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<th><strong>Title</strong></th>
<th>Strongyloidiasis in a nonagenarian who previously worked in conservancy services</th>
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<td><strong>Author(s)</strong></td>
<td>Shea, YF; Chau, KM; Hung, IFN; Chu, LW</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 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, ultrasonography 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 *Clostridiun 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 pneumonia resolved after treatment.

**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 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 IgEs, 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
References

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