Q J Med 2001; **94**:357–363

Endemic strongyloidiasis on the Spanish Mediterranean coast

P. ROMÁN SÁNCHEZ[1], A. PASTOR GUZMAN[2], S. MORENO GUILLEN[3], R. IGUAL ADELL[4], A. MARTIN ESTRUCH[4], I. NAVARRO GONZALO[5] and C. RICART OLMOS[2]

From the [1]Department of Internal Medicine, Requena General Hospital, Valencia, [2]Department of Internal Medicine, [4]Microbiology Laboratory, and [5]Haematology Laboratory, Francesc de Borja Hospital, Valencia, and [3]Department of Infectious Diseases, Hospital Ramón y Cajal, Madrid, Spain

Received 10 January 2001 and in revised form 23 April 2001

Summary

Diagnosis and treatment of *Strongyloides stercoralis* infection can be difficult, and a high degree of clinical suspicion in patients who have visited an endemic area is required. We describe the epidemiology and clinical features of 152 prospectively identified cases of strongyloidiasis in an European region, and identify risk factors for the development of severe forms of the disease. This was a prospective study of all patients admitted to a single institution over an 8-year period. Patients (*n* = 152) were mainly elderly male farmers (79%) who had acquired the disease by working barefoot in contact with soil and ingesting non-drinking water. Eosinophilia was a sensitive marker for the infection (82%). Twenty patients (13%) developed severe forms of the illness and six patients (4%) died. A significant association was found between severe forms of strongyloidiasis and steroid usage (OR 9.0, 95%CI 2.1–37.6), immunodebilitating illness (OR 10.1, 95%CI 3.2–32.3) and other immunosuppressive therapy (OR 13.7, 95%CI 2.9–58.7), but by logistic regression analysis, only immunodebilitating disease was as a risk factor (OR 2.1, 95%CI 1.78–2.43). *S. stercolaris* infection is endemic in the Spanish Mediterranean coast. The frequent development of severe forms of the disease, with a high mortality, makes early recognition and treatment essential.

Introduction

Strongyloides stercoralis is a nematode with a unique, complex lifecycle that causes an auto-infection in the host. The infection can persist for decades, even after departure from endemic areas, and under certain conditions, *S. stercoralis* can trigger a life-threatening form of strongyloidiasis, (the so-called hyperinfection syndrome) and disseminated strongyloidiasis.[1] Although usually associated with immunosuppression or debilitating diseases,[2] risk factors for the severe forms of the disease have not been thoroughly evaluated. The mortality rate is very high in these syndromes, despite early and correct treatment.[3]

S. stercoralis is one of the most difficult parasites to diagnose. Definitive diagnosis requires direct visualization of the parasite, but because no test is 100% sensitive there is no absolute reference standard.[4,5] Treatment is also difficult. Only total eradication eliminates the threat of potentially serious disease, and relapses are frequent with standard drugs (thiabendazole, ivermectin).[6] Consequently, it is critical for physicians to be

Address correspondence to Dr P. Román Sánchez, Servicio de M. Interna, Hospital General de Requena, C/ Paraje Casablanca s/n, Requena, 46340 Valencia, Spain. e-mail: gilroman@terra.es

aware of this type of infectious process and the groups of people who may be at risk, to initiate adequate therapy before the development of severe manifestations of the disease. Knowledge of the prevalence of strongyloidiasis and its geographical distribution is thus of practical importance.[7]

S. stercoralis infects a large proportion of tropical and temperate populations. The endemic areas are South America, Southeast Asia, sub-Saharan Africa and the Appalachian region of the USA.[8,9] In Europe, clinical reports on sporadic cases or series of patients who acquired the parasite locally have been published in several countries, including the UK,[10,11] France,[12] Switzerland,[13] Italy,[14,15] Yugoslavia,[16] Poland,[17] Hungaria,[18] Romania,[19] Belgium,[20] and Spain.[21–23]

With a few exceptions, previously reported series of patients with *S. stercoralis* were retrospective or included relatively few patients. In this study, we describe the epidemiological and clinical features of a prospective series of 152 patients diagnosed as having autochthonous strongyloidiasis in our hospital, as well as the risk factors for development of the severe forms of the disease, and discuss the available evidence that supports the endemicity of the parasite in this area.

