Tidsskriftet Tidsskrift for Den norske legeforening

Multimorbid woman in her sixties with abdominal pain and dark stools

EDUCATIONAL CASE REPORT

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Background

A middle-aged woman was admitted to hospital several times in a period of six weeks where the underlying cause of her symptoms was related to a disease rarely seen in Norwegian hospitals.

Case presentation

The patient was born and raised in Southeast Asia, but had lived in Norway since the 1970s. She was prescribed occasional courses of prednisolone for gout. The patient presented at hospital with abdominal pain. Examinations revealed gastrointestinal bleeding, metabolic acidosis and gram-negative sepsis. However, a ventricular biopsy revealed an underlying aetiology of *Strongyloides stercoralis* larvae.

Interpretation

Strongyloidiasis is an infection caused by the intestinal nematode *Strongyloides stercoralis*. Transmission mainly occurs in tropical and subtropical areas, and the primary mode of infection is through larvae penetrating the skin. The parasite has a complex life cycle and, due to autoinfection, an infected person can have an active infection for several decades after leaving an endemic area. Subsequent impairment of host immunity can lead to accelerated autoinfection and cause hyperinfection, which could be fatal if left untreated. Before initiating immunosuppressive therapy, all patients should be considered for serological screening for strongyloidiasis, regardless of the time since possible exposure.

A woman was admitted to hospital on repeated occasions with various medical issues. The underlying cause turned out to be a potentially life-threatening complication of a disease that is likely to be underdiagnosed in Norway.

A woman in her sixties was admitted to hospital as an emergency with abdominal pain, low haemoglobin, dark stools and suspected gastrointestinal bleeding. She had been living in Norway for decades but was originally from Southeast Asia, and had visited her country of origin regularly. She was known to have α-thalassemia, gout, diet-controlled type 2 diabetes and hypertension. She had been hospitalised the preceding year with cerebral haemorrhage. A brain MRI three months later found regression of the haemorrhagic lesions and a small sequela of cerebral infarction, after which treatment with clopidogrel and atorvastatin was initiated. Due to repeated flares of gout and a history of presumed allergic reaction to allopurinol, the patient's general practitioner had intermittently prescribed 5 mg prednisolone and probenecid tablets. The patient had recently been started on treatment with daily 20 mg prednisolone tablets due to an episode of gout.

In the emergency department, the woman was hypertensive with blood pressure of 187/81 mmHg. Other vital signs were normal. A faecal occult blood test was positive, and the patient's haemoglobin was 9.8 g/dL (reference range 11.7–15.3). Since there was no suspicion of an acute life-threatening haemorrhage, she was started on a proton-pump inhibitor and received an iron infusion. She was discharged and referred for rapid outpatient gastroscopy due to a presumed gastric ulcer caused by the combination of prednisolone and clopidogrel. The clopidogrel was paused for five days, and the prednisolone was discontinued without tapering.

The woman was re-admitted two days later with non-specific twitching in her extremities, but she was not otherwise uncomfortable or feverish. She reported that her general condition had been poor in the previous few days, with pollakiuria and a slight cough over the preceding 2–3 weeks, but no other specific symptoms. Her haemoglobin was 9.1 g/dL (11.7–15.3), CRP 94 mg/L (< 5), leukocytes 9.87 × 10^9/L (3.5–10.0 × 10^9) and eosinophils 0.24 × 10^9/L (< 0.4 × 10^9). Creatinine was 109 µmol/L (45–90), sodium 132 mmol/L (137–145) and potassium 4.7 mmol/L (3.5–5.0). She had high plasma glucose of 24 mmol/L (fasting 4.0–6.0) and HbA1c of 82 mmol/mol (20–42). A chest X-ray showed a small retrocardiac opacity, unchanged from previous imaging. A urine dipstick was positive for nitrite, protein 1+, ketones 2+, glucose 4+, but she did not have pyuria or dysuria.

