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<th>Strongyloidiasis in a nonagenarian who previously worked in conservancy services</th>
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<tr>
<td><strong>Citation</strong></td>
<td>Hong Kong Medical Journal, 2013, v. 19 n. 1, p. 74-6</td>
</tr>
<tr>
<td><strong>Issued Date</strong></td>
<td>2013</td>
</tr>
<tr>
<td><strong>URL</strong></td>
<td><a href="http://hdl.handle.net/10722/185996">http://hdl.handle.net/10722/185996</a></td>
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Introduction

*Strongyloides stercoralis* was first discovered in 1876 as the agent causing Cochin China diarrhoea in French troops returning from South-East Asia. It is a soil-transmitted intestinal nematode that is endemic in tropical and subtropical areas including South and South-East Asia, Eastern Europe, Central America, South America, and sub-Saharan Africa. In Hong Kong, the reported incidence in faecal specimens is 0.1 to 0.3%. There have been reports of strongyloidiasis in Hong Kong causing severe disseminated disease, and its association with nephrotic syndrome and how the disease mimics ulcerative colitis. We report strongyloidiasis in an elderly woman born in Hong Kong, who endured chronic diarrhoea and possibly became infected via her prior occupation in conservancy services.

Case report

A 96-year-old woman born in Hong Kong was admitted in June 2011 following a 2-month history of diarrhoea with passage of loose stools up to 5 times per day and associated anorexia. There was no history of vomiting, nausea, abdominal pain, per-rectal bleeding, mucus in stools, flushing, wheezing, or travel to the mainland or elsewhere in the past few years. Nor had she recently received antibiotics, laxatives, or topical/oral steroids. She had a history of Alzheimer’s disease, cholecystectomy, spinal stenosis with multi-level laminectomies, osteoporotic collapse of multiple lumbar vertebrae, osteoarthritis of her knee joints, and single right-sided kidney with chronic renal failure. Prior to this admission, she was able to walk with a quadripod.

On physical examination, her body temperature was 37.8°C. Her blood pressure, pulse rate and oxygen saturation (room air) were 131/65 mm Hg, 109 beats per minute, and 91%, respectively. She was dehydrated and noted to have extensive scratch marks over both thighs. Respiratory examination revealed left lower zone coarse crepitations. Cardiovascular, abdominal, and neurological examinations yielded nil abnormal. Investigations showed leukocytosis of 22 x 10^9/L (neutrophil 21.56 x 10^9/L, lymphocyte 0.44 x 10^9/L, and eosinophil was not present), haemoglobin level of 80 g/L, creatinine level of 360 μmol/L, and potassium level of 3.4 mmol/L. Chest X-ray showed left lower zone consolidation. Abdominal X-rays did not reveal any faecal-loaded bowel. She was given intravenous amoxicillin-clavulanate for her pneumonia and fluid replacement for the dehydration.

Further review yielded a history of dyspepsia on-and-off for over 13 years and three previous upper endoscopies showing unremarkable findings. She had had eosinophilia for 6 years, which fluctuated between 15 and 38% (absolute eosinophil count 0.6-2.0 x 10^9/L; reference range, 0.2-0.45 x 10^9/L). Six months ago, she had an episode of *Clostridium perfringens* bacteraemia. Despite extensive investigations including gynaecological examination, ultrasound of the abdomen and pelvis, and bone scans, the source of her infection was not identified. Further investigation of her chronic diarrhoea showed negative real-time polymerase chain reaction stool results for norovirus, bacterial culture, as well as *Clostridium difficile* and its cytotoxin. Investigation of her thyroid function, iron status, and serum vitamin B12 and folate levels yielded no abnormality. Finally, microscopy...
of one of her four stool samples revealed the presence of *S. stercoralis* rhabditiform larvae. Urine for *Legionella pneumophila* serogroup 1 antigen was negative. Her serum immunoglobulin (Ig) E level was 770 (reference level, < 100) IU/mL. We failed to collect a sputum sample from her. Her daughter said that she was not a farmer but had worked in conservancy services before retirement. With the diagnosis of *S. stercoralis* infection, she was treated with albendazole 400 mg twice daily for 1 week. Both her diarrhoea and stercoralis services before retirement. Before the previous episode of strongyloidiasis infestation in our patient included the intermittent epigastric pain, fluctuating eosinophilia, chronic diarrhoea, and her previous occupation. The extensive scratch marks over her thighs is also a clue for strongyloidiasis that was stressed in older medical literature. The previous episode of *C. perfringens* bacteraemia could be related to invasion of the gut by colonic flora secondary to penetration of the colonic mucosa by the *Strongyloides* larvae. To our knowledge, the association of clostridium bacteraemia with strongyloidiasis has not been reported previously. The control of *Strongyloides* infection involves innate immunity (eosinophils) and adaptive immunity which involves the specific Igs, in the form of IgG and IgE production by B cells and T-helper 2 responses and corresponding cytokine profiles. Previous studies have shown that T-helper cells are particularly affected in renal failure, which might explain the occurrence of more symptoms ensuing in the patient late in her life. Judging by her gastro-intestinal and respiratory symptoms and her *C. perfringens* bacteraemia, she probably had hyperinfection, although we are unable to save any sputum to demonstrate the presence of *S. stercoralis* in the respiratory tract. During her acute presentation, the patient did not manifest eosinophilia, which might have been related to concurrent pyogenic infection. The current episode of pneumonia may also have been related to bacteria on the surface of *Strongyloides* invading the alveolar space or could have been non-infective (as a result of haemorrhages and/or eosinophilic infiltration) or coincidental.

