjects was not associated with infection status (single or co-infection) ($P > 0.05$). There were however higher risk of developing low hemoglobin concentration with *P. falciparum* (Prevalence = 71.0%, OR = 6.0, CI = 3.2–11.0) with children with *S. haematobium* infection being weakly predisposed to developing abnormal neutrophils (Prevalence = 53.3%, OR = 1.3, CI = 0.7–2.3). Low hemoglobin associated risk in single infection with *S. haematobium* (OR = 2.0, CI = 1.1–3.6) was increased with co-infection with *P. falciparum* (OR = 4.0, CI = 1.8–8.7). There seemed to be no difference in abnormal leukocytes and eosinophils associated risk in the three infection categories.
Conclusions: There were variations in *Schistosoma* and malaria parasite induced hematologic pathologies and more studies are needed to unravel the underlying mechanisms in such variations.

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1. Introduction

Plasmodium falciparum and *Schistosoma haematobium* are two important parasites that have raised serious public health concern in sub-Saharan Africa. In the last decade, it was estimated that 85% and 90% of malaria parasite and *Schistosoma* infected populations live in sub-Saharan Africa.[1,2] While malaria is often associated with acute disease (although chronic infections are not uncommon), schistosomiasis is an insidious chronic infection with the adult worms capable of surviving for several years within the human host.[3] These parasitic infections are poverty associated and therefore oftentimes coexist in communities with poor socio-cultural development; a feature notable among many regions of sub-Saharan Africa. Besides poverty, environmental contamination of water bodies, lack of preventive measures as well as immunological interactions have been implicated as some of the factors responsible for the observed overlap in the epidemiology of the two infections.[4]

The overlapping in the distribution of malaria parasite and *Schistosoma* spp may result in high co-infection rate; an observation that had been established in malaria and intestinal helminth infections.[5,6]

Malaria has been implicated in anemia which is often caused by mechanisms such as hemolysis, increased spleenic clearance of infected and uninfected red blood cells and cytokine induced dyserythropoeisis.[7] Although little is known about the effect of *Schistosoma* infection on hematopoietic status, the visible presentation of hematuria in *Schistosoma* infected individuals could result in synergistic effect especially in concomitant infection with *P. falciparum*. With the current controversies on the effect of *Schistosoma* infection on susceptibility to malaria,[8–10] the importance of effect of co-infection of the two parasites on hematopoietic status cannot be overemphasized.

While there have been several studies on malaria and schistosomiasis epidemiology in Nigeria and other parts of sub-Saharan Africa,[11,12] there have been dearth of information on effect of concomitant occurrence of the causal organisms on hematopoietic status in endemic areas. This study therefore seeks to assess the impact of concomitant occurrence of malaria and schistosomiasis as risk factor for abnormal hematologic profiles in pupils residing in a rural community of Nigeria.

2. Methodology

The study was carried out in Ijaka-Oke community located in Yewa North Local Government Area (LGA) of Ogun State, Nigeria. Ijaka-Oke is a small village with less than 2000 dwellers. Yewa River, one of the major water bodies in the LGA passes through the village and this serves as source of water supply for all domestic purposes in the area. The village is surrounded by thick forest and cultural practices of the people such as water storage in open earthen container and stagnant waste water around dwelling places serve as mosquito breeding sites.

The study was conducted among primary school pupils of age ranging 6–18 years. A non-randomized school based, cross-sectional and descriptive study was adopted. Sample size was determined by the method of Naing et al.[13] A prevalence of 50.0% (for *S. haematobium* and *P. falciparum*) was used to compute the sample size.[14] The precision adopted was 0.8 (due to resource limitation/low population level of target subjects). The minimum sample size calculated for the study was 150 participants. A statistical power of 90.0% was used. The study was conducted between February and May, 2013. *S. haematobium* prevalence study was conducted on 202 participants. All subjects (167) without visible hematuria were screened for malaria parasite infection. The proportions of individuals in the later category with *Schistosoma* and malaria parasite infections were included in the analysis of hematologic parameters. Participants showing symptoms related to underlying chronic *S. haematobium* infection (gross hematuria) were administered Praziquantel at a dose of 40 mg/kg and then excluded from the study. Also, all the subjects with visible signs of ailment were excluded from the study.