Unfortunately, urine was not sent for culturing before the initiation of ampicillin and gentamicin adjusted for renal function to treat a suspected respiratory or urinary tract infection. Half a day later, blood cultures were found to be positive for Klebsiella pneumoniae.

A short while later, the woman started to vomit and passed a large amount of melena. She became hypotensive (blood pressure 94/38 mmHg), tachycardic (heart rate approximately 120 bpm), somnolent and exhibited an inadequate response when addressed. She did not have hypoxemic respiratory failure or dyspnoea, but arterial blood gas analysis revealed metabolic acidosis with respiratory compensation and pH 7.44 (7.36–7.44), pCO_2 2.1 kPa (4.5–6.1), pO_2 13.1 kPa (9.6–12.4), HCO_3 11 mmol/L (20–26) and base excess –13.1 mmol/L (± 3.0). In addition, capillary blood beta-hydroxybutyrate (ketones in blood) was found to be elevated at 5.3 mmol/L (< 0.6). Her blood pressure increased with intensive fluid therapy, and the antibiotic treatment was changed to cefotaxime. A brain MRI demonstrated small subacute cerebral

infarctions, but the patient had no neurological deficits. Lumbar puncture was not performed due to recent treatment with clopidogrel, and she had a fluctuating level of consciousness associated with severe infection/sepsis. It emerged that, prior to admission, the patient had been on 5 mg prednisolone for longer than previously thought. Due to circulatory failure and suspected iatrogenic adrenal insufficiency as a result of prolonged steroid use and sudden discontinuation of prednisolone, the patient was administered 50 mg intravenous hydrocortisone four times a day.

At that time, the woman's condition was interpreted as urosepsis complicated by ketoacidosis and gastrointestinal bleeding. Sepsis is defined as life-threatening organ failure triggered by a suspected or diagnosed infection. The organ failure is caused by a dysregulated immune response affecting one or more vital organs leading to respiratory or circulatory failure, coagulopathy, hepatic failure, renal failure, and/or a reduced level of consciousness. In most cases, sepsis is caused by bacterial infections, although other microbial infections, such as viral, fungal and parasitic infections, can also trigger sepsis.

Gastroscopy on day two revealed a 2 × 4 cm antral ulcer with no stigmata of bleeding or signs of perforation. It was assumed that a bleeding ulcer was the cause of the decrease in haemoglobin, and no biopsy was taken due to the risk of haemorrhage.

There was a gradual improvement in the patient's condition. On day 13, she was discharged to a nursing home with 500 mg ciprofloxacin tablets twice daily for three days and follow-up gastroscopy arranged for two weeks later. Prednisolone was tapered and discontinued before discharge. The woman was in a fatigued and weakened condition, but she appeared stable and without signs of infection.

She was re-admitted three days later due to elevated CRP, low blood pressure, cardiac arrhythmia, abdominal pain, frequent loose stools and presumed therapeutic failure. She had pitting oedema, was subfebrile (body temperature 37.7°C), tachycardic (heart rate 103 bpm) and hypotensive (blood pressure 77/54 mmHg), but responded well to fluid resuscitation. She had acute renal failure with creatinine of 211 µmol/L (45–90), as well as hyponatraemia 127 mmol/L (137–145) and potassium 4.4 mmol/L (3.5–5.0). The patient's albumin was 18 g/L (32–43), and she was presumed to have intravascular volume depletion. Leukocytes, eosinophils and CRP were elevated at 16.1 × 10^9/ L, 1.23 × 10^9, and 176 mg/L, respectively. Intravenous piperacillin/tazobactam was initiated in the emergency department, but this was rapidly discontinued due to a suspected Clostridioides difficile *infection. Faecal samples were negative for* C. difficile *toxins A and B. The woman developed a fever, and urine culture yielded* Enterococcus faecium. *It was decided to cautiously take this finding into account and start intravenous treatment with linezolid. The patient underwent colonoscopy due to persistent diarrhoea and elevated faecal calprotectin of 523 mg/kg (< 50). There were no macroscopic signs of colitis. Routine mucosal biopsies were taken, including a mucosal sample with the appearance of a sessile polyp.*