Methods

All patients diagnosed as having strongyloidiasis at the Francesc de Borja Hospital between January 1990 and December 1997 were evaluated prospectively. Ours is an acute care general hospital with 300 beds, located in Gandia, on the Mediterranean coast of the Valencian Community in Spain. At present, approximately 18% of the population living in the area are farm workers, and most of them have worked barefooted in contact with soil and ingested non-drinking water.

Patients were identified through the Laboratory of Parasitology. Every patient diagnosed with *S. stercolaris* infection was seen by one of the authors and was then prospectively followed. The following variables were collected within 48 h of notification of any positive sample for *S. stercoralis*: sex, age, occupation, workplace behaviour (barefoot working, ingestion of potentially contaminated water, use of lavatory), number of years in contact with the soil, travel history, underlying diseases, reason for admission, antecedents, prior treatment, clinical symptoms, reason to suspect *S. stercoralis*, method of diagnosis, number of samples studied. We also evaluated the dose, duration and side-effects of treatment, major diagnosis at discharge from the hospital, and the outcome of patients. Blood counts and biochemical determinations were also recorded.

Survivors were contacted 1 and 3 months after the date of the treatment of infection. Clinical symptoms were re-evaluated, three stool samples were examined and the eosinophil count was measured.

The diagnosis was made by direct microscopic visualization of the rabditiforms larvae in the stool samples (before 1994) or with the faecal Agar plate culture[24] (after 1994). Microscopic visualization of sputum and microscopic visualization of the tissues in necropsy samples were used in some patients.

We have distinguished between the hyperinfection syndrome and disseminated strongyloidiasis. The hyperinfection syndrome was diagnosed when the patient presented with severe symptoms in organs usually involved in the parasite life cycle, the intestinal tract and the lung, together with a high load of larvae in feces or sputum, whether accompanied or not by severe infections caused by Gram-negative bacteria. Disseminated strongyloidiasis was diagnosed when the widespread involvement of organs that are not ordinarily a part of their life cycle of larvae was demonstrated.[25,26]

Study data were analysed using the χ^2 test with Yates'correction for categorical variables and Student's t test for comparison of the means. The association between various possible risk factors for the development of the severe forms of the illness was calculated as an estimated odds ratio. The statistical significance of associations was calculated using the χ^2 statistic. Multivariate analysis used logistic regression analysis.

Results

Epidemiology

During the study period, 152 of 16 607 patients admitted to the hospital were diagnosed as having strongyloidiasis (0.9%). Most of the infected people were elderly men, with a mean age of 67 years. All the men except one, were farmers or ex-farmers who had worked barefooted (Table 1). The youngest patient, a 12-year-old student, habitually played on farm land. Of the women, 15 had been farm workers; in the remaining 17 women, being farmer's spouses ($n = 9$) or farmer's widows ($n = 8$) was the only recognizable risk factor for strongyloidiasis. Their husbands were examined in seven cases: four had larvae isolated from faeces, and two had eosinophilia and symptoms suggestive of *S. stercoralis* infection.

Table 1 Demographic and epidemiological features of 152 patients with autochthonous strongyloidiasis on the Spanish Mediterranean coast

	Chronic infection ($n = 132$)	Severe forms of illness ($n = 20$)	p
Age (mean ± SD)	66.03 ± 10.74	68.80 ± 9.4	NS
Sex (number of patients)			
Men	101	19	NS
Women	31	1	0.05
Profession (number of patients)			
Farmer	40 men; 3 women	4 men	NS
Ex-farmer	60 men; 11 women	15 men; 1 woman	NS
Housewife	17 women	0	NS
Years in contact with the soil (mean ± SD)	22.41 ± 15.97	26.15 ± 13.69	NS
Probable penetration pathway of the worm			
Skin (working shoeless)	83	16	NS
Mucosa (ingesting non-drinking water)	12	1	NS
Unrecognized	37	3	NS

NS, no significant difference found between groups.