Volunteered participants were given a clean, dry, screw-capped universal bottle carrying the same identification number as entered in the record book. Freshly passed mid-day urine samples collected between 10 and 2 pm were inspected macroscopically for gross hematuria. From the same sample, 10 mL of urine was measured and centrifuged at 4000 rpm for 4 min.[15] The supernatant was discarded and the sediment placed on clean microscope slide and covered with a coverslip. The slide was observed under the ×10 magnification. Urine samples showing elliptical eggs with terminal spine were considered positive for *S. haematobium* infection.[16]

Thick and thin blood smears were made from the blood samples collected through venipuncture and stained using 10% Giemsa stain. The presence of either ring forms or gametocytes is conclusive diagnosis of *Plasmodium* infection.[17] Blood samples that were used for the determination of hematologic parameters were stored at 4 °C and were transported to the laboratory. The following parameters were determined; the percentage cell volume in the blood (hematocrit), the hemoglobin level in the red blood cells, the total leukocyte counts, the percentage of neutrophil in the blood, the percentage of lymphocyte and the eosinophil level. The cut off values[18] for these parameters are presented in Table 1. A control group of subjects without malaria and

Table 1 – Cut off values for each of the hematologic parameters.

Blood Parameters		Age group (years)	
		6–12	13–18
Hematocrit (%)	Low	<35.0	<33.0
	Normal	35.0–45.0	33.0–51.0
Hemoglobin (g/dL)	Low	<11.5	<12.0
	Normal	11.5–15.5	12.0–16.0
Neutrophil (%)	Low	<32.0	<34.0
	Normal	32.0–61.0	34.0–64.0
Leukocyte (WBC/μL)	Normal	4.0–12.0	4.5–13.0
	High	>12.0	>13.0
Lymphocyte (%)	Normal	28.0–48.0	25.0–45.0
	High	>48.0	>45.0
Eosinophil (%)	Normal	0–3.0	0–3.0
	High	>3.0	>3.0

Table 2 – Age and sex prevalence pattern of single infection with *S. haematobium*.

Categories	No. examined	No. infected	Prevalence (%)	OR (95%CI)	*P-value*
Age (years)					
6–9	46	25	54.3	1.48 (0.74–2.99)	0.848
10–13	115	58	50.4	0.87 (0.50–1.51)	
≥14	41	22	53.6	1.38 (0.69–2.70)	
Gender					
Male	101	48	47.5	0.70 (0.40–1.22)	0.096
Female	101	57	56.4	1.43 (0.82–2.50)	
Total	202	105	52.0		

schistosomiasis were also recruited within the community and their hematologic profiles were compared with the single and co-infection statuses.

Ethical approval was obtained from the University of Ibadan/University College Hospital Institutional Ethical Review Board and the Ogun State Ministry of Health through the state Universal Basic Education Board of Ogun State. Written informed consent was obtained from the parents or guardians of the pupils after the objectives and benefits of the study had been adequately explained to them. Only the volunteered participants were included in the study.

Participants were divided into three age groups; 6–9, 10–13 and ≥14 years. The data were analyzed using SPSS statistics version 18.0 (IBM, Armonk, NY, USA). The statistical significance of differences in prevalence of single and co-infection status was determined via Chi-square analysis. Multivariate analysis was also conducted with different infection status as the dependent variable and each blood parameter as independent variable. Multivariate logistic regression analysis was used to predict the extent of association between variables and the disease occurrence. $P < 0.05$ was considered statistically significant.

3. Results

The overall prevalence of *S. haematobium* was 52.0%. The highest (54.3%) and the least prevalence (50.4%) of urogenital schistosomiasis were observed in the age groups 6–9 and 10–13 years respectively (Table 2). While prevalence of *S. haematobium* was neither age nor sex dependent ($P > 0.05$), there was higher risk of infection in female children with 56.4% prevalence level. Unlike in *S. haematobium* infection, the upper age group ≥14 years with prevalence level (67.4%) was most predisposed to *P. falciparum* infection while like in *S. haematobium* infection children in the age group 10–13 years were least infected (55.9%) (Table 3). *P. falciparum* infection pattern was similar in the two sexes and like in *S. haematobium* infection, infection was not associated with age and sex of the children ($P > 0.05$). The prevalence of co-occurrence of the two parasites was highest (48.4%) and least (27.5%) in age groups 6–9 and 10–13 years respectively (Table 4). Female children (36.9%) were more predisposed to concomitant *Schistosoma*-malaria parasites infection than their male counterparts (31.3%). Co-infection status like in the case of single infection with *S. haematobium* and *P. falciparum* was not age and sex dependent ($P > 0.05$).

