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THE EPIDEMIOLOGY OF SCHISTOSOMIASIS IN EGYPT: MENOFIA GOVERNORATE

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Abstract. Health questionnaires and parasitologic examinations of urine and stool were performed upon a stratified random sample of 10,899 individuals from 1,537 households in 27 rural communities in Menofia Governorate in Egypt in 1992 to investigate the prevalence of, risk factors for, and changing pattern of infection with *Schistosoma* sp. in the governorate. A subset, every fifth household, or 1,480 subjects, had physical and ultrasound examinations to investigate prevalence of and risk factors for morbidity. The prevalence of *S. mansoni* ranged from 0.3% to 72.9% and averaged 28.5%. The geometric mean egg count was 81.3 eggs/gram of stool. Age-stratified prevalence and intensity of infection was 30–40% and 60–80 eggs/gram of stool from the age of 10 onward; males had higher infection rates and ova counts than females in all age groups > 10 years old. *Schistosoma haematobium* was rare, being consequential in only 1 community. Risk factors for *S. mansoni* infection were male gender; age > 10 years; living in smaller communities; exposures to canal water; history of or treatment for schistosomiasis or blood in the stool; detection of splenomegaly by either physical or ultrasound; and ultrasound-detected periportal fibrosis (PPF). The more severe grades of PPF were rarely (21 of 1,450 examinations) detected. Risk factors for morbidity, i.e., ultrasound-detected PPF, were similar to those for infection. *Schistosoma mansoni* has almost totally replaced *S. haematobium* in Menofia. The prevalence of *S. mansoni* in rural communities remains high and average intensities of infection are moderate. The association of morbidity with schistosomal infection was variable and is obviously markedly influenced by both the frequent use of antischistosomal chemotherapy in communities in Menofia and by the prevalence of complications from chronic viral hepatitis in the population: hepatomegaly did not correlate with infection; PPF and splenomegaly, however, were related to *S. mansoni* infection in both individuals and communities.

Menofia is in the southern part of the Nile Delta 100–150 km north of Cairo, Egypt.[1] The 2 branches of the River Nile are its southern eastern, and western borders. Irrigation canals in Menofia branch from both the Rashid and Demiatta branches of the Nile. The 1,600,000 inhabitants of the governorate are primarily employed in agriculture. However, there has been a relatively large increase in manufacturing during the past few years, particularly in southern Menofia, nearest to Cairo. Many males work both in factories earning wages and in the fields planting and harvesting crops.

Under sponsorship of the Egyptian Ministry of Health/United States Agency for International Development-sponsored Schistosomiasis Research Project, we investigated the prevalence and intensity of infection with *Schistosoma* sp., the prevalence and magnitude of morbidity caused by schistosomiasis, the changing pattern of distribution of *S. mansoni* and *S. haematobium*, and the determinants of infection and morbidity in a random sample of the rural inhabitants of Menofia Governorate in 1992. Herein, we report the results of this survey.

SUBJECTS AND METHODS

The sample size, selected by multistage, stratified, random sampling, was calculated to detect a prevalence of *Schistosoma* sp. as low as 5% in villages or ezbas (satellite group of dwellings) with an 80% precision and 90% confidence level. The findings are considered representative of the rural areas of the entire governorate.[1] The total sample population was 10,899 individuals from 1,537 households in 23 ezbas and 4 villages. Randomizing took place at the village/ezba and household levels but the total household was included in the study sample. The interview technique for collecting vital, environmental, sociodemographic, and medical data has been described in detail.[1] Data collection forms were used for recording village, household, family, and individual information. Quantitative microscopic counting of *Schistosoma* ova in a single stool specimen from 8,254 subjects using a modified Kato technique (2 slides) and in urine from 5,113 subjects using the Nucleopore (Pleasanton, CA) filter technique were performed as described.[2]

Physical examination and abdominal ultrasonography were performed by trained physicians upon 1,581 inhabitants from 285 households as described.[3] Children less than 5 years old were excluded from the physical and ultrasonographic examinations by protocol, but some were examined and these results are included.