The patient was stable, but nauseous and anorectic, and experiencing increasing gout pain. After initiation of 20 mg prednisolone tablets once daily and 0.5 mg colchicine tablets three times daily, her CRP decreased to 11, and

her clinical condition improved with almost normal bowel movements and no abdominal pain. Eosinophils decreased from 3.49 × 10^9/L to 0.78 × 10^9/L. The eosinophilia was interpreted as either non-specific or drug-induced. After eight days in hospital, the woman was discharged to a nursing home with 600 mg linezolid tablets twice daily for a total of 7 days, a short course of prednisolone, nystatin suspension, insulin and nutrition drinks. The findings of the mucosal biopsy were received after discharge and demonstrated both acute and chronic inflammation, but no eosinophilia. No polyp tissue was found.

Eosinophilia can occur in drug-induced allergic reactions, as well as in parasitic diseases, connective tissue disorders and certain cancers (1). In some cases, no specific cause can be found. It is important to take a thorough case history, particularly as regards recently started medication. Although the most common causes of eosinophilia are non-infectious, enquiries should be made about the patient's travel history and country of origin. Consideration should also be given to screening stools with microscopy or PCR testing and serological testing for intestinal worms. Protozoa rarely cause eosinophilia.

The day following discharge, the woman was referred to the emergency department for the fourth time in a month with increasing dyspnoea, heart rate > 130 bpm and recurrence of abdominal pain. On admission, she was somnolent, tachycardic and slightly hypothermic (35.7 °C). She had normal blood pressure and oxygen saturation. Recurrence of severe ketoacidosis was detected with beta-hydroxybutyrate of 6.0 mmol/L, HCO_3 2.0 mmol/L and pH 6.98, despite respiratory compensation and $PaCO_2$ of 1.6 kPa. pO_2 was 15.0 kPa, and lactate was slightly elevated at 2.3 mmol/ L (0.5–1.6). The patient was transferred to the intensive care unit, where she received standard treatment for ketoacidosis. There was no change in her haemoglobin levels but a slight deterioration in her renal function with creatinine at 123 µmol/L. CRP was 9.5 mg/L, leukocytes 22.7 × 10^9/L and eosinophils 0.04 × 10^9/L. Blood cultures were negative, but growth of E. coli *was found in a urine sample. Intravenous cefotaxime was initiated for a presumed urinary tract infection. Intravenous fluids and enteral nutrition were administered due to extreme nausea. Gastroscopy was repeated and revealed a fibrin-covered ulcer, and biopsies were taken from the ulcer margin.*

The woman's condition gradually improved, and after three days she was transferred to a ward. However, she developed a gout flare in her right index finger and recurrence of eosinophilia at 3.06 × 10^9/L (< 0.4). A sample was sent for Strongyloides *serology and a stool sample was sent for microscopy. The findings of the gastric biopsy were returned the same day and demonstrated histopathological changes consistent with* Strongyloides *larvae (Figure 1a). An abundance of* Strongyloides *larvae was also detected in stools, and* Strongyloides *serology was positive with high antibody levels and a* Strongyloides *IgG index of 4.1 (index ≥ 1 is a positive test, ≥ 2 is considered a high level). The colonic biopsies from the previous admission were re-examined and* Strongyloides *larvae were identified (Figure 1b).*