The average time spent in farming was 22.9 ± 15.7 years (range 7–60). Time since the ex-farmers had given up farm work was very variable (mean 14.8 ± 14.6). Only three patients usually defaecated in fields. None of the patients had travelled to other areas known to be endemic for *S. stercoralis*. All had always lived and worked on the Mediterranean coast.

Clinical manifestations

Patients were admitted to the hospital for different reasons. Table 2 shows the major diagnosis of the patients during the admission in which chronic strongyloidiasis was diagnosed. Clinical symptoms that were attributable to strongyloidiasis were non-specific (Table 3). Patients were asymptomatic ($n = 63$) or had minimal complaints ($n = 69$), intermittently over years, that did not give rise to any examination (68% of the individuals had not consulted a physician, while the remaining 32% had received antacids as treatment). In fact, the main reason for suspecting *S. stercolaris* infection was eosinophilia in otherwise asymptomatic persons (77%), followed by gastrointestinal (11%) and cutaneous symptoms (4%). The remaining patients complained of respiratory symptoms (1%) or had a mixture of all these manifestations (7%).

A severe form of the disease was diagnosed in 20 (13%) patients: hyperinfection syndrome in 17 (11%) and disseminated strongyloidiasis in three (2%). An immunosuppressive illness, usually a solid or haematological neoplasia, was present as underlying disease. The presence of this risk

Table 2 Major diagnosis at discharge from hospital in 152 autochthonous strongyloidiasis patients

Major diagnosis	Number of patients	Frequency (%)
Chronic obstructive lung disease	44	28.9
Heart diseases	38	25
S. stercoralis hyperinfection syndrome	17	11.1
Gram-negative bacterial complication	13	
Bacteraemia	7	
Pneumonia	3	
Meningitis	2	
Liver abcess	1	
Cerebrovascular disease	12	7.8
Gastrointestinal bleeding	9	5.9
Solid malignancy	7	4.6
Lymphoreticular malignancies	7	4.6
Pneumonia	5	3.2
Disseminated strongyloidiasis	3	1.9
Liver disease	3	1.9
Anaemia	2	1.3
Pancreas disease	2	1.3
Chronic renal failure	1	0.6
Septic shock of urinary origin	1	0.6
Whipple's disease	1	0.6
Amyloidosis	1	0.6
HIV infection	1	0.6

Table 3 Symptoms in 152 autochthonous strongyloidiasis patients

Gastrointestinal	*n*	Pulmonary	*n*	Cutaneous	*n*	Several***	*n*
None	73 (48%)	None	122 (80.3%)	None	91 (59.8%)	GI+C	25 (16.4%)
Diarrhoea	21 (13.8%)	Cough	9 (5.9%)	Pruritis	23 (15.3%)	GI+P	2 (1.3%)
Abdominal pain	14 (9.2%)	Wheezing	4 (2.6%)	Urticaria	10 (6.6%)	P+C	0 (0%)
Pruritis ani	6 (3.9%)	Haemoptysis	1 (0.7%)	Larva currens	3 (2%)	GI+P+C	0 (0%)
Nausea/vomiting	5 (3.2%)	Several*	2 (1.3%)	Several*	2 (1.8%)		
Several*	23 (15.1%)	Non-specific**	14 (9.2%)	Non specific**	23 (15.1%)		
Non-specific**	10 (6.5%)						

*Patient exhibited several symptoms included in the previous lines. **Patient exhibited symptoms which could be ascribed to *Strongyloides* infection or to their associated illness. ***Patient exhibited symptoms of several organs: GI, gastrointestinal; C, cutaneous; P, pulmonary.

factor, as well as the use of steroids or other immunosuppressive drugs (metotrexate, cyclophosphamide, adryamicin), was significantly higher among patients with severe forms of the illness than among those who suffered chronic infection. Other factors, including alcoholism, gastrectomy and antacid treatment, were similar in the two groups (Table 4). By multivariate analysis, only the presence of immunodebilitating diseases was associated with the development of a severe form of the disease (OR 2.1, 95%CI 1.78–2.43, $p<0.05$) (Table 5).