Not all data from the 1,581 subjects having physical and ultrasonographic examinations were available for complete analysis for the following reasons: 1) 745 (47.1%) did not provide urine specimens for parasitology, hematuria, and proteinuria examinations; 2) 131 (8.3%) did not provide stool specimens for parasitology examinations; 3) 72 (4.6%) did not have height and weight recorded; 4) 154 (9.7%) did not respond to the question regarding a history of schistosomiasis; 5) 155 (9.8%) did not respond to the question regarding prior treatment for schistosomiasis; and 6) 19 (1.2%) failed to respond to the question regarding blood in the stools. The most important among these omissions are the absence of stool specimens from 8% of the subjects, which excludes them from any analyses based upon *S. mansoni* infection; and the absence of height measurement in 5%, which excludes them from any analyses of hepatic enlargement since height was used to adjust for differences in body size.[3]

All data was transferred from the data collection forms to standard precoded sheets for computer entry using Epi-Info 5.01b (Centers for Disease Control and Prevention, Atlanta,

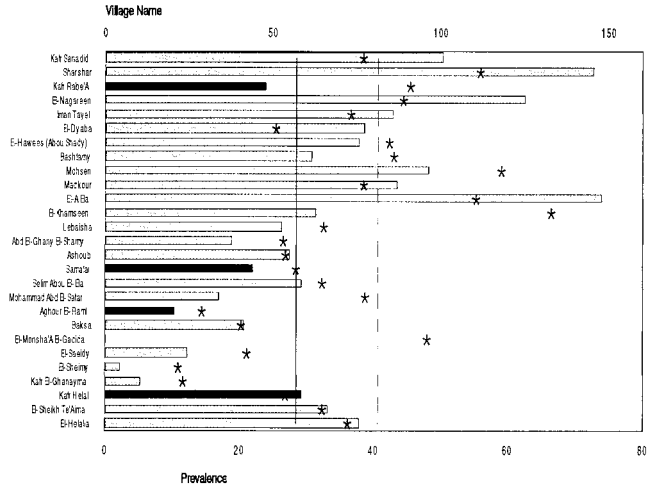

FIGURE 1. Prevalence (%) (**bars**) and intensity (**stars**) of *Schistosoma mansoni* infection in the surveyed communities in Menofia Governorate. Solid horizontal bars show prevalences in villages, shaded horizontal bars show prevalences in ezbas, the solid vertical line is the mean prevalence for all communities, the broken vertical line is the mean intensity of infection, and the stars are the geometric mean egg counts/gram of stool for each community.

GA). Data was verified by the Statistical Core Team prior to analysis.[1] Survey Data Analysis (SUDAAN) software was used to calculate *Schistosoma* prevalence and the geometric mean egg count (GMEC) in entirety and stratified by community, gender, and age as described.[1] Further analysis was performed after transformation to SPSS/PC + 4.01 (SPSS, Inc., Chicago, IL). This software was used to calculate odds ratios (ORs) and 95% confidence intervals (CIs). Graphic presentations were prepared using Harvard Graphics 3.0 (Software Publishing Corp., Mountain View, CA).

RESULTS

The prevalence of *S. mansoni* in the 27 surveyed communities ranged from 0.3% to 72.9% and averaged 28.5 ± 0.5% (± SEM) (Figure 1). The average intensity of infection in the surveyed villages was 81.3 GMEC/g of stool and ranged from 21.8 to 133.1 GMEC/g of stool. In most cases the ezbas had a higher prevalence and GMEC than their mother villages. The OR of having a *S. mansoni* infection in inhabitants of ezbas in comparison with village dwellers was 1.67 with a 95% CI of 1.49–1.87 (Table 1). Prevalence and intensity of infection was in the 30–40% and 60–80 GMEC/g of stool ranges, respectively, from the age of 10 onward (Figure 2). Except for the youngest age group, i.e., those less than 5 years old, infection rates were higher in males than in females. The intensity, like the prevalence, of infection was greater in males than females in every age group.

Schistosoma haematobium infection was detected in 14 communities. However, in 9 of these only 1 subject had ova detected, suggesting no transmission of that species was occurring locally. Only one ezba, Kafr Ghanayma, had a sufficiently high prevalence (16.7%) to suggest that local transmission was important. This community had one of the lowest prevalence (12.3 %) and intensity of *S. mansoni* infection (Figure 1).