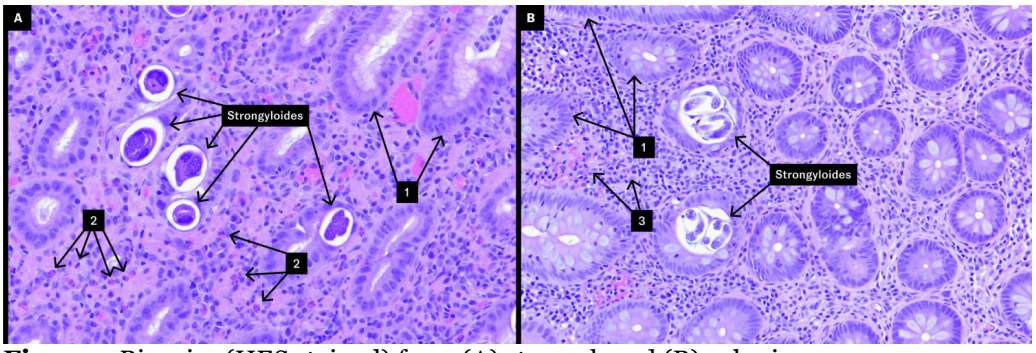

Figure 1 Biopsies (HES stained) from (A) stomach and (B) colonic mucosa demonstrated parasites in crypts (*Strongyloides*) and mild reactive lesions in the crypt epithelium consistent with inflammation (arrows 1). In addition, there were areas of lamina propria with (A) increased number of eosinophils (arrows 2) and (B) increased cellularity with mainly mononuclear inflammatory cells (arrows 3).

The patient was administered 12 mg ivermectin tablets (0.2 mg/kg/day) once daily for a total of 20 days, i.e. 13 days after the first negative microscopic examination of faeces. At an outpatient check-up 3.5 months after discharge, the patient's condition was still improving, but due to elevation in eosinophils she was receiving another course of ivermectin, 9 mg on days 1 and 2, and days 14 and 15, which was the recommended treatment at that time. No larvae were observed on repeat microscopic examination of faeces, and neither were Strongyloides *larvae detected in PCR testing. Follow-up serological testing after 6 and 12 months showed a significant decrease in antibody levels, indicating treatment success.*

Discussion

Strongyloides stercoralis is an intestinal roundworm (nematode) that is considered to be a neglected tropical disease (2, 3). It is widely distributed in tropical and subtropical areas, but is also found in temperate regions of North America, Japan, Australia and Europe. There is considerable variation in the prevalence rate reported by different sources, but the World Health Organization (WHO) estimates that 300–600 million people are affected by the parasite (4).

Strongyloides stercoralis has a complex lifecycle, which can include both a parasitic cycle in humans and a free-living cycle on the ground (Figure 2). Infection usually takes place by the penetration of intact skin by infective (filariform) larvae in soil. The larvae enter the circulation and travel to the lungs, where they migrate across the alveolar walls into the bronchi, move upwards and are coughed up into the throat and then swallowed. In the gastrointestinal tract, the larvae develop into adult female worms that start to produce eggs. The eggs hatch on the intestinal mucosa. The non-infective (rhabditiform) larvae are excreted in the stools and must develop into infective larvae before they can infect a new host. This usually takes place in moist soil. Infective larvae can mature into adult worms, establish a free-living cycle and sexually reproduce in the soil. However, non-infective larvae can also develop

into infective larvae in the intestine and reinfect the host by penetrating the intestinal mucosa or perianal skin. This autoinfection cycle means that active infection can continue for several decades after leaving an endemic region (5).

