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# Clinical manifestations of strongyloidiasis in southern Taiwan

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The diagnosis and management of strongyloidiasis present a continuous challenge in developing countries including Taiwan. In this study, the clinical characteristics and microbiological findings of 27 patients with *Strongyloides stercoralis* infection were retrospectively analyzed. Intestinal infection was identified in 17 patients and hyperinfection syndrome or disseminated disease in 10 (including 2 autopsy cases). The most frequent clinical findings were diarrhea (74%), fever (70%), abdominal pain (59%), cough (37%), dyspnea (33%), and constipation (26%). The common initial laboratory abnormalities were leukocytosis (81%), anemia (67%), liver function impairment (52%), and eosinophilia (44%). Most of the 27 patients had comorbid conditions, including malnutrition in 20 (74%), corticosteroid dependence in 15 (55%), chronic obstructive pulmonary disease in 9 (33%), chronic liver disease or cirrhosis in 8 (30%), and peptic ulcer disease in 7 (26%). There was no difference in the time interval from symptom onset to diagnosis between the intestinal infection group and the hyperinfection/disseminated group (22 ± 15 vs 17 ± 9 days). Larvae of *S. stercoralis* were identified in the stool of 24 patients, in the sputum smear of 5, in the gastric biopsy of one, and on histology of autopsy specimens in 2. Twenty-six patients received antiparasitic drug therapy of variable duration (mebendazole in 24, albendazole in 2, combined therapy in one). The overall cure rate was 52% (14/27). Relapse occurred in 4 patients. The overall mortality was 26% (7/27). There was a high mortality (up to 50%) in the hyperinfection/disseminated disease group. In conclusion, diagnosis of strongyloidiasis is often delayed and overlooked because of nonspecific symptoms. Physicians in endemic regions should include strongyloidiasis in the differential diagnosis when patients present with gastrointestinal and/or pulmonary symptoms with peripheral eosinophilia.

**Key words:** Hyperinfection syndrome, strongyloidiasis, *Strongyloides stercoralis*

*Strongyloides stercoralis* is an intestinal nematode that is widely distributed throughout the tropics and subtropics [1]. Human infection begins with the penetration of the skin by filariform larvae, which migrate hematogenously to the lungs. Larvae then ascend the airway, are swallowed, and mature in the gut. Unlike other nematodes, *S. stercoralis* can autoinfect the same host and persist for decades. Clinical manifestations of strongyloidiasis can be divided into the following categories: (1) asymptomatic carriage, (2) intestinal infection, and (3) hyperinfection syndrome with or without dissemination. Fifteen to thirty percent of chronically infected people are asymptomatic [2]. When infected, immunocompromised individuals may develop overwhelming dissemination, which causes fatal sepsis, meningitis, and acute respiratory distress syndrome [3-5]. There have been several case reports of disseminated strongyloidiasis in Taiwan [6,7], but a large case series has not been previously reported. This study assessed the clinical findings in a series of 27 patients with strongyloidiasis.

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## Patients and Methods

The medical and microbiological records in Kaohsiung Veterans General Hospital were reviewed to identify patients with the diagnosis of strongyloidiasis during the period from October 1990 through March 2001. Strongyloidiasis was defined as the presence of *S. stercoralis* in at least one stool sample examined, or in any body fluids or tissues. Eosinophilia was defined as an eosinophil count of >500 /mm$^3$ in the blood.

Data were collected on the demographic characteristics, predisposing factors, the time interval from symptom onset to definite diagnosis, use of corticosteroids, previous surgery, certain clinical characteristics (abdominal pain, abdominal fullness, diarrhea, fever, nausea, vomiting, constipation, pulmonary symptoms, eosinophilia, leukocytosis, anemia, and liver function impairment), diagnostic tools, treatment, and outcome of the patients. The intestinal phase of the disease was defined as the presence of colicky abdominal pain, often epigastric, associated with nausea, vomiting, diarrhea, and body weight loss, and may also include a protein-losing enteropathy with larvae of *S. stercoralis* in the stool. Disseminated infection was defined as (1) fever accompanied by one of the following symptoms: abdominal pain, diarrhea, ileus, pulmonary infiltrates, meningeal symptoms and signs, gram-negative bacteremia, and gram-negative bacterial meningitis; and (2) the presence of filariform larvae of *S. stercoralis* in any body fluid or in the feces (constituting a probable diagnosis). Disseminated disease was diagnosed when documented by biopsy or direct smear [8]. Hyperinfection syndrome was defined as a systemic illness with chronic diarrhea, abdominal pain, loss of weight, cough, edema, hypoproteinemia, and anemia, with involvement of 2 or more organs (usually the lung, intestines, liver, and the central nervous system) and the presence of *Strongyloides* larvae in the stool and sputum [9].