Risk factors significantly associated with *S. mansoni* infection in Menofia were an age >10 years old, male gender, living in ezbas; males bathing in, women washing clothing or utensils in, or children swimming or playing in canals; a history of, or treatment for, schistosomiasis; and recent history of blood in the stool (Table 1). Abdominal pain and hepatomegaly detected by physical examination were not associated with *S. mansoni* infection, but splenomegaly on physical examination had an OR of 4.19 in infected subjects compared with noninfected subjects.

Abdominal ultrasonography confirmed the physical examination findings: liver size, in either the midclavicular line (MCL) or midsternal line (MSL), did not correlate with *S. mansoni* infection. However, splenomegaly was detected twice as frequently in *S. mansoni*-infected subjects than in noninfected ones; 134 (9.4%) of 1,423 had hepatomegaly and 53 (3.6%) of 1,480 had splenomegaly detected by physical examination, while 289 (20.6%) of 1,400 had ultrasonography-detected hepatic enlargement in the MCL and 444 (30.7%) of 1,447 had ultrasonography-detected splenomegaly. Left lobe hepatic enlargement was detected by ultrasonography in 146 (10.4%) of 1,401 subjects. Periportal fibrosis (PPF) was associated with *S. mansoni* infection: the OR of *S. mansoni*-infected subjects in comparison with noninfected subjects having PPF was 1.71. This relationship between infection and PPF was more apparent in adults than in children. A total of 514 (35.4%) of 1,450 subjects had ultrasonography-detected PPF. However, only 20 had grade II lesions and only 1 subject had grade III PPF. This latter subject was a 56-year-old farmer from the village having the highest GMEC who had a history of heavy exposure to canal water and prior treatment for schistosomiasis. He had 100 *S. mansoni* ova/g of stool and hepatomegaly and splenomegaly on both physical examination and ultrasonography. His spleen was enlarged to 15 cm in the longitudinal axis and portal vein dilation and periumbilical vein collaterals were detected during the ultrasonographic examination.

Morbidity, as measured by PPF detected during the ultrasonographic examination, increased with age, with male gender, and with a domicile in ezbas rather than in villages. Exposures to canal water increased the morbidity rate but only a history of bathing, swimming, or playing in the water by children was statistically significant (Table 2). A history of schistosomiasis, or treatment for schistosomiasis, and *S. mansoni* ova in the stools were all associated with morbidity. A history of blood in the stools or abdominal pain correlated with PPF in adults, but not in children. Hepatomegaly detected during a physical examination was less frequent (OR = 0.54) in those with PPF, but splenomegaly was more common (OR = 2.53). Hepatomegaly in both the MCL (306 of 1,506, 20.3%) and the MSL (151 of 1,506, 10.0%) and splenomegaly (471 of 1,568, 30.0%) were frequent ultrasonographic findings. The frequency of detecting hepatomegaly by physical examination was less than half that detected by ultrasonography. However, splenomegaly was detected 8 times as often by ultrasonography than by physical examination. Ultrasonography-detected hepatomegaly in both the MCL and MSL were less frequent in those having PPF and splenomegaly had an association with PPF only in adults.

The prevalence of both hepatomegaly and splenomegaly detected by physical examination increased with age. Ultra-

TABLE 1

Odds ratio and 95% confidence limits for risk factors for infection with *Schistosoma mansoni* in El-Menofia Governorate*