Regarding diagnostic methods, traditional stool examination has poor sensitivity (30-50%) with

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**Discussion**

*Strongyloides stercoralis* has two separate life cycles, the free-living cycle and the parasitic cycle. The free-living male and female adults mate in the soil and produce eggs that hatch to become the rhabditiform larvae. These larvae become male or female adults in the environment and either establish an external sexual life cycle or differentiate into the infective filariform larvae. Filariform larvae inside the soil penetrate the intact skin (bare foot humans) to start the parasitic cycle. The larvae enter the venous circulation, reach the lungs and penetrate the alveolar spaces. The larvae ascend via the bronchial tree and after being swallowed reach the small bowel. The female worms embed in the submucosa of the duodenum and produce embryonated eggs (up to a dozen per day). The eggs hatch and release the rhabditiform larvae in the intestinal wall. The larvae migrate into the lumen and are either passed into the faeces or mature into filariform larvae. The filariform larvae can infect the intestinal mucosa or peri-anal skin to re-start the parasitic cycle. Through this process of auto-infection, the infection can persist for many years before its discovery. Our patient was probably one of the oldest subjects with this condition described in the literature. Rhabditiform larvae that passed out together with the faeces can become infectious filariform larvae directly, or go through a free-living cycle of development in the soil.

The disease is well-known to occur in farmers through frequent contact with soil contaminated with human faeces. It has also been described as an occupational hazard of miners in Germany through similar mechanism. Our patient had worked in the conservancy services before her retirement. Before the 1960s, most houses for the general public in Hong Kong did not have any domestic sewage collection system. The faecal domestic waste was collected by workers every day from 12 midnight till 6 am in the morning. Such faecal domestic waste was either transported to the New Territories or to Mainland China to be used as fertiliser. This work was often undertaken by women with a low level of education. Moreover, when they were working they did not wear any gloves or mask. Our patient probably acquired the infection whilst handling contaminated faeces. This case illustrates how elderly persons who had previously worked in the conservancy services might become infected. Such elderly persons may regard their previous occupation as an embarrassment and may not willingly volunteer this part of their history.

The clues to the presence of chronic strongyloidiasis infestation in our patient included the intermittent epigastric pain, fluctuating eosinophilia, chronic diarrhoea, and her previous occupation. The extensive scratch marks over her thighs is also a clue for strongyloidiasis that was stressed in older medical literature. The previous episode of *C. perfringens* bacteraemia could be related to invasion of the gut by colonic flora secondary to penetration of the colonic mucosa by the *Strongyloides* larvae.

To our knowledge, the association of clostridium bacteraemia with strongyloidiasis has not been reported previously. The control of *Strongyloides* infection involves innate immunity (eosinophils) and adaptive immunity which involves the specific Igs, in the form of IgG and IgE production by B cells and T-helper 2 responses and corresponding cytokine profiles. Previous studies have shown that T-helper cells are particularly affected in renal failure, which might explain the occurrence of more symptoms ensuing in the patient late in her life. Judging by her gastro-intestinal and respiratory symptoms and her *C. perfringens* bacteraemia, she probably had hyperinfection, although we are unable to save any sputum to demonstrate the presence of *S. stercoralis* in the respiratory tract. During her acute presentation, the patient did not manifest eosinophilia, which might have been related to concurrent pyogenic infection. The current episode of pneumonia may also have been related to bacteria on the surface of *Strongyloides* invading the alveolar space or could have been non-infective (as a result of haemorrhages and/or eosinophilic infiltration) or coincidental.

Regarding diagnostic methods, traditional stool examination has poor sensitivity (30-50%) with
a single sample, but can be improved to about 80% with multiple samples. In our patient, the organism was only detected in the fourth stool sample. The sensitivity can also be improved to around 96% with agar cultures, in which the stool is placed on a nutrient agar plate for 2 days and thereafter tracks can be detected as the larvae carry bacteria during their movement. Enzyme-linked immunosorbent assays (ELISA) to detect antibodies in the serum have high sensitivity (83-93%) and specificity (95-98%), but the test is limited by its inability to differentiate current from past infection, as well as the need for a constant supply of filariform larvae. Other newer ways include faecal ELISA to detect antigens in faecal samples, serum indirect fluorescent antibody test (sensitivity 97% and specificity 100%), and real-time polymerase chain reaction to detect *S. stercoralis* DNA in faecal samples (high sensitivity and 100% specificity). Other methods include detection of *S. stercoralis* by collecting bronchoalveolar lavage or gastric aspirate via bronchoscopy or endoscopy directly in patients with disseminated strongyloidiasis presenting with gastro-intestinal bleeding and/or significant respiratory symptoms.

The drug of choice for strongyloidiasis should be ivermectin 200 μg/kg with two doses that are given 1 to 14 days apart and has a cure rate of 94 to 100%. Our patient was treated with albendazole because ivermectin was not available in our hospital. Albendazole 10 mg/kg/day has a lower efficacy of 38 to 45%, hence relapse is possible. Our management plan was to follow-up the patient, monitor any abdominal symptoms, and perform repeated stool sampling to assess the efficacy of therapy.

In summary, the combination of eosinophilia and abdominal symptoms should raise the suspicion of strongyloidiasis. Repeated stool samples should be collected to look for any *S. stercoralis*. In the history, farming as well as working in conservancy services should be explored. Ivermectin is the drug of choice for treatment of strongyloidiasis, failing that albendazole can be used. Strongyloidiasis may be associated with clostridial bacteraemia.

References

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