## Statistical analysis

Chi-square test and Fisher exact test were used to analyze the difference in clinical symptoms, laboratory abnormalities, underlying disease, and outcome between the intestinal infection group and the hyperinfection/dissemination group. The *t* test and Mann-Whitney U test were used to assess the time interval from onset of clinical symptoms to diagnosis, as well as the age distribution among the 2 groups. A *p* value of less than 0.05 was considered statistically significant.

Table 1. Clinical and laboratory findings of 27 patients with strongyloidiasis in southern Taiwan treated from October 1990 through March 2001

| | No. of patients with strongyloidiasis (%) | |
|---|---|---|
| | Intestinal infection n = 17 | Hyperinfection syndrome[a] n = 10 |
| Clinical findings | | |
| Dyspnea | 2 (12) | 9 (90) |
| Fever | 10 (59) | 9 (90) |
| Cough | 3 (18) | 7 (70) |
| Diarrhea | 13 (76) | 7 (70) |
| Abdominal pain | 11 (65) | 5 (50) |
| Abdominal distension | 7 (41) | 4 (40) |
| Hematemesis | 2 (12) | 4 (40) |
| Cyanosis | 0 | 3 (30) |
| Nausea/vomiting | 8 (47) | 3 (30) |
| Constipation | 5 (29) | 2 (20) |
| Hemoptysis | 0 | 1 (10) |
| Chest pain | 2 (12) | 1 (10) |
| Hematochezia | 4 (24) | 1 (10) |
| Ecchymosis | 1 (6) | 1 (10) |
| Rash | 3 (17) | 1 (10) |
| Pruritus | 2 (12) | 1 (10) |
| Anorexia | 4 (24) | 1 (10) |
| Change of consciousness | 2 (12) | 1 (10) |
| Jaundice | 3 (18) | 0 |
| Chills | 5 (29) | 0 |
| Laboratory findings | | |
| Leukocytosis (WBC >10 x $10^9$/L) | 13 (76) | 9 (90) |
| Anemia (Hb <12 g/dL) | 13 (76) | 5 (50) |
| Impairment of liver function | 9 (53) | 5 (50) |
| Eosinophilia (>500/$mm^3$) | 9 (53) | 3 (30) |

Abbreviations: WBC = white blood cell; Hb = hemoglobin

[a]Includes 2 patients with autopsy.

## Results

### Demographic and clinical characteristics

A total of 28 strongyloidiasis cases were diagnosed and treated at the Kaohsiung Veterans General Hospital from October 1990 through March 2001. One patient with intestinal infection was excluded because of inadequate clinical information. Seventeen patients presented with intestinal infection only and 10 with hyperinfection/disseminated infection. Most (22/27) of these patients were men, with a male/female ratio of 22:5 and a median age of 70 years (range, 6-86 years).

### Clinical and laboratory manifestations

Diarrhea was the initial presentation in 20 (74%) patients, fever in 19 (70%), abdominal pain in 16 (59%), nausea/vomiting in 11 (41%), cough in 10 (37%), dyspnea in 9 (33%), and constipation in 7 (26%), as shown in Table 1. The most common laboratory abnormalities were leukocytosis (81%), anemia (67%), impairment of liver function test (52%), and eosinophilia (44%). For patients in the hyperinfection/disseminated group, presentation with dyspnea, cough, and cyanosis was more frequently seen (Pearson chi-square test, $p<0.05$). In the intestinal infection group, 13 patients had normal chest radiographs and 4 had bilateral chronic interstitial infiltrates. In the hyperinfection/disseminated infection group, multiple alveolar patches were present in 6 patients, right upper lobe lobar consolidation in one, diffuse bilateral interstitial infiltrates in 7, and pleural effusion in one.