Risk factor	Total in group	Infected No. (%)	Odds ratio	Confidence limits
Demographics				
Age groups (years)				
0–10	2,211	341 (15.4)		
11–20	2,125	752 (35.4)	3.00	2.60–3.47
21–35	1,722	576 (33.4)	2.76	2.37–3.21
36–55	1,357	404 (29.8)	2.32	1.97–2.74
>55	709	165 (23.3)	1.66	1.33–2.05
Gender				
Female	4,059	856 (21.1)		
Male	4,065	1,382 (34.0)	1.93	1.74–2.13
Domicile				
Village (≥500 houses)	2,326	494 (21.2)		
Ezba (<500 houses)	5,928	1,839 (31.0)	1.67	1.49–1.87
Exposure to canal water				
Bathing (males)				
No	1,903	478 (25.1)		
Yes	1,515	750 (49.5)	2.92	2.53–3.38
Washing (females)				
No	1,892	309 (16.3)		
Yes	1,770	485 (27.4)	1.93	1.65–2.27
Playing (children <15 years old)				
No	1,678	260 (15.5)		
Yes	854	310 (36.3)	3.11	2.56–3.77
Clinical findings				
History of schistosomiasis				
No	5,630	1,375 (24.4)		
Yes	1,271	560 (44.1)	2.44	2.15–2.77
Prior treatment of schistosomiasis				
No	5,780	1,444 (25.0)		
Yes	1,249	548 (43.9)	2.34	2.06–2.66
History of blood in stools				
No	1,332	354 (26.6)		
Yes (total)	145	79 (54.5)	3.31	2.33–4.69
<15 years	52	26 (50.0)	3.97	2.22–7.11
≥15 years	93	53 (57.0)	2.93	1.89–4.54
History of abdominal pain				
No	977	281 (28.8)		
Yes	497	152 (30.6)	1.09	0.86–1.38
<15 years	189	50 (26.5)	1.34	0.90–2.00
≥15 years	308	102 (33.1)	0.95	0.71–1.27
Hepatomegaly in MCL (by PE)				
No	1,289	371 (28.8)		
Yes	134	46 (34.3)	1.29	0.89–1.88
<15 years	47	16 (34.0)	1.87	0.99–3.54
≥15 years	87	30 (34.5)	3.68	1.99–6.79
Splenomegaly (by PE)				
No	1,427	403 (28.2)		
Yes	53	33 (62.3)	4.19	2.38–7.39
<15 years	6	3 (50.0)	3.46	0.69–17.32
≥15 years	47	30 (63.8)	3.68	1.99–6.79
Ultrasonography				
Hepatomegaly in MCL				
No	1,111	351 (31.6)		
Yes	289	59 (20.4)	0.56	0.41–0.76
<15 years	135	19 (14.1)	0.48	0.28–0.82
≥15 years	154	40 (26.0)	0.64	0.43–0.94

TABLE 1
Continued

Risk factor	Total in group	Infected No. (%)	Odds ratio	Confidence limits
Hepatomegaly in MSL				
No	1,255	372 (29.6)		
Yes	146	37 (25.3)	0.81	0.54–1.19
<15 years	78	15 (19.2)	0.78	0.43–1.41
≥15 years	68	22 (32.4)	0.94	0.56–1.60
Splenomegaly				
No	1,003	251 (25.0)		
Yes	444	179 (40.3)	2.02	1.60–2.57
<15 years	120	43 (35.8)	2.20	1.42–3.41
≥15 years	324	136 (42.0)	1.76	1.32–2.34
Periportal fibrosis				
No	936	241 (25.7)		
Yes (≥3 mm)	514	191 (37.2)	1.71	1.35–2.15
<15 years	158	45 (28.5)	1.45	0.96–2.20
≥15 years	356	146 (41.0)	1.68	1.26–2.23
Grade I (3–<5 mm)	493	181 (36.7)	2.00	1.55–2.13
Grade II (5–<7 mm)	20	9 (45.0)	2.36	0.97–5.76
Grade III (≥7 mm)	1	1 (100.0)		

* MCL = midclavicular line; PE = physical examination; MSL = midsternal line.

sonography-detected hepatomegaly in both the MCL and MSL had a higher prevalence in children and older adults than in younger adults, while both splenomegaly and PPF were more frequent in adults than in children (Figure 3).

DISCUSSION

In 1935, Azim reviewed the early 20th century epidemiologic studies of schistosomiasis in Egypt.[4] He noted that *S. haematobium* was the only species present in Upper Egypt and that it was more prevalent in lower Egypt than *S. mansoni*. The first countrywide survey was published by Scott in 1937.[5] At that time, *S. haematobium* infections were very common in rural areas in the Nile Delta, with a prevalence ranging from 55% to 75% in villages. The prevalence of *S. mansoni* varied widely in the Delta, ranging from 60% in the north to 4% in the south nearer to Cairo, e.g., in Menofia.