Thirteen of the 20 (65%) patients developed a Gram-negative bacterial complication (bacteraemia 7, pneumonia 3, meningitis 2, liver abscess 1). A total of six patients died, including the three patients with disseminated disease and three with the hyperinfection syndrome (in all cases secondary to bacterial infections). The autopsy showed invasion of the parasite into multiple organs in patients with disseminated disease.

Laboratory diagnosis

Up to 82% of the cases had >500 eosinophils/mm.^{3} Eosinophilia was significantly greater in patients with chronic infection (1357 ± 811 eosinophils/mm^3) than in those with severe forms (694 ± 309 eosinophils/mm^3) ($p<0.001$). There was no evidence of any other significant alteration in laboratory tests that could be attributed to the parasite.

Stools were the diagnostic specimen in nearly all of the patients. A mean of 2.3 stool samples per patient were necessary for the diagnosis, but 21 (14%) patients needed four or more samples. Two patients were diagnosed by examination of the sputum and in three patients the diagnosis was made only at autopsy.

Treatment

Patients with chronic infection received treatment with thiabendazole (15 mg/kg twice daily for 5 days). Four patients reported mild gastric problems that did not require discontinuation of the treatment. Response to therapy was good. Symptoms disappeared in all complaining patients, eosinophilia persisted in only three patients, and examination of the stools performed at 1 and 3 months post-treatment in 115 patients showed persistence of the parasite in only one patient. A new course of treatment was administered to this patient, and the three patients with persistent eosinophilia, with resolution of abnormal findings in all cases.

Patients with hyperinfection or dissemination were treated with two courses of 25 mg/kg of thiabendazole, twice daily for 7 days, 2 weeks apart, in addition to antimicrobial therapy and supportative care measures. Response was poor, with five (25%) patients dying despite therapy; one of the deaths occured the same day that the diagnosis was made but before the antihelminthic treatment was given.

As in patients with chronic infection, patients with severe disease who survived had negative stool examination.

Discussion

Infection with *S. stercolaris* is frequent in our area among patients hospitalized for any reason (0.9%). Most patients have been asymptomatic for years, but morbidity and mortality is still significant. Development of severe forms of the disease in 13% of infected patients, with a high mortality rate (30%), underscores the importance of recognizing and treating the disease in the early, asymptomatic period.

Table 4 Characteristics of 152 strongyloidiasis patients according to clinical status.

Clinical status	*n*	Deaths	Eosinophils/mm^3 (mean (SD))	Immunodebilitating state or contributing factor*					
				Corticosteroids	Immunosuppression	Immunodebilitating illness	Alcoholism	Gastrectomy	Antacids
Chronic infection									
Asymptomatic	63	0	1393 (821)	1	0	7	4	3	1
Symptomatic	69	0	1095 (857)	5	4	10	9	1	19
Hyperinfection syndrome									
Uncomplicated	4	0	379 (224)	3	1	2			3
Sepsis	7	2	328 (120)		3	5	1		1
Pneumonia	3	0	372 (165)	2		1			1
Meningitis	2	1		1		1	1	1	1
Hepatic abscess	1	0	320				1	1	
Diss. strong.	3	3	410 (198)		2	3			2

Deaths, deaths attributed to *Strongyloides stercoralis*. Diss. strong., disseminated strongyloidiasis. *Patients suffering from a debilitating state (immunosuppressive illness or therapy) or having certain conditions which might increase the worm burden (alcoholism, gastrectomy, antacid medication).