Generally, there were no significant differences in the proportion of subjects which showed abnormal hematologic profiles in the three categories of infection status (*S. haematobium*, *P. falciparum* and co-infection) ($P > 0.05$). However, there were wide variations in the predisposition to abnormal hematologic profiles in the three infection status. The risk (OR = 6.0, CI = 3.2–11.0) of developing low hemoglobin concentration was highest in children with *P. falciparum* infection. Low hemoglobin associated risk in single infection with *S. haematobium* (OR = 2.0, CI = 1.1–3.6) was increased in co-infection with *P. falciparum* (OR = 4.0, CI = 1.8–8.7). Children with single infection with *S. haematobium* were weakly predisposed to developing of abnormal neutrophil (OR = 1.3, CI = 0.7–2.3) while there was no risk associated with single infection with *P. falciparum* and co-infection status. There seemed to be no difference in abnormal leukocyte and eosinophil associated risk in the three infection categories. The risk of developing abnormal lymphocyte was higher in *S. haematobium* infection (OR = 302.8, CI = 84.6–1083.2) than in malaria parasite infection (OR = 176.5, CI = 59.6–523.1), however, co-infection with the two parasites (OR = 108.2, CI = 29.5–396.0) seemed to reduce abnormal lymphocyte associated risk (Table 5). There were no significant differences in the mean values of hematologic profiles in the different infection statuses (Table 6).

Table 3 – Age and sex prevalence pattern of single infection with *P. falciparum*.

Categories	No. examined	No. infected	Prevalence (%)	OR (95%CI)	*P-value*
Age (years)					
6–9	31	20	64.5	1.27 (0.57–2.86)	0.390
10–13	102	57	55.9	0.65 (0.34–1.24)	
≥14	34	23	67.4	1.52 (0.69–3.37)	
Gender					
Male	83	49	59.0	0.93 (0.50–1.73)	0.741
Female	84	51	60.7	1.07 (0.58–1.99)	
Total	167	100	59.9		

Table 4 – Age and sex prevalence pattern of co-infection status with *S. haematobium* and *P. falciparum*.

Categories	No. examined	No. coinfected	Prevalence (%)	OR (95%CI)	*P-value*
Age (years)					
6–9	31	15	48.4	2.10 (0.95–4.64)	0.089
10–13	102	28	27.5	0.47 (0.24–0.90)	
≥14	34	14	41.2	1.47 (0.68–3.18)	
Gender					
Male	83	26	31.3	0.78 (0.41–1.48)	0.515
Female	84	31	36.9	1.28 (0.68–2.44)	

4. Discussion

Age and sex prevalence pattern of schistosomiasis in the study area is similar to a study previously conducted in some of the other communities within the LGA.[14] Lack of potable water supply which is a general problem common to all the communities in the LGA and presence of appropriate snail intermediate host for intramolluscan development of parasite in the water bodies that serve as alternative water supply are factors of epidemiological importance. Poor knowledge about the cause of the disease, occupation and some sociodemographic characteristics like religion have also been reported to be the predisposing factors to schistosomiasis in the LGA.[19]

Malaria has been acclaimed a serious health problem in rural communities. This is evident in this study prevalence (60%) being significantly higher than the values reported (7–54%) in both urban and peri-urban areas of Nigeria and other African countries.[20–22]

Schistosomiasis and malaria are highly endemic in the study area with more than half of the children harboring single infection due to either *S. haematobium* or *P. falciparum*. The prevalence of concomitant occurrence of *S. haematobium* and *P. falciparum* in our study is significantly higher than 2.3% prevalence level reported in Ethiopia population.[23] The high level of co-infection of the two parasites suggests some overlaps in their epidemiology of which lack of sensitization and provision of basic social amenities could be the implicating factors. Understanding the epidemiology of these parasitic infections in the target population and their supposed additive or synergistic effects in impairing normal hematologic profile is important as findings may support design of integrated disease control strategies.[24]