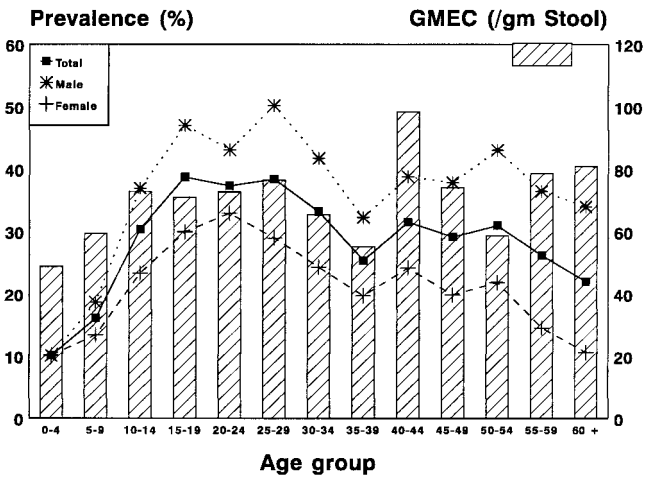

FIGURE 2. Age- and gender-adjusted prevalence and intensity of *Schistosoma mansoni* infection in 8,254 randomly selected subjects in Menofia Governorate. GMEC = geometric mean egg count.

Later studies in the 1950s confirmed and extended Scott's findings.[6] However, following construction of the Aswan High Dam in the 1960s, changes in the distribution of *Schistosoma* infections took place. Decreases in the prevalence of human *S. haematobium* infections in conjunction with increases in *S. mansoni* and a replacement of the reservoir of the former, *Bulinus truncatus*, with the reservoir of the latter, *Biomphalaria alexandrina* were reported.[7–10] For instance, the prevalence of *S. mansoni* and *S. haematobium* in Menofia were 20% and 19% in 1983 compared with 5% and 3% in 1990.[9,10] In addition, almost half of those examined with *S. mansoni* infections had > 100 ova/g of stool while none of those with *S. haematobium* had > 50 ova/10 ml of urine, clearly documenting major differences in intensities of infection between the 2 species. The most recent reported Ministry of Health data on prevalence and intensity of schistosomiasis in Menofia was based on 1985 data.[11] Our results confirm that *S. mansoni* has almost completely replaced *S. haematobium* in Menofia. The prevalence of *S. haematobium* is very focal and has decreased to 1.1% in the governorate. Only 1 of the 27 communities we investigated appeared to be having sustained transmission of this parasite. However, we did not investigate snails in El-Khamseen.

The smaller communities (ezbas) had higher prevalences of infection than the larger villages for several reasons: 1) medical care is less readily available in the smaller communities; 2) the larger communities have shop keepers, school teachers, commuting factory workers, and others who do not work in agriculture, and have less exposure to infection, while the smaller communities are almost exclusively farmers; 3) those living in the larger communities are usually better educated, and would be more likely to avoid exposures to infectious water than those living in ezbas; and 4) ezbas have less facilities, e.g., piped water supplies, sewage disposal, electricity, than the villages, which increase exposures to infection. This association of prevalence of infection to the intensity of exposure of the population has been well

TABLE 2

Odds ratio and 95% confidence limits for risk factors for morbidity (periportal fibrosis) with *Schistosoma mansoni* in El-Menofia Governorate*