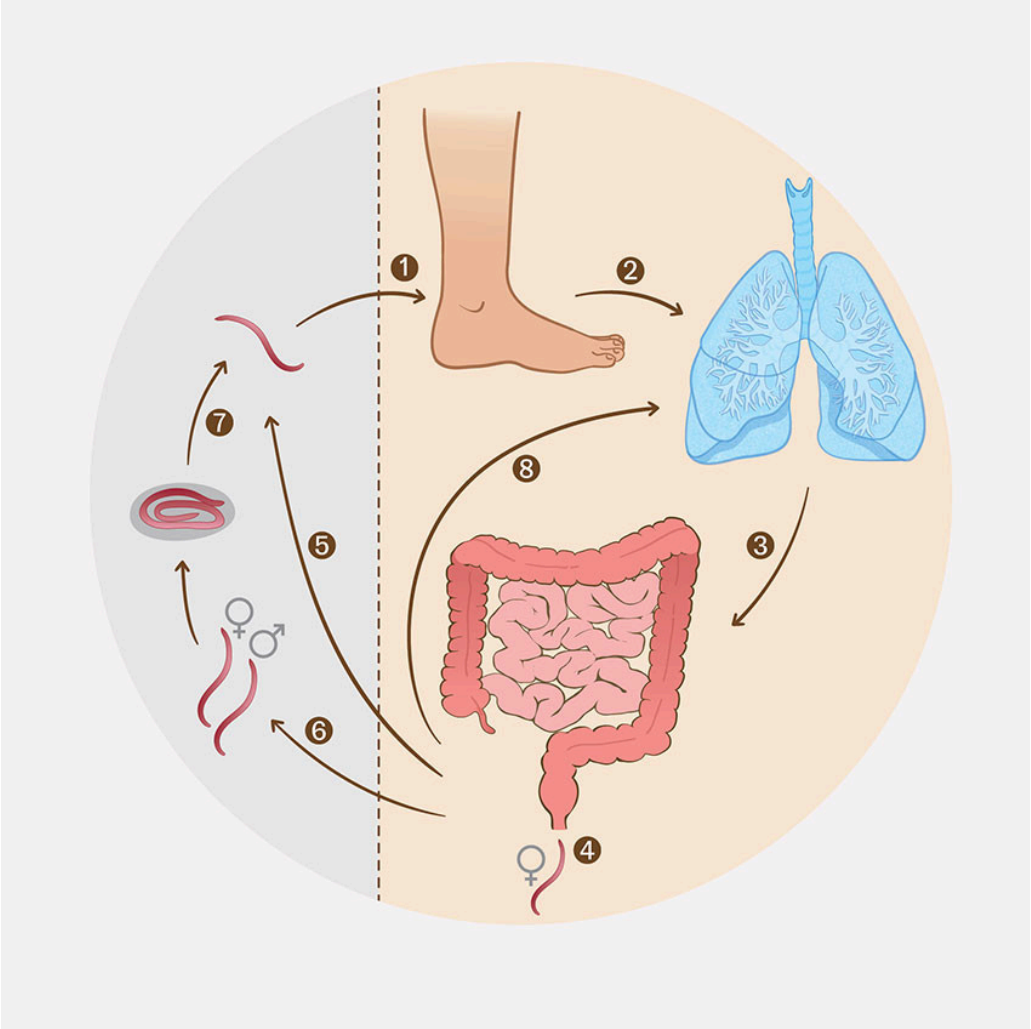

Figure 2 Diagram of the lifecycle of *Strongyloides* inside and outside the human body. Infective (filariform) larvae penetrate intact skin (1). The larvae are transported in blood or lymph fluid to the lungs (2), where they penetrate the alveolar wall, migrate to the pharynx and are swallowed (3). In the intestine, the larvae mature into adult females that lay eggs through asexual reproduction. The eggs hatch into non-infective (rhabditiform) larvae, which are excreted in stools (4). In the soil, the larvae mature directly into infective larvae (5) or into adult male or female worms that reproduce and lay eggs (6). The eggs hatch into non-infective larvae and mature into new infective larvae (7). In addition, non-infective larvae can develop into infective larvae in the intestine, and thus re-infect the host by penetrating the intestinal mucosa or perianal skin (autoinfection) (8). Illustration: Jeanette Engqvist/Illumedic.

The vast majority of infections with *S. stercoralis* are asymptomatic or mild. After larval penetration of the skin, local irritation with itching and possibly urticaria may occur. The rash can last for days or weeks. A few weeks after infection, an intermittent dry cough may develop when the larvae migrate from the lungs into the throat. When the larvae reach the small intestine, diffuse abdominal symptoms may develop, such as abdominal pain, diarrhoea, constipation and poor appetite.