**Table 2.** Underlying disease, diagnosis, treatment, and outcome among 27 patients with strongyloidiasis in southern Taiwan treated from October 1990 through March 2001

| | No. of patients with strongyloidiasis (%) | |
|---|---|---|
| | Intestinal infection | Hyperinfection syndrome |
| | n = 17 | n = 10 |
| Age (year) | | |
| Mean ± SD | 63 ± 21 | 72 ± 5 |
| Median | 70 | 72 |
| Range | 6-86 | 68-83 |
| Predisposing factors | | |
| Malnutrition | 11 (65) | 9 (90) |
| COPD | 2 (12) | 7 (70) |
| Adrenal failure | 9 (53) | 6 (60) |
| Antacid use | 7 (41) | 6 (60) |
| Tuberculosis | 0 | 4 (40) |
| Peptic ulcer | 5 (29) | 2 (20) |
| Chronic liver disease | 6 (35) | 2 (20) |
| Hypertension | 5 (29) | 2 (20) |
| Diabetes mellitus | 2 (12) | 1 (10) |
| Rheumatoid arthritis | 0 | 1 (10) |
| Gouty arthritis | 4 (24) | 0 |
| Hepatoma | 2 (12) | 0 |
| Gastrectomy | 2 (12) | 0 |
| Thalassemia | 1 (6) | 0 |
| Multiple myeloma | 1 (6) | 0 |
| Diagnosis | | |
| Stool | 17 (100) | 7 (70) |
| Sputum smear | 0 | 5 (50) |
| Autopsy | 0 | 2 (20) |
| Gastric biopsy | 1 (6) | 0 |
| Treatment | | |
| Mebendazole | 16 (94) | 8 (80) |
| Albendazole | 2 (12) | 0 |
| Outcome | | |
| Cure | 11 (65) | 3 (30) |
| Relapse | 2 (12) | 2 (20) |
| Mortality | 0 | 5 (50) |

Abbreviation: COPD = chronic obstructive pulmonary disease

## Predisposing factors

Most patients had comorbidity. Malnutrition (defined as albumin <3 g/dL) was found in 20 (74%) cases, corticosteroid dependency (adrenal insufficiency) in 56% (15/27), chronic obstructive pulmonary disease (COPD) in 33% (9/27), chronic liver disease in 30% (8/27), and peptic ulcer disease in 26% (7/27). Tuberculosis and COPD were present more frequently in patients with hyperinfection/disseminated disease ($p$ <0.05). Three of the patients with hyperinfection/disseminated disease had old pulmonary tuberculosis and one had active tuberculosis.

## Time interval from symptom onset to diagnosis

The time interval from symptom onset to diagnosis was estimable in 23 patients, with a mean of 20 ± 13 days. The time intervals were not significantly different between the intestinal infection group and the hyperinfection/disseminated disease group (mean, 22 ± 15 vs 17 ± 9 days).

## Diagnosis and treatment

Larvae of *S. stercoralis* were found in the stool of 24 (89%) patients, sputum smear of 5, and gastric biopsy of one. Autopsy was performed in 2 patients. Diagnosis was made postmortem in one patient.

In the intestinal infection group, 16 patients received treatment with mebendazole of variable duration and 2 received albendazole. One patient relapsed under mebendazole treatment and was treated with albendazole successfully. In the hyperinfection/disseminated group, 9 patients received a median 10 days of treatment with mebendazole (range, 6-35 days).

## Case demonstrations

### *Case 1*

A 70-year-old man presented with exacerbated shortness of breath and cough for 10 days. He had been a heavy smoker for decades. A diagnosis of COPD was made 2 years before this visit based on a pulmonary function test (forced expiratory volume in 1 second 0.95 L/min, forced vital capacity 2.4 L/min, severely obstructive ventilatory impairment), for which he received intermittent corticosteroid treatment. On examination, he had a cushingoid appearance and was in severe respiratory distress. Lung auscultation revealed bilateral rhonchi and rales. Laboratory tests showed a white cell count of 13 x $10^9$/L with 89% neutrophils, 6% lymphocytes, 4% monocytes, and 1% eosinophils; hemoglobin 13.6 g/dL, total bilirubin 1.8 mg/dL, and potassium 3.1 mmol/L. Chest radiography disclosed interstitial and alveolar infiltrates over bilateral lung fields. He was intubated soon after admission, and was given high-dose corticosteroid, bronchodilators, and antibiotics due to COPD with suspicion of secondary infection. On Day 12 after admission, larvae of *S. stercoralis* were detected on sputum smear. Mebendazole at a dose of 100 mg twice daily was administered. He died from septic shock 19 days after admission. Autopsy showed disseminated infection of strongyloidiasis in the lung, heart, small and large intestines, mesentery, kidney, thyroid, and subcarinal lymph nodes with bronchopneumonia.