Risk factor	Total in group	Infected No. (%)	Odds ratio	Confidence limits
Demographics				
Age groups (years)				
0–10	399	93 (23.3)		
11–20	427	167 (39.1)	2.11	1.56–2.86
21–35	330	129 (39.1)	2.11	1.53–2.91
36–55	275	97 (35.3)	1.79	1.28–2.52
>55	124	57 (46.0)	2.80	1.83–4.27
Gender				
Female	794	215 (27.1)		
Male	761	328 (43.1)	2.04	1.65–2.52
Domicile				
Village (≥500 houses)	451	137 (30.4)		
Ezba (<500 houses)	1,130	414 (36.6)	1.33	1.05–1.68
Exposure to canal water				
Bathing (males)				
No	374	157 (42.0)		
Yes	296	139 (47.0)	1.22	0.90–1.66
Washing (females)				
No	398	108 (27.1)		
Yes	352	100 (28.4)	1.07	0.77–1.47
Playing (children <15 years old)				
No	353	82 (23.2)		
Yes	175	64 (36.6)	1.91	1.28–2.83
Parasitologic findings				
S. mansoni infection				
No	1,018	323 (31.7)		
Yes	432	191 (44.2)	1.71	1.35–2.15
<100 ova/gram of stool	272	111 (40.8)	1.48	1.13–1.95
≥100 ova/gram of stool	160	80 (50.0)	2.15	1.54–3.01
Clinical findings				
History of schistosomiasis				
No	1,142	380 (33.3)		
Yes	254	115 (45.3)	1.66	1.26–2.19
Prior treatment of schistosomiasis				
No	1,167	385 (33.0)		
Yes	248	116 (46.8)	1.78	1.32–2.36
History of blood in stools				
No	1,402	476 (34.0)		
Yes	160	71 (44.4)	1.55	1.11–2.16
<15 years	56	19 (33.9)	1.52	0.85–2.73
≥15 years	104	52 (50.0)	1.52	1.01–2.28
History of abdominal pain				
No	1,037	350 (33.8)		
Yes (total)	522	196 (37.5)	1.18	0.95–1.47
<15 years	188	53 (28.2)	1.17	0.80–1.73
≥15 years	334	143 (42.8)	1.14	0.87–1.49
Hepatomegaly in MCL (by PE)				
No	1,356	496 (36.6)		
Yes	143	34 (23.8)	0.54	0.36–0.81
<15 years	47	6 (12.8)	0.39	0.16–0.94
≥15 years	96	28 (29.2)	0.55	0.34–0.87
Splenomegaly (by PE)				
No	1,505	513 (34.1)		
Yes	60	34 (56.7)	2.53	1.50–4.26
<15 years	5	3 (60.0)	4.33	0.72–26.14
≥15 years	55	31 (56.4)	1.96	1.23–3.79

TABLE 2
Continued

Risk factor	Total in group	Infected No. (%)	Odds ratio	Confidence limits
Ultrasonography				
Hepatomegaly in MCL				
No	1,200	445 (37.1)		
Yes	306	87 (28.4)	0.67	0.51–0.89
<15 years	143	40 (28.0)	1.14	0.75–1.74
≥15 years	163	47 (28.8)	0.51	0.35–0.74
Hepatomegaly in MSL				
No	1,355	492 (36.3)		
Yes	151	40 (26.5)	0.63	0.43–0.92
<15 years	81	21 (25.9)	0.99	0.58–1.69
≥15 years	70	19 (27.1)	0.50	0.29–0.86
Splenomegaly				
No	1,097	335 (30.5)		
Yes	471	210 (44.6)	1.83	1.47–2.29
<15 years	124	37 (29.8)	1.29	0.83–1.99
≥15 years	347	173 (49.9)	1.83	1.40–2.39

* MCL = midclavicular line; PE = physical examination; MSL = midsternal line.

documented.[12] The present investigation, using univariant analysis, documented that a history of 3 different types of increased exposure to canal water increased frequency of *S. mansoni* infection by 2- or 3-fold. Since females do not frequently bath in canals, males seldom wash dishes or clothing in canals, and children are more likely than adults to play or swim in the water, these variables were stratified into the appropriate age and gender groups. When the data are not stratified in this manner prior to analysis, the results are meaningless. The higher prevalence of infection in males than in females and in the 11–55-year-old age groups could also be explained by their greater exposure to canal water while farming.[12]

Although the prevalence of *S. mansoni* in Menofia remains relatively high, i.e., 28.5%, the intensity of infection is relatively low, i.e., GMEC = 81.3 ova/g of stool. This may explain some of the difficulties we had in demonstrating

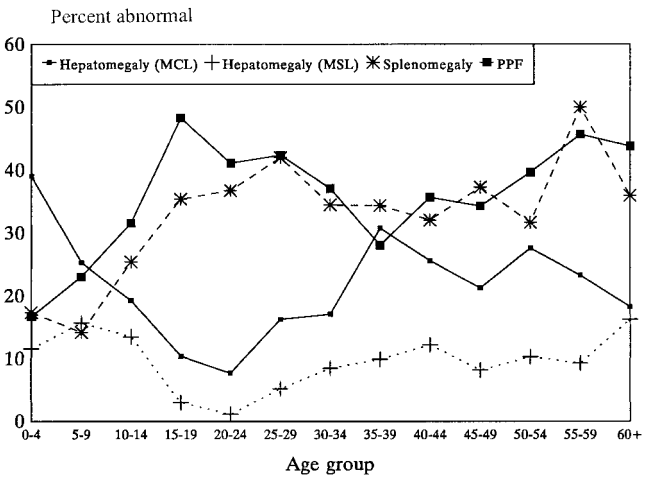

FIGURE 3. Age-adjusted prevalence of ultrasound-detected hepatomegaly, splenomegaly, and periportal fibrosis (PPF) in 1,581 randomly selected subjects in Menofia Governorate. MCL = midclavicular line; MSL = midsternal line.