In immunocompromised individuals, infection with *Strongyloides* can cause serious illness and potentially life-threatening complications because the normal immunological 'brake' is inhibited. This can lead to the production of thousands of larvae. This type of accelerated autoinfection with high worm burden in the skin, gastrointestinal tract and lungs is called *Strongyloides* hyperinfection syndrome. In rare cases, widespread (disseminated) disease develops, which can affect any organ of the body. Clinical presentation can vary from chronic unexplained weight loss to acute, life-threatening conditions. A common complication is sepsis with gram-negative bacteria due to translocation of enteric bacteria when larvae migrate from the intestine.

Patients taking corticosteroids are at particular risk because the drugs reduce circulating eosinophils, which play a key role in the immune response against parasites. Even short courses of corticosteroids and doses as low as 20 mg daily are associated with hyperinfection syndrome (6). There is also a presumed increased risk of a severe course of the disease in patients receiving other immunosuppressant treatment (2, 7).

It is possible to become infected by organ transplantation. Norheim et al. have reported two cases from Norway in which the recipients developed hyperinfection syndrome after receiving an organ from the same donor, who was subsequently found to be seropositive for *Strongyloides* (8).

Detection of *Strongyloides* larvae by microscopic examination of faeces has low sensitivity. Therefore, serology is the primary diagnostic tool and is performed at the University Hospital of North Norway. Elevated IgG can be detected approximately six weeks after infection and in cases of chronic infection, with sensitivity of 83–90 % and specificity of 97–98 % (9, 10).

In cases of hyperinfection syndrome, larvae are usually easy to detect on microscopic examination of gastric aspirate or faeces and on histological examination of intestinal biopsies. PCR testing of stools is an alternative diagnostic option. Oslo University Hospital offers specific PCR testing for *Strongyloides*, and *Strongyloides* is included in multiplex helminth PCR testing, which is now available at several Norwegian hospitals. Many patients have elevated eosinophils at some point after infection, but eosinophilia is not always present at the time of diagnosis. Eosinophilia has been found in a third of immunocompromised patients with hyperinfection syndrome, and in these patients eosinophilia was associated with a favourable prognosis (11).