The high risk of low hemoglobin in *P. falciparum* infected children has been widely reported.[21,22] Malaria contribution to anemia has been linked to a number of mechanisms including destruction of parasitized red blood cells, shortening of life span of non-parasitized red blood cells and decreased production of red blood cells in the bone marrow.[7,25] However, schistosomiasis seems not to mediate anemic condition in *S. haematobium* infected children in present study. Although there have been conflicting reports on the relationship between schsitosomiasis and anemia,[26] many studies have favored the association of the disease with anemia.[27–29] Proposed mechanisms of schistosomiasis-mediated anemia have been summarized into: (i) iron deficiency due to extracorporeal loss; (ii) anemia of inflammation; (iii) splenic sequestration and (iv) autoimmune hemolysis.[25]

Other etiologies of low neutrophil like severe bacterial infection and vitamin B12 deficiency are suggested since no high risk of low neutrophil was observed in any of the infection status. This may probably explain the low prevalence of individuals with high leukocytes in the populace since neutrophils can make up about 70% of the white blood cells.

Table 5 – Hematological parameters in association with different infection status.

Blood parameters	Classification	Control ($n = 32$)	Infection status			*P-value*
			Malaria ($n = 100$)	*Schistosoma* ($n = 92$)	Co-infection ($n = 57$)	
Hematocrit	Low	18 (56.2)	47 (47.0)	40 (43.5)	28 (49.1)	0.779
	Normal	14 (43.8)	53 (53.0)	52 (56.5)	29 (50.9)	
	OR (95%CI)	1.7 (0.6–4.4)	0.8 (0.5–1.4)	0.6 (0.3–1.1)	0.9 (0.4–1.9)	
Hemoglobin	Low	19 (59.4)	71 (71.0)	54 (58.7)	38 (66.7)	0.184
	Normal	13 (40.6)	29 (29.0)	38 (41.3)	19 (33.3)	
	OR (95%CI)	2.1 (0.8–5.8)	6.0 (3.2–11.0)	2.0 (1.1–3.6)	4.0 (1.8–8.7)	
Neutrophil	Low	12 (37.5)	48 (48.0)	49 (53.3)	28 (49.1)	0.731
	Normal	20 (62.5)	52 (52.0)	43 (46.7)	29 (50.9)	
	OR (95%CI)	0.4 (0.1–2.0)	0.9 (0.5–1.5)	1.3 (0.7–2.3)	0.9 (0.5–1.9)	
Leukocyte	Normal	28 (87.5)	89 (89.0)	81 (88.0)	49 (86.0)	0.880
	High	4 (9.4)	11 (11.0)	11 (12.0)	8 (14.0)	
	OR (95%CI)	0.02 (0.004–0.1)	0.02 (0.01–0.04)	0.02 (0.01–0.04)	0.03 (0.01–0.08)	
Lymphocyte	Normal	3 (9.4)	7 (7.0)	5 (5.4)	5 (8.8)	0.798
	High	29 (90.6)	93 (93.0)	87 (94.6)	52 (91.2)	
	OR (95%CI)	93.4 (17.4–501.9)	176.5 (59.6–523.1)	302.8 (84.6–1083.2)	108.2 (29.5–396.0)	
Eosinophil	Normal	26 (81.3)	67 (67.0)	60 (65.2)	35 (61.4)	0.785
	High	6 (18.7)	33 (33.0)	32 (34.8)	22 (38.6)	
	OR (95%CI)	0.05 (0.02–0.2)	0.2 (0.1–0.4)	0.3 (0.2–0.5)	0.4 (0.2–0.8)	

Table 6 – Mean values of blood parameters and infection status of the study participants.

