

Dexamethasone/ivermectin

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Paradoxical dissemination of *Strongyloides stercoralis* infection, *Strongyloides* hyperinfection syndrome and treatment failure: case report

A 61-year-old man developed *Strongyloides* hyperinfection syndrome (SHS) during treatment with dexamethasone for *Escherichia coli* meningitis, and he developed paradoxical dissemination of *Strongyloides stercoralis* infection and experienced treatment failure during antihelminthic treatment with ivermectin [*not all routes and times to reactions onsets stated*].

The man, who had a 3-month history of severe diarrhoeas and 33 pound weight loss, was admitted to the ICU due to impaired consciousness, fever and stiff neck. For the management of community-acquired meningitis, he started receiving empirical anti-infective therapy with cefotaxime, amoxicillin and aciclovir [acyclovir] in addition to dexamethasone 10 mg/6h. Cerebral MRI showed severe cerebral oedema and acute necrotizing encephalopathy. On the day following ICU admission, human T-lymphotropic virus-1 (HTLV-1) infection [*aetiology unknown*] was detected. Lumbar puncture showed pleiocytosis. Also, *Escherichia coli* infection [*aetiology unknown*] was diagnosed. Enhanced abdominal CT scan showed nonspecific wall thickening in the jejunum. Retrospective study revealed untreated *Strongyloides stercoralis* infection, which had been diagnosed 2 months previously. He developed a periumbilical, purpuric rash, which extended to the flanks, after admission. A punch biopsy of the rash confirmed parasitic chronic infection and showed filariform larvae between the collagen bundles in the dermis. After 1 day of treatment for community-acquired meningitis, which included dexamethasone 40mg (total), he started receiving oral ivermectin 200 mcg/kg daily during 2 days for disseminated strongyloidiasis in addition to cefotaxime for *Escherichia coli* meningitis. However, 1 day after the initiation of ivermectin therapy, he presented coma and acute respiratory failure.

Therefore, the man required endotracheal intubation, and subsequently, he experienced severe haemodynamic instability. He developed abundant haemoptysis, with alveolar haemorrhage that evolved towards acute respiratory distress syndrome. A chest CT scan revealed bilateral ground-glass opacities. Numbers of larvae of *Strongyloides stercoralis* were detected in bronchoalveolar lavage. He subsequently developed eosinophilia that remained elevated. He showed haemodynamic and respiratory improvements; however, he suffered severe neurological impairment. A repeat MRI demonstrated a global stability of the lesions. Several EEGs demonstrated unreactive slow wave activity. Control lumbar puncture (4 days after antibiotic initiation) revealed CSF sterilisation. However, stool and sputum smears showed persistent *Strongyloides* larvae. After 3 weeks of intensive care, he did not show any neurological improvement, and care was then withdrawn. He developed complication of fatal multiorgan failure syndrome and died from neurological failure. Autopsy was not performed. It was noted that the treatment with ivermectin failed based on the persistence of larvae in the stool. Additionally, he experienced significant dissemination of *Strongyloides stercoralis* as a paradoxical reaction after initiation of antihelminthic ivermectin treatment, and the abrupt dissemination of *Strongyloides stercoralis*, leading to SHS, had also been caused by treatment with dexamethasone (corticosteroid) without preventive deworming along with HTLV-1 co-infection.