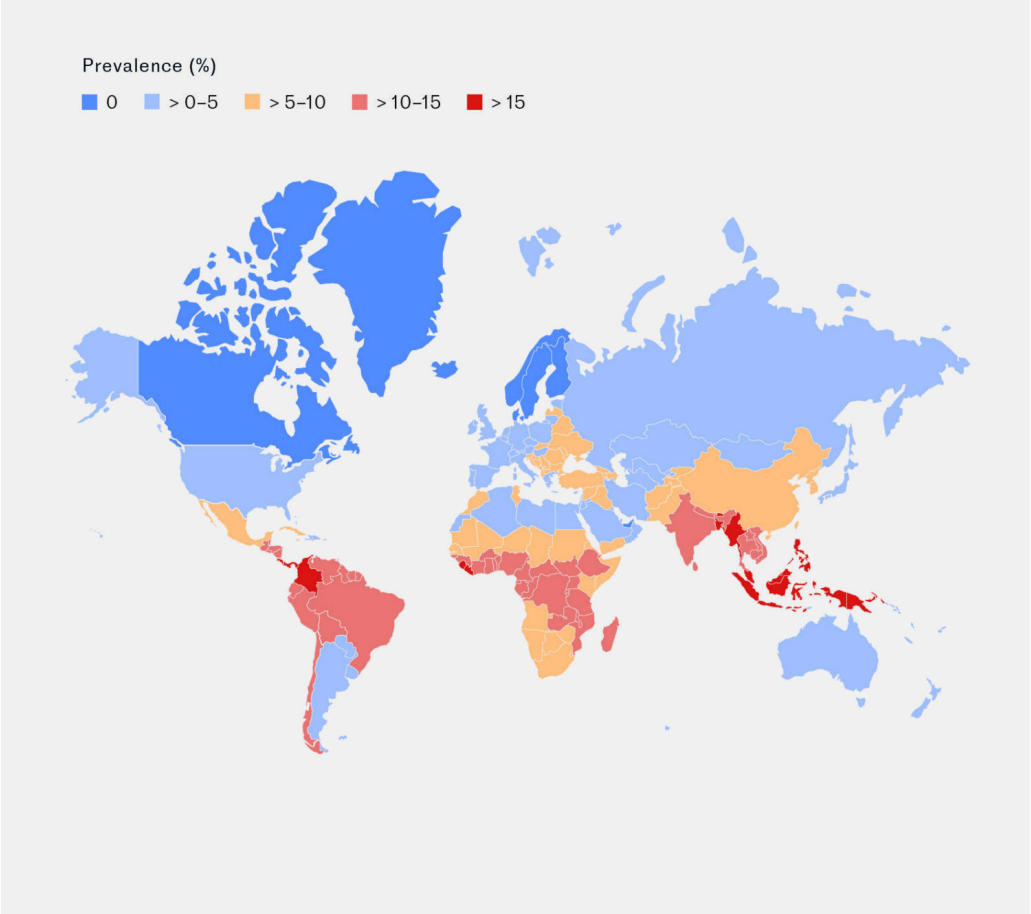

Figure 3 Estimated global distribution of infection with *Strongyloides stercoralis* (4).

The first-line treatment for uncomplicated strongyloidiasis is a single dose of ivermectin, which results in a cure rate of 86–95 % (2, 12). The dose is repeated ≥ 14 days after the first dose (13). It is recommended that all infected individuals, with or without symptoms, are offered treatment due to the risk of developing hyperinfection syndrome if they subsequently become immunosuppressed. In cases of *Strongyloides* hyperinfection, ivermectin must be administered daily using an oral or, if necessary, subcutaneous, formulation until microscopic examination of faeces has been negative for 1–2 weeks. A 50 % decrease in antibody levels and/or normalisation of eosinophilia after 6–12 months indicates a good treatment effect (2, 12).

It is reasonable to assume that *Strongyloides* hyperinfection syndrome with migration of larvae from the gastrointestinal tract was the cause of *Klebsiella* bacteraemia in our patient (14). The acid-base imbalance was presumably due to a combination of diabetes, metformin treatment and renal failure. The woman's HbA1c levels may have been underestimated due to α-thalassaemia, which causes increased destruction and shortened half-life of red blood cells. Decreased food intake over time may have led to 'starvation ketosis' and contributed to the acidosis. Strongyloidiasis can cause diffuse gastrointestinal symptoms, but it is possible that the nausea was to some extent caused by paralytic ileus, which can occur in strongyloidiasis (15). Gastric ulcer is not a common presentation of strongyloidiasis, but it has been reported (16).

Conclusion

Chronic strongyloidiasis is often asymptomatic in immunocompetent individuals. The infection can persist for several decades due to autoinfection, and *Strongyloides* hyperinfection syndrome can occur if the patient later becomes immunocompromised. Deceased organ donors are now screened for *S. stercoralis* in Norway, but there is no routine testing of live donors, recipients or other individuals who are immunosuppressed for other reasons. The use of immunomodulating and immunosuppressant medication is increasing. There should be greater awareness of Strongyloidiasis so that the disease can be detected before it leads to serious consequences, as highlighted by this and similar case reports (17). Consideration should be given to serological testing before starting immunosuppressant treatment in patients who have spent time in endemic areas, even if this was a long time ago.

We would like to thank pathologist Bendik Holmsen Sundrehagen for the images and caption.

The patient has consented to publication of the article.