### *Case 2*

A 73-year-old man was admitted because of dyspnea for 7 days. He had hypertension and atrial fibrillation for 3 years, but denied having a history of COPD, abdominal surgery, use of corticosteroid, or immunosuppressive drugs. On admission, he was weak and afebrile. Pulmonary rales over bilateral lung fields were heard on auscultation. The abdomen was soft but epigastric tenderness was elicited. Laboratory tests showed a white cell count of 11.7 x $10^9$/L without eosinophilia, and hemoglobin was 10 g/dL. Impairment of renal and liver function was detected, with a blood urea nitrogen of 94 mg/dL (normal range, 7-20 mg/dL), serum creatinine of 3.8 mg/dL (normal range, 0.5-1.5 mg/dL), aspartate aminotransferase 53 U/L (normal range, 5-35 U/L), alanine aminotransferase 48 U/L (normal range, 0-40 U/L), and a total bilirubin of 2.7 mg/dL (normal range, 0.2-1.6 mg/dL). Chest radiography disclosed diffuse alveolar and interstitial infiltrates (Fig. 1), and an abdominal computerized tomography showed massive ascites. On Day 3 after admission, he developed respiratory failure requiring endotracheal intubation and mechanical ventilation. Culture of the blood and ascites yielded *Aeromonas hydrophila*, and the sputum culture grew *Escherichia coli*. He died from septic shock 6 days after admission, and parasitic drugs have not been prescribed. The autopsy findings revealed strongyloidiasis involving the gastrointestinal tract (esophagus, stomach, duodenum, jejunum, ileum, appendix, colon, rectum, pancreas, and gall bladder) (Fig. 2), and lung, with bronchopneumonia.

### *Case 3*

A 6-year-old boy was admitted because of watery diarrhea and abdominal pain for 2 weeks. Laboratory findings included mild leukocytosis (white cell count 10.8 x $10^9$/L) with marked eosinophilia (34%) and anemia (hemoglobin 11.5 g/dL). Alpha-thalassemia was

later diagnosed. Routine stool tests disclosed many white cells. Direct examination by microscopy revealed larvae of *S. stercoralis*. He was successfully treated with mebendazole 100 mg twice daily for 6 days.

### Complication and outcome

In the intestinal infection group, one patient had infectious diarrhea caused by *A. hydrophila* and *Aeromonas sobria*. In the hyperinfection/disseminated infection group, 8 patients had pneumonia. Bacterial pathogens were identified in 6 cases, including *E. coli* in one, *Klebsiella pneumoniae* in 2, *Haemophilus influenzae* in 2, and polymicrobial (*Streptococcus oralis*, *Pseudomonas aeruginosa*, and *E. coli*) in one. Two patients had negative sputum cultures, but a Gram's stain of the sputum showed gram-negative bacilli. Bacteremia was relatively common in the hyperinfection/disseminated infection group; one patient had *A. hydrophilia* bacteremia, one *K. pneumoniae*, one *H. influenzae*, and 2 had polymicrobial bacteremia (*S. oralis*, *P. aeruginosa*, and *E. coli* in one and *E. coli* and *Morganella morganii* in the other). The mortality in the hyperinfection/disseminated disease group was 50%. Two cases in each group relapsed and were treated successfully with another course of antiparasitic drugs. Among the 5 patients in the hyperinfection/disseminated group who survived, a mean duration of 22 days (range, 7-35 days) of treatment with mebendazole was given.