The real prevalence of strongyloidiasis in our area is probably underestimated. We have diagnosed only patients with eosinophilia or clinical symptoms suggestive of strongyloidiasis, but most patients with the infection are completely asymptomatic, and up to 20% may not have eosinophilia. In addition, the diagnosis is difficult, and false negative results are frequent due to a scarcity or absence of worms at the moment of the examination.[27] Furthermore, we have identified being a farm worker as the most significant risk factor for acquiring strongyloidiasis, so the prevalence in this group must be significantly higher.

The worldwide prevalence of *S. stercoralis* is unknown, but it is estimated that 30 to 60 million people are infected.[1,7] Prevalence rates that define endemicity for the parasite have not been established, due to the lack of epidemiological studies in the general population. The studies published to date are heterogeneous and incomplete, and are limited to identifying the prevalence of *S. stercoralis* in a series of stools taken in parasitology laboratories or in a clinical series of patients in a hospital, in institutions, in schools or war veterans. The prevalences observed in these situations have ranged from 0.6% to 40% and the whole range is considered endemic.[8] We therefore believe that the Mediterranean region where this study has been performed is very likely an endemic area for *S. stercoralis*, with especially high prevalence rates in some groups of the population (farm workers).

Infection in women in this study is an intriguing issue. Although many of them were or had been farm workers, more than half did not have this risk factor. Other potential mechanisms of transmission, such as direct oral-faecal or by skin-faeces contact due to unhygienic behaviour,[28,29] can be reasonably ruled out. Although sexual transmission has been advocated by some authors, it must rarely, if ever, occur, and cannot account for the transmission in all these cases.

The ‘larva currens’, a serpiginous creeping urticarial eruption provoked by the passage of the larva on the perianal skin at a speed of 5–10 cm/hour, pathognomonic of SS, was only seen in three patients (1.97%). This figure is similar to that of the milder series of hospitalized patients in Kentucky[30] and far lower than those of the prisoners in Indochina in World War II,[31] where it was found in up to 92% of cases, and therefore constituted a differential characteristic of the parasite in this part of Asia.

Eosinophilia was present in nearly 80% of the cases which is similar to the rates reported.[1,7,8] However, we believe that this rate was overestimated, as it was the main reason for suspecting *S. stercoralis* and asymptomatic patients without

Table 5 Risk factors for the development of severe forms of strongyloidiasis

	Chronic infection ($n = 132$)	Severe forms ($n = 20$)	OR (95%CI)	p
Immunodebilitating illness*	17 (13%)**	12 (60%)***	10.1 (3.2–32.3)	<0.001
Corticosteroid therapy	6 (5%)	6 (30%)	9 (2.1–37.6)	<0.001
Immunosuppressive therapy	4 (3%)	6 (30%)	13.7 (2.9–58.7)	<0.001

*Single risk factor identified in multivariate analysis (OR 2.1, 95%CI 1.7–2.4, $p<0.05$). **Solid or haematological malignancies, chronic renal failure, HIV infection, liver disease, malabsortion. ***Solid or haematological malignancies.

this alteration may have gone unnoticed. At the same time, misdiagnosis of hyperinfection or dissemination in patients who suffered bacterial complications without evident symptoms of strongyloidiasis seems likely, since eosinophilia is not usually present in these situations.[32]

There are no data in the literature about the rate of severe forms of illness among infected people. In our series, it is up to 13%, a far from negligible figure, especially if we consider that 30% of them died. It is accepted that patients with debilitating diseases or treatments are at higher risk of developing the severe forms of illness, since a vast majority of reported patients had one or more of these underlying conditions; these have been identified as predisposing to infection as well.[33] We have confirmed that immunodebilitating disease is the main risk factor for severity of the disease. Gastrectomy, and drugs that produce achlorhydria, considered by other authors as risk factors for the multiplication of the parasite,[34] were not identified in our study. It is possible that the large percentage of patients taking antacids in published clinical series of strongyloidiasis received the drug for treatment of symptoms caused by the parasite itself. Many of the patients in this study were prescribed antacids for treatment of symptoms that were later shown to be caused by the infection.