a relationship between infection and the classical morbid findings and could be a result of repeated courses of chemotherapy in the Egyptian Ministry of Health schistosomiasis control program.[11] Passive case detection and treatment with praziquantel is performed when symptomatic patients seek medical care from rural health centers or other medical facilities. Active case detection is performed in the schools when children have scheduled parasitologic examinations. School children with *Schistosoma* ova in their stool or urine are treated with praziquantel. Detection and treatment is followed by repeated exposures and reinfection in large segments of the rural population. This suppresses the egg count, and thus morbidity, more than the prevalence of infection. Therefore, the association between morbidity and infection is not as clear-cut as it was when chemotherapy was less readily available. Also, due to the cycle of infection and treatment not being synchronized, infection and morbidity clear and reoccur in different time frames.

Previous infection with schistosomiasis or treatment for schistosomiasis were also risk factors for current infection. The greater than 2-fold increase in risk associated with these variables suggests that the same individuals having infections in the past continue to have exposures to infection. Prior infection will be a risk for future infection as long as transmission remains high and exposures to infectious canal water continue. Other clinical findings associated with *S. mansoni* infection were a history of blood in the stools, hepatomegaly in the MCL only in adults, and splenomegaly. In contrast to the lack of a relationship between *S. mansoni* infection and abdominal pain, fecal blood has been a useful diagnostic sign for intestinal schistosomiasis.[12,13] Abdominal ultrasonography did not associate hepatomegaly of the left lobe of the liver with *S. mansoni* infection and did not confirm the association between infection and enlargement of the right lobe of the liver noted in the physical examination of adults. In fact, *S. mansoni* infection reduced the risk of hepatomegaly in the study population by almost 50%. However, there was a 2-fold increased risk of *S. mansoni* infec-

tions in subjects with ultrasonography-detected splenomegaly. Clinical and pathologic studies have reported portal hypertension caused by schistosomal PPF leads to splenomegaly.[14,15] However, schistosomal PPF could not have been a major cause of splenomegaly in our subjects since there was a very weak correlation between *S. mansoni* infection and PPF, and only 21 of our subjects had the higher grades of PPF.

Risk factors for, and associations with, PPF were similar, but often less, than for *S. mansoni* infection. This further suggests that other factors that were confounding the physical examination—and ultrasonography-detected hepatosplenic morbidity in our study population. The frequent use of praziquantel therapy to treat schistosomiasis in Menofia, which impacts upon morbidity greater than prevalence, has already been discussed. Other factors influencing our assessment of hepatic morbidity are the complications of viral hepatitis, chronic viral hepatitis, and cirrhosis, which can cause changes in liver size (enlargement or shrinkage), splenomegaly or PPF;[16] 5–8% of the adults living in rural communities in Menofia are chronic hepatitis B surface antigen carriers and this population also has one of the highest (40–50%) prevalence of chronic infection with hepatitis C virus (HCV) in the world.[17,18] For example, 171 patients seeking medical care at the Menofia Liver Institute and their age- and gender-matched neighborhood controls had anti-HCV rates of 59% and 47%, respectively (El-Hefni H, Raouff AA, Abdel Hamid M and others, unpublished data). Unfortunately, we did not draw blood from our subjects and cannot test their sera for markers of infection with viral hepatitis. However, there is no reason to suspect that this random sample of rural inhabitants of Menofia would differ from other similar populations in the governorate that have been tested for HCV and HBV.[17–19]

The results reported herein differ somewhat from previous cross-sectional studies of morbidity in the *S. mansoni*-endemic areas of the Delta.[13,20] The subjects in these earlier studies were children with higher *S. mansoni* infection rates and less manifestations of chronic viral hepatitis than adults. These factors would lead to considerably less confounding than results from studying adults and would make it much easier to correlate schistosomal infection with morbidity.

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