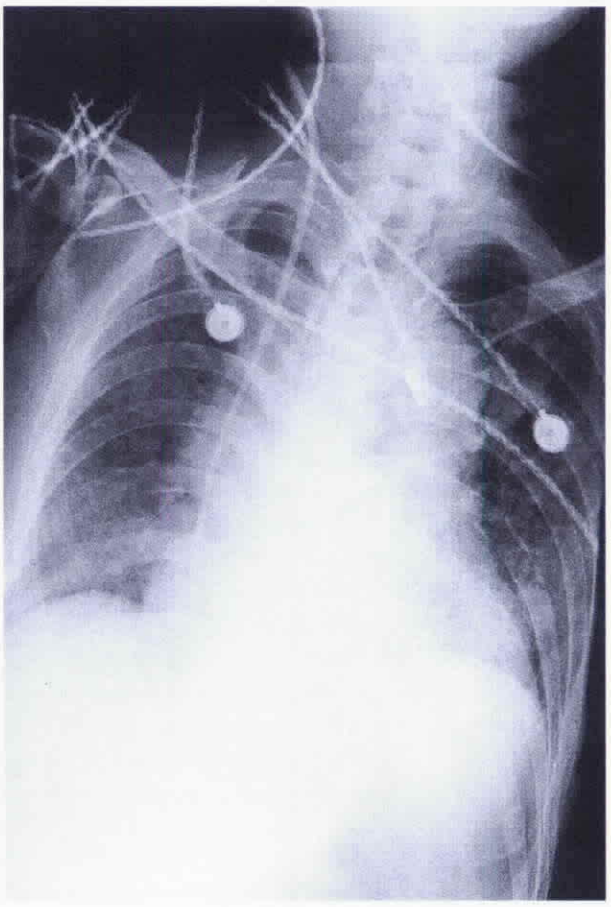

**Fig. 1.** Chest roentgenography revealed bilateral interstitial and alveolar infiltrates on admission.

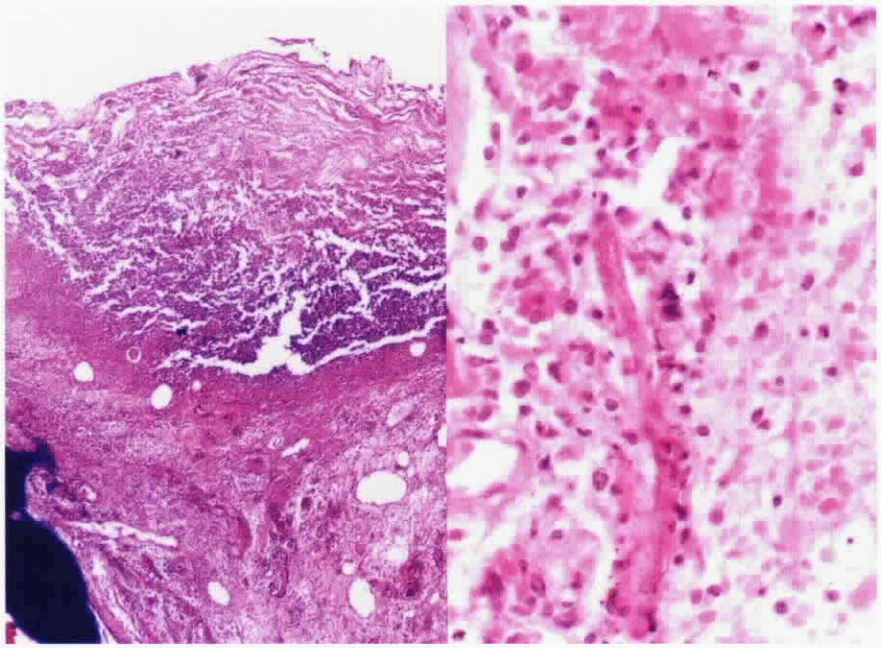

**Fig. 2.** Autopsy disclosed larvae of *S. stercoralis* over the gall bladder (left: 10 x 10, right 10 x 40; hematoxylin and eosin stain).

## Discussion

In 1876, Normand [10] identified a new intestinal worm as the cause of severe diarrhea in French troops repatriated from Vietnam. Although initially named *Anguillula stercoralis*, the parasite was eventually renamed *S. stercoralis* after subsequent investigators elucidated the parasite's unique life cycle [10]. In immunocompetent persons, this parasite usually induces a silent or limited intestinal infection. It is also known to cause a hyperinfection syndrome or disseminated disease in immunocompromised persons. The clinical symptoms and signs of strongyloidiasis reflect the organs involved in the parasite's natural life cycle. Most patients present primarily with nonspecific gastrointestinal complaints and pulmonary symptoms. In Grove's [11] study, the following complaints were significantly more prevalent in infected individuals: indigestion, crampy abdominal pain, diarrhea with malabsorption, and weight loss. Patients with hyperinfection syndrome may manifest cutaneous lesions in the form of petechial and purpuric rashes. Pulmonary involvement results in cough, wheezing, or hemoptysis. Because signs and symptoms may mimic bronchitis or bronchospasm, the diagnosis may be delayed [12].