The response to treatment is strikingly good in the present series, not only in terms of efficacy but also in the lack of adverse events. This contrasts with the data previously published, with approximately 30% of the patients developing adverse effects and a high rate of therapeutic failure.[7] The excellent tolerance and efficacy among our patients may be due to the use of lower doses than usual (15 mg/kg instead of 25–50 mg/kg) for a longer period (5 days instead of 2 or 3).

In conclusion, *S. stercolaris* infection seems to be endemic in this region of the Mediterranean coast, although point-prevalence surveys in the general population must be carried out in order to confirm this assumption. This finding is of practical importance both for the residents in the area and, since it is a coastal zone popular with tourists, for people travelling there. Specially, people with underlying diseases known to predispose to severe forms of strongyloidiasis must be aware of the risk of acquiring the disease. Screening of the population at highest risk (farm workers in this study) using adequate markers, such as eosinophilia, should be evaluated in order to provide therapy that can prevent dissemination and mortality from the disease, particularly the patients who might receive corticosteroid or other types of immunosuppressive therapies.

References

1. Adel AF, Mahmoud. Strongyloidiasis. *Clin Infect Dis* 1996; **23**:949–53.
2. Heyworth MF. Parasitic diseases in inmunocompromised hosts: cryptosporidiasis, isosporidiasis and strongyloidiasis. *Gastroenterol Clin North Am* 1996; **25**:691–707.
3. Simpson WG, Gerhardstein DC,Thompson JR. Disseminated Strongyloides stercoralis infection. *South Med J* 1993; **86**:821–5.
4. Van der Feltz M, Slee PH, van Hees PA, Tersme M. Strongyloides stercoralis infection: how to diagnose best? *Neth J Med* 1999; **55**:128–31.
5. Kaminsky RG. Evaluation of three methods for laboratory diagnosis of Strongyloides stercoralis infection. *J Parasitol* 1993; **79**:277–80.
6. Gann PH, Neva FA, Gam AA. A randomized trial of single and two dose ivermectin versus thiabendazol for treatment of strongyloidiasis. *J Infect Dis* 1994; **169**:1076–9.
7. Liu L, Weller P. Strongyloidiasis and other intestinal nematode infections. *Infect Dis Clin N Am* 1993; **3**:655–82.
8. Genta R. Global prevalence of strongyloidiasis: critical review with epidemiologic insights into the prevention of disseminated disease. *Rev Infect Dis* 1989; **5**:755–67.
9. Sorozcan W. Strongyloides stercoralis intestinal roundworm. II. Geographic distribution. *Wiad Parazytol* 1994; **40**:115–26.
10. Harcourt Webster JN, Scaravilli F, Darwish AH. Strongyloides stercoralis infection in an HIV positive patient. *J Clin Pathol* 1991; **44**:346–8.
11. Sprott V, Selby CD, Isaphani P, Toghill PJ. Indigenous strongyloides in Nottingham. *Br Med J* 1987; **294**:741–2.
12. Doury P. Autochthonous angulluliasis in France. *Bull Soc Pathol Exot* 1993; **86**:116.