Blood Parameters	Mean ± S.D				*P-value*
	Control	Schistosomiasis	Malaria	Co-infection	
Hematocrit (%)	34.8 ± 3.3	34.6 ± 3.3	34.4 ± 3.3	34.2 ± 3.3	0.999
Hemoglobin (g/dL)	11.2 ± 1.1	11.2 ± 1.1	11.0 ± 1.0	11.0 ± 1.1	0.999
Neutrophil (%)	32.0 ± 7.7	32.3 ± 8.1	32.9 ± 8.2	33.6 ± 8.6	0.998
Leukocyte (WBC/μL)	7.9 ± 2.3	8.5 ± 2.9	8.2 ± 2.7	8.7 ± 3.1	0.998
Lymphocyte (%)	60.2 ± 7.5	60.0 ± 8.1	59.5 ± 8.5	58.9 ± 8.8	0.998
Eosinophil (%)	2.9 ± 1.3	3.0 ± 1.2	3.1 ± 1.4	3.1 ± 1.2	0.999

Higher occurrence of lymphocyte in schistosomiasis and malaria single infection status in our study was in contrast to other studies.[30–32] The high level could be caused by parasites activation of lymphocytes and its subsequent peripheral blood distribution in highly endemic areas.[22] The decrease in risk associated with abnormal lymphocytes in co-infection status could be due to parasites' interactions which mediate the release of cytokines that downregulate the production of lymphocyte. The lack of association between *Schistosoma* infection and eosinophil in this study needs further investigation as the latter has been frequently reported as strong indicator of schistosomiasis.

This study has demonstrated the high endemicity of schistosomiasis and malaria in children and also showed that concurrent occurrence of *S. haematobium* and *P. falciparum* is very common in the study area. However, the effect of single infection with either *S. haematobium* or *P. falciparum* on presenting abnormal hematologic profiles varied with co-infection of the two sometimes reducing the risk of some of these abnormal blood profiles. Whatever the case may be, integrated control approach against the two parasites is advocated. Further studies on *Schistosoma*-malaria co-infection that will put in place possible confounders are recommended.

Contributors

OAM designed and supervised both field sampling and laboratory procedures. OA and EEI gathered both field and laboratory data. OTS participated in field sampling. OTS and OA analyzed the data and wrote the first draft of the manuscript. OAM revised the manuscript. All the authors approved the final draft of the manuscript.

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Conflicts of interest

All authors have none to declare.

REFERENCES

1. Chitsulo L, Engels D, Montresor A, Savioli L. The global status of schistosomiasis and its control. *Acta Trop*. 2000;77:41–51.
2. Greenwood B, Mutabingwa T. Malaria in 2002. *Nature*. 2002;415:670–672.
3. Maizels RM, Balic A, Gomez-Escobar N, Nair M, Taylor MD, Allen JE. Helminth parasites—masters of regulation. *Immunol Rev*. 2004;201:89–116.
4. Booth M. The role of residential location in apparent helminth and malaria infection. *Trends Parasitol*. 2006;22:359–362.
5. Keiser J, N'Goran EK, Traore M, et al. Polyparasitism with *Schistosoma mansoni*, geohelminths, and intestinal protozoa in rural Cote d'Ivoire. *J Parasitol*. 2002;88:461–466.
6. Adrienne E, Edridah M, Jennifer K. Epidemiology of helminth infection and their relationship to clinical malaria in southwest Uganda. *Trans R Soc Trop Med Hyg*. 2005;99:18–24.
7. McDevitt MA, Xie J, Gordeuk V, Bucala R. The anemia of malaria infection: the role of inflammatory cytokines. *Curr Hematol Rep*. 2004;3:97–106.
8. Nacher M, Singhasivanon P, Yimsamran S, et al. Intestinal helminth infections are associated with increased incidence of *Plasmodium falciparum* malaria in Thailand. *J Parasitol*. 2002;88:55–58.
9. Sokhna C, Le Hesran JY, Mbaye PA, et al. Increased malaria attacks among children presenting concomitant infections by *S. mamsoni* in Senegal. *Malar J*. 2004;3:43.
10. Spiegel A, Tall A, Raphenon G, Trape JF, Druilhe P. Increased frequency of malaria attacks in subjects co-infected by intestinal worms and *Plasmodium falciparum* malaria. *Trans Roy Soc Trop Med Hyg*. 2003;97:198–199.
11. Klinkenberg E, McCall PJ, Hastings IM, et al. Malaria and irrigated crops, Accra, Ghana. *Emerg Infect Dis*. 2005;11:1290–1293.
12. Salawu OT, Odaibo AB. Urogenital schistosomiasis and urological assessment of hematuria in preschool-aged children in rural communities of Nigeria. *J Pediatr Urol*. 2014;10:88–93.
13. Naing L, Winn T, Rusli BN. Practical issues in calculating the sample size for prevalence studies. *Arch Orofac Sci*. 2006;1:9–14.
14. Hassan A, Ntiaidem U, Morenikeji O, et al. Urine turbidity and microhaematuria as rapid assessment indicators for *Schistosoma haematobium* infection among school children in endemic areas. *Am J Infect Dis*. 2012;8:60–64.
15. Stoller M, Carrol PR. In: Tieney LM, Mcphee SJ, Papadakis MA, eds. *Urology in Current Medical Diagnosis and Treatment*. 42nd ed. Lange Med. Books; 2003:907.
16. Loker ES. A comparative study of the life-histories of mammalian schistosomes. *Parasitol*. 1983;87:369–373.
17. Warhurst DC, Williams JE. Laboratory diagnosis of malaria. *J Clin Pathol*. 1996;49:533–538.