Laboratory abnormalities are very common in strongyloidiasis. In contrast to previous studies, we found no significant difference in the peripheral eosinophil count between the intestinal infection group and the hyperinfection/disseminated disease group (53% vs 30%, $p$=0.247). This finding was most likely due to the small case number in this series and a similar frequency of corticosteroid dependency between the 2 groups. Igra-Siegman *et al* [13] reviewed 60 cases with hyperinfection syndrome and found that clinical or histological involvement of the lungs was present in 48% of patients. Among the 48 patients who had peripheral blood eosinophil counts, only 17% showed a peripheral eosinophilia ≥8%. Spry [14] found that the mean percentage eosinophil count was 10% in one series of immunocompetent patients with strongyloidiasis, and that the absolute count was $1 \times 10^9$/L in another group.

The chest radiographic manifestations in strongyloidiasis are varied and nonspecific. Pulmonary strongyloidiasis, with documented *S. stercoralis* in the sputum or bronchoscopic specimens, has been associated with a normal chest radiograph, nonsegmental patchy infiltrates, multiple infiltrates, nodular infiltrates, lung abscess, pleural effusion, and small nodules mimicking miliary tuberculosis [2]. In this study, 4 different chest radiographic manifestations were found in patients with hyperinfection/disseminated disease, including pleural effusion, bilateral diffuse interstitial infiltrates, lobar consolidation, and multiple patchy infiltrates.

Hyperinfection or disseminated strongyloidiasis may occur in immunocompromised patients. Those who may be affected include COPD patients on long-term treatment with corticosteroids, oncology patients on chemotherapy, organ transplant recipients receiving immunosuppressive agents, patients with severe burns or malnutrition, and patients with acquired immunodeficiency syndrome. In this series, many patients had predisposing factors, including malnutrition, adrenal insufficiency, antacid or $H_2$-blocker use, COPD, and chronic liver diseases. The incidence of COPD differed significantly between the 2 groups. The combination of COPD in an individual with *Strongyloides* infestation can be disastrous when corticosteroids are added to the treatment regimen [15]. Patients with asthma or COPD and eosinophilia should be screened for *Strongyloides* before the initiation of corticosteroid therapy. If their conditions appear to worsen clinically after the addition of corticosteroids, investigation for strongyloidiasis is indicated. Among the patients in this series who had hyperinfection/disseminated disease, 3 had old pulmonary tuberculosis and one had active tuberculosis. As in the study of Dwarakanath *et al* [16], latent strongyloidiasis can become overt in the presence of tuberculosis, and the diagnosis of strongyloidiasis must be kept in mind for patients who have previously resided in endemic regions.

The pathogenesis of tissue lesions produced by *S. stercoralis* suggests that many clinical manifestations of the infection are immunologically mediated. Thus, it seems that immunosuppression, besides reducing natural defenses against the nematode, may also ameliorate tissue lesions induced by the infection, thereby delaying the clinical manifestations. In this series, the mean time interval from symptom onset to diagnosis was 20 ± 13 days, and no significant difference in the interval was found between the intestinal infection group and hyperinfection/disseminated infection group. Previous study of prisoners of war during the World War II who had acquired *Strongyloides* infections when working on the Burma-Thailand railroad has shown that infections can persist for more than 40 years after acquisition [17]. It is generally known that the detection of *S. stercoralis* larvae in the feces may be very difficult, especially in chronic cases with low-level infection [17,18]. Early diagnosis of the hyperinfection/disseminated disease group in this series was achieved by a high index of

clinical suspicion, frequent examination of sputum smears in patients with pneumonia, and autopsies in patients who died without a definite diagnosis.

The diagnosis and treatment of strongyloidiasis remains a challenge. Accurate estimates of the prevalence of infection with *Strongyloides* are lacking due to the insensitivity of available diagnostic surveys. The numbers of larvae excreted in the stool at any one time are usually few and variable. Examination of serial stool specimens from infected patients will yield a diagnosis in 50% to 80% of cases [12,19,20]. In patients with disseminated disease, examination of the sputum, bronchial washings, or skin biopsy specimens may provide a diagnosis. Unfortunately, the diagnosis of disseminated strongyloidiasis is often made post-mortem, and mortality is high even when the disease is recognized during life.