13. Berthoud F, Berthoud S. A propos de 18 cas d'anguillulose diagnostiqués à Genève. *Schweiz Med Wochenschr* 1975; **105**:110–15.
14. Genta RM, Gatti S, Linke MJ, Cevini C, Scaglia M. Endemic strongyloidiasis in northern Italy: clinical and inmunologic aspects. *Q J Med* 1988; **68**:679–90.
15. Ronchetto F, Pistono PG, Guasco C. Strongyloides stercoralis in a region of northwestern Italy: an epidemiological note and presentation of a case of eosinophilic infiltration of the lung. *Recenti Prog Med* 1995; **86**:234–7.
16. Suvajdzic N, Kranjcic Zec I, Jovanovic V, Popovic D, Colovic M. Fatal strongyloidiasis following cortisco-steroid therapy in a patient with chronic idiopathic thrombocytopenia. *Haematologia (Budap)* 1999; **29**:323–6.
17. Sadowska H, Konieczny B. A case of Strongyloides hyper-invasion in the course of bronchial asthma. *Pol Tyg Lek* 1990; **45**:29–31, 628–9.
18. Károly Z, Erös N, Kriston R. Cutaneous manifestation of strongyloidiasis. *Orv Hetil* 1999; **140**:191–4.
19. Panaitescu D, Capraru T, Bugarin V. Study of the incidence of intestinal and systemic parasitoses in a group of children with handicaps. *Roum Arch Microbiol Immunol* 1995; **54**:1–2, 65–74.
20. De Goede E, Martens M, Van Rooy S, Van Moerkerke I. A case of systemic strongyloidiasis in an excoal miner with idiopathic colitis. *Eur J Gastroenterol Hepatol* 1995; **7**:807–9.
21. Gomez J, Plaza V, Muñoz C, Franquet T. Hiperinfección por Strongyloides stercoralis y otros patógenos en un paciente con enfermedad obstructiva crónica corticodependiente. *Med Clin Barcelona* 1997; **109**:609.
22. Cremades Romero MJ, Igual Adell R, Ricart Olmos C, Estelles Piera F, Pastor Guzman A, Menendez Villanueva R. Infección por Strongyloides stercoralis en la comarca de la Safor (Comunidad Valenciana). *Med Clin Barcelona* 1997; **109**:212–15.
23. Rodriguez-Calabuig D, Oltra-Alcaraz C, Igual-Adell R, Parra-Godoy F, Martinez-Sanchez J, Angel-Rodenas C, *et al*. Treinta casos de estrongyloidiasis en un centro de atención primaria: características y posibles complicaciones. *Aten Primaria* 1998; **21**:271–4.
24. Arakaki T, Iwanaga M, Kinjo F, Atsushi Saito A, Asato R, Ikeshiro T. Efficacy of agar-plate culture in detection of Strongyloides stercoralis infection. *J Parasitol* 1990; **76**:425–8.
25. Longworth DL, Weller PF. Hyperinfection syndrome with strongyloidiasis. In: Remington JS, Swartz MN, eds. *Current Clinical Topics in Infectious Diseases*, Vol. 7. New York, McGraw Hill, 1986.
26. Stone WJ, Schaffner W. Strongyloides infections in transplant recipients. *Semin Respir Infect* 1990; **5**:58–64.
27. Uparunukraw P, Phongsri S, Morakote N. Fluctuation of larval excretion in Strongyloides stercoralis infection. *Am J Trop Med Hyg* 1999; **60**:967–73.
28. Lindo JF, Robinson RD, Terry SI, Vogel P, Gam AA, Neva FA, *et al*. Age-prevalence and household clustering of Strongyloides stercoralis infection in Jamaica. *Parasitology* 1995; **110**:97–102.
29. Mitchell I, Burken. Strongyloides stercoralis infection in a chronically institutionalized patient with schizophrenia and dementia. *Clin Infect Dis* 1995; **21**:1047.
30. Milder JE, Walzer PD, Kilgore G, Rutheford Y, Klein M. Clinical features of Strongyloides stercoralis infection in an endemic area of the United States. *Gastroenterology* 1981; **80**:1481–8.
31. Pelletier L, Baker C, Gam A, Nutman T, Neva F. Diagnosis and evaluation of treatment of chronic strongyloidiasis in ex-prisoners of war. *J Infect Dis* 1988; **157**:573–6.
32. Cook GD. Strongyloides stercoralis hyperinfection syndrome: how often is it missed? *Q J Med* 1987; **64**:625–9.
33. Davidson RA, Fletcher RH, Chapman LE. Risk factors for strongyloidiasis: case-control study. *Arch Intern Med* 1984; **144**:321–4.
34. Gelber M, Rodrig J. Strongyloides stercoralis hyperinfection. *Harefuah* 1996; **130**:90–2, 143.