18. Porter RS, Kaplan JL, eds. *The Merck Manuals of Diagnosis and Therapy*. 19th ed. Merck Sharp & Dohme Corp., a subsidiary of Merck & Co, Inc; 2011. www.merckmanuals.com.
19. Salawu OT, Odaibo AB. Schistosomiasis transmission; socio-demographic, knowledge and practices as transmission risk factors in pregnant women. *J Parasit Dis* DOI 10.1007/s12639-014-0454-2.
20. Oyibo WA, Agomo CO, Anorlu RI, Agomo P. Prevalence of malaria in pregnant women in Lagos, South-West Nigeria. *Korean J Parasitol*. 2009;47:179–183.
21. Maina R, Walsh D, Gaddy C, et al. Impact of *Plasmodium falciparum* infection on hematological parameters in children living in Western Kenya. *Malar J*. 2010;9:S3–S4.
22. Adedotun AA, Salawu OT, Morenikeji OA, Odaibo AB. Plasmodial infection and hematological parameters in febrile patients in a hospital in Oyo town, South-western Nigeria. *J Public Health Epidemiol*. 2013;5:144–148.
23. Deribew K, Tekeste Z, Petros B. Urinary schistosomiasis and malaria associated anemia in Ethiopia. *Asian Pac J Trop Biomed*. 2013;3:307–310.
24. Kinung'hi SM, Magnussen P, Kaatano GM, Kishamawe C, Vennervald BJ. Malaria and helminth co-infections in school and preschool children: a cross-sectional study in Magu District, North-Western Tanzania. *PLoS One*. 2014;9:e86510.
25. Menendez C, Fleming AF, Alonso PL. Malaria related anemia. *Parasitol Today*. 2000;16:469–476.
26. Friedman JF, Kanzaria HK, McGarvey ST. Human schistosomiasis and anemia: the relationship and potential mechanisms. *Trends Parasitol*. 2005;21:386–392.
27. Prual A, Daouda H, Develoux M, Sellin B, Galan P, Hercberg S. Consequences of *Schistosoma haematobium* infection on the iron status of schoolchildren in Niger. *Am J Trop Med Hyg*. 1992;47:291–297.
28. Sturrock RF, Kariuki HC, Thiongo FW. Schistosomiasis mansoni in Kenya: relationship between infection and anemia in schoolchildren at the community level. *Trans R Soc Trop Med Hyg*. 1996;90:48–54.
29. Koukounari A, Fenwick A, Whawell S, et al. Morbidity indicator of schistosoma mansoni: relationship between infection and anemia in Uganda school children before and after Praziquantel and Albendazole chemotherapy. *Am J Trop Med Hyg*. 2006;75:278–286.
30. Richards MW, Behrens RH, Doherty JF. Short report: hematological changes in acute, imported *Plasmodium falciparum* malaria. *Am J Trop Med Hyg*. 1998;59:859.
31. Erhart LM, Yingyuen K, Chuanak N, et al. Haematologic and clinical indices of malaria in a semi-immune population. *Am J Trop Med Hyg*. 2004;70:8–14.
32. Mohammed EHA, Eltayeb M, Ibrahim H. Hematological and biochemical morbidity of *Schistosoma haematobium* in school children in Sudan. *Sultan Qaboos Univ Med J*. 2006;6:59–64.