Strongyloidiasis requires treatment even when patients are asymptomatic. Thiabendazole (25 mg/kg twice per day for 3 days) is the mainstay of treatment. The efficacy of thiabendazole is considerable with rates of parasite eradication in the range of 90% [12]. However, serious adverse reactions occur in as many as 30% of patients, which include nausea, dizziness, drowsiness, visual disturbance, hypotension, and neuropsychiatric symptoms such as disorientation and delirium [21]. For patients with hyperinfection syndrome and disseminated strongyloidiasis, treatment should be continued for 7 to 10 days or longer as needed [22]. Albendazole has replaced thiabendazole because the former has equal efficacy but less toxicity. Two patients in this study were successfully treated with albendazole 400 mg/d orally for 3 days. Liu and Weller [21] reported that albendazole had a cure rate of 81% when given at 400 mg/d for 3 days. Ivermectin appears to be as effective as thiabendazole and albendazole, with fewer side-effects [12,21,23]. Because thiabendazole is not available in Taiwan, patients were mostly treated with mebendazole. The cure rate was 65% (11/17) in the intestinal infection group and 30% in the hyperinfection/disseminated disease group. In a Japanese study [24], 47 patients with strongyloidiasis were treated with 100 mg of mebendazole twice a day for 5 days, and the treatment regimen was repeated at 1, 3, and 4 weeks later. The cure rate was 88.1% after 2 courses, 92.3% after 3 courses, and 100% after 4 courses were completed. In that study, the percentage of patients with hyperinfection syndrome was not reported. Wilson *et al* [25] reported a 55-year-old man with *S. stercoralis* infection who underwent a Roux-en-Y operation, and was successfully treated with mebendazole at a dose of 1.5 g/d for 14 days. The cure rate with mebendazole treatment has probably been overestimated. In a study from Thailand [18], fluctuations of larval excretion in *S. stercoralis* infection were very common, consisting of 52% of infected individuals, probably representing low-level infection. On the other hand, in a study of Far East ex-prisoners of World War II, 2 out of 60 Burma-Thailand veterans with negative stool examinations had a previous diagnosis of strongyloidiasis and had not received treatment [17]. In this study, cure was achieved in patients with hyperinfection/disseminated disease by a prolonged course of antiparasitic drugs and a reduced dose or discontinued use of corticosteroids.

In immunocompromised patients with strongyloidiasis, translocation of enteric bacteria with the worm's migration may cause secondary sepsis, meningitis, peritonitis, and endocarditis, which often occur simultaneously and may even suggest the diagnosis in high-risk patients. In this series, 5 patients had bacteremia, one had peritonitis (*A. hydrophilia*), and 8 had pneumonia. None of the patients in the intestinal infection group had systemic complications. Immunosuppression allows or may even promote intense multiplication of the worms. The parasite can then disseminate to organs not involved in its natural life cycle, such as the lymph nodes, liver, spleen, pancreas, thyroid, brain, and meninges [13].

The mortality of disseminated strongyloidiasis is high. In the study of Igra-Siegman *et al* [13], the overall mortality among 89 immunocompromised patients was 86%. This high mortality is a result of frequent delays in diagnosis. Prognosis depends on the time at which chemotherapy is started. If given early, treatment is effective in immunocompetent patients. In this series, the mortality in the hyperinfection/disseminated disease group was 50%, lower than that reported in the literature. This is likely due to the small case numbers in this series, longer duration of treatment with mebendazole in patients who survived (mean, 22 days in the hyperinfection/disseminated disease group), and early withdrawal or reduction of immunosuppressive agents used.

There are several limitations in this study. First, because this was a retrospective study based on chart review, the history and clinical information of the patients may be incomplete. Second, with the variable sensitivity of stool examination for detecting *Strongyloides* larvae, confirmation of eradication is difficult. In this study, the definition of cure was based on a series of negative stool examination results plus resolution of symptoms, as well as a decreasing peripheral blood eosinophilia, and adjunctive sero-

logical testing for antibody was not performed.

*S. stercoralis* infestation is not common in Taiwan. However, given the poor prognosis of disseminated strongyloidiasis, consideration should be given to screening patients at increased risk of infection, that is, those with gastrointestinal or pulmonary symptoms receiving immunosuppressive therapy, especially before therapy with immunosuppressive drugs. Early diagnosis, appropriate therapy and control of the underlying diseases, and reduced use of corticosteroids will help prevent hyperinfection syndrome or dissemination of the parasite.

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