REFERENCES

1. Klion AD, Ackerman SJ, Bochner BS. Contributions of Eosinophils to Human Health and Disease. Annu Rev Pathol 2020; 15: 179–209. [PubMed] [CrossRef]

2. Gordon CA, Utzinger J, Muhi S et al. Strongyloidiasis. Nat Rev Dis Primers 2024; 10: 6. [PubMed][CrossRef]

3. Olsen A, van Lieshout L, Marti H et al. Strongyloidiasis–the most neglected of the neglected tropical diseases? Trans R Soc Trop Med Hyg 2009; 103: 967–72. [PubMed][CrossRef]

4. Buonfrate D, Bisanzio D, Giorli G et al. The Global Prevalence of Strongyloides stercoralis Infection. Pathogens 2020; 9: 468. [PubMed] [CrossRef]

5. Prendki V, Fenaux P, Durand R et al. Strongyloidiasis in man 75 years after initial exposure. Emerg Infect Dis 2011; 17: 931–2. [PubMed][CrossRef]

6. Ghosh K, Ghosh K. Strongyloides stercoralis septicaemia following steroid therapy for eosinophilia: report of three cases. Trans R Soc Trop Med Hyg 2007; 101: 1163–5. [PubMed][CrossRef]

7. Richards C, Penner J, Colmegna I et al. Methotrexate exposure and risk of strongyloidiasis. Trop Med Int Health 2019; 24: 1032–41. [PubMed] [CrossRef]

8. Nordheim E, Olafsson Storrø M, Natvik AK et al. Donor-derived strongyloidiasis after organ transplantation in Norway. Transpl Infect Dis 2019; 21. doi: 10.1111/tid.13008. [PubMed][CrossRef]

9. van Doorn HR, Koelewijn R, Hofwegen H et al. Use of enzyme-linked immunosorbent assay and dipstick assay for detection of Strongyloides stercoralis infection in humans. J Clin Microbiol 2007; 45: 438–42. [PubMed][CrossRef]

10. Bisoffi Z, Buonfrate D, Sequi M et al. Diagnostic accuracy of five serologic tests for Strongyloides stercoralis infection. PLoS Negl Trop Dis 2014; 8. doi: 10.1371/journal.pntd.0002640. [PubMed][CrossRef]

11. Geri G, Rabbat A, Mayaux J et al. Strongyloides stercoralis hyperinfection syndrome: a case series and a review of the literature. Infection 2015; 43: 691–8. [PubMed][CrossRef]

12. Buonfrate D, Salas-Coronas J, Muñoz J et al. Multiple-dose versus single-dose ivermectin for Strongyloides stercoralis infection (Strong Treat 1 to 4): a multicentre, open-label, phase 3, randomised controlled superiority trial. Lancet Infect Dis 2019; 19: 1181–90. [PubMed][CrossRef]

13. Pettersen FOD, Mørch K. Rundmarker, rundormer og nematoder. Nasjonal metodebok i infeksjonsmedisin. https://metodebok.no/emne/7WVSscZ9/rundorminfeksjoner/h%C3%A5ndbok--i-infeksjonsmedisin-(ous) Lest 30.10.2025.

14. Kassalik M, Mönkemüller K. Strongyloides stercoralis hyperinfection syndrome and disseminated disease. Gastroenterol Hepatol (N Y) 2011; 7: 766–8. [PubMed]

15. Nonaka D, Takaki K, Tanaka M et al. Paralytic ileus due to strongyloidiasis: case report and review of the literature. Am J Trop Med Hyg 1998; 59: 535–8. [PubMed][CrossRef]

16. Costa Silva R, Carvalho JR, Crespo R et al. Strongyloides stercoralis Gastric Ulcer: A Rare Cause of Upper Gastrointestinal Bleeding. GE Port J Gastroenterol 2021; 28: 274–8. [PubMed][CrossRef]

17. Mordal GC, Parker K, Namtvedt T. En nyretransplantert lymfompasient begynner å hoste. Tidsskr Nor Legeforen 2014; 134: 627–30. [PubMed] [CrossRef]

Publisert: 2 February 2026. Tidsskr Nor Legeforen. DOI: 10.4045/tidsskr.25.0318
Received 8.5.2025, first revision submitted 5.10.2025, accepted 30.10.2025.