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Enterococcal meningitis associated with *Strongyloides* infection: a case report and literature review

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SUMMARY

Strongyloides stercoralis is an intestinal nematode endemic throughout tropical and subtropical areas, with a life cycle consisting of free-living and parasitic components. Unlike other soil-transmitted nematodes, it is capable of self-infection, which can cause chronic disease that lasts for decades, or cause overwhelming hyperinfection in people taking corticosteroids or other immunosuppressive drugs or who have impaired Th2 cell-mediated immunity, particularly those infected with human T-lymphotropic virus 1. During hyperinfection, a large numbers of larvae have access to the bloodstream, lungs, central nervous system, and other organs. Bacteremia and polymicrobial meningitis can occur due to disruption of the intestinal mucosa and the presence of bacteria on the surface of foreign larvae. Enterococcal meningitis for instance may occur concurrently with strongyloidiasis as a consequence of haematogenous dissemination. We present a clinical case of a 45-year-old, man from Bangladesh, in which co-infection occurred. The patient was not immunocompromized and had no apparent risk factors, which represents the unusual aspect of this case report.
A literature review on enterococcal meningitis and *Strongyloides* coinfection in adult patients was performed encountering 21 cases. Cases have been reviewed and discussed. Clinicians may suspect *S. stercoralis* co-infection when identifying an enterococcal meningitis in adult patients coming from endemic areas.

Key words: Strongyloides, strongiloydiasis, epidemiology, central nervous system, meningitis, Enterococcus, parasitic disease.

INTRODUCTION

Two species of Strongyloides affect humans: *Strongyloides stercoralis* and *Strongyloides fuelleborni*. *S. stercoralis* has a cosmopolitan distribution in tropical and subtropical regions. In average, healthy individuals, the infection causes relatively little over pathology but may compromise an individual's nutritional status. *S. stercoralis* is a soil-transmitted helminth, classified as a roundworm or nematode. *S. stercoralis*, commonly known as threadworm, is a soil-transmitted human parasite belonging to a group of nematodes called roundworms. Although prevalent almost worldwide, except only in the far north and south, the global burden of this parasitic infection is still underestimated because of the unavailability of precise data from endemic areas. *S. stercoralis*, therefore, becomes one of the most overlooked parasitic infections among the "neglected tropical diseases".
There are more than 50 species of *Strongyloides*. Most of them do not affect humans. Strongyloidiasis, the disease caused by *S. stercoralis* in humans,

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is contracted mainly throughout transcutaneous route. Other modes of transmission include the fecal-oral and anal-oral routes. It can also be transmitted through organ transplantation from an infected donor [1].
However, *Strongyloides* infection in immunocompromised individuals (particularly following prolonged exposure to corticosteroids or transplant surgery) can result in disseminated strongyloidiasis, in which worms, particularly larvae, move beyond the confines of the gut into other organ [1].
Enterococci are typically commensal species in humans but become pathogenic under specific host conditions. Infections can be acquired in community and nosocomial settings, affecting various areas such as urinary or biliary tracts, surgical sites, endocardium, and meninges [2].
Enterocooccus species is considered as causative agent for urinary tract infections, hepatobiliary sepsis, endocarditis, surgical wound infection, bacteremia, neonatal sepsis, and nosocomial infection with high mortality. *Enterococcus faecalis* is the most common species associated with clinical infection, while *Enterococcus faecium* poses the higher antibiotic resistance threat. However enterococci are considered an uncommon causative pathogen for central nervous system infection [2]. Enterococcal meningitis accounts for only 0.3% to 4% of cases of bacterial meningitis which is nevertheless associated with a high mortality rate. It has been described most frequently in patients with neurosurgical conditions (i.e. head trauma, shunt devices, or cerebrospinal fluid leakage), although it can also occur as a "spontaneous" infection complicating remote enterococcal infections such as endocarditis or pyelonephritis [3]. During our clinical practice, we encountered a case of an adult patient in which *Strongyloides* infection and enterococcal meningitis coexisted in the absence of apparent risk factors for the latter disease. For this reason, we decided to review the literature on this subject to understand the possible pathological mechanisms underlying this co-infection.

CASE REPORT

We present the case of a 45-year-old man from Bangladesh who presented to the emergency department (ED) with complaints of fever (body temperature up to 39°C) and headache. He also reported an unintentional weight loss of 20 kg. His medical history was characterized by a grade 1 meningeal tumor at the level of the left parasellar site (stable and known since 2016). Chest X-ray and head computed tomography (CT) scan were negative, furthermore neurological evaluation showed no pathological signs. Serology for HTLV and HIV resulted negative. Laboratory examinations showed increased values of C-reactive protein (23.4 mg/L, average value <5 mg/L) and white blood cells (20x10/m^3). After the following investigations, the patient decides to self-discharge. He then re-attended the ED after four days due to persistent symptoms and new onset nausea, complaining of mild nuchal pain. A lumbar puncture was performed in the ED: the cerebrospinal fluid was cloudy with glucose 41.3 (serum/cerebrospinal fluid ratio 0.4), protein 86 mg/dl, 1559 cells/m^3, 70% polymorphonuclear); the multiplex PCR testing for meningitis pathogens (Filmarray meningitis/encephalitis panel, bioMèrieux, Italy) was negative. A new head CT scan was unremarkable. The patient was then admitted to the Infectious Diseases ward and started on empirical therapy with intravenous ceftriaxone 2 g every 12 hours, acyclovir and dexamethasone 10 mg every 6 hours (continued for 11 days). Ampicillin was not added since the patient had no risk factors for *Listeria monocytogenes.* Despite the antibiotic therapy, the patient did not improve, and a second lumbar puncture was then performed. The culture of cerebrospinal fluid resulted positive for *E. faecalis* (fully susceptible to the tested antimicrobials), and a targeted therapy with ampicillin 4 g every 6 hours was added to the previous therapy with ceftriaxone 2 g every 12 hours. Acyclovir was then discontinued. A marked and rapid improvement of headache was observed, and concomitant resolution of *rigor nucalis* after 48 hours/days. Since normocytic anemia (hemoglobin was 8,8 g/deciliter) with a positive fecal occult blood test was detected during hospitalization, he underwent an urgent colonoscopy, which revealed edematous mucosa and the presence of small aphthous ulcers. throughout the colon (Figure 1).
The parasitological examination of *S. stercoralis* was confirmed by stool specimen examination, and the histological examination confirmed the diagnosis of parasitosis. Histological examination showed typical mucosal architecture with a diffuse inflammatory infiltrate consisting mainly of lymphocytes and plasma cells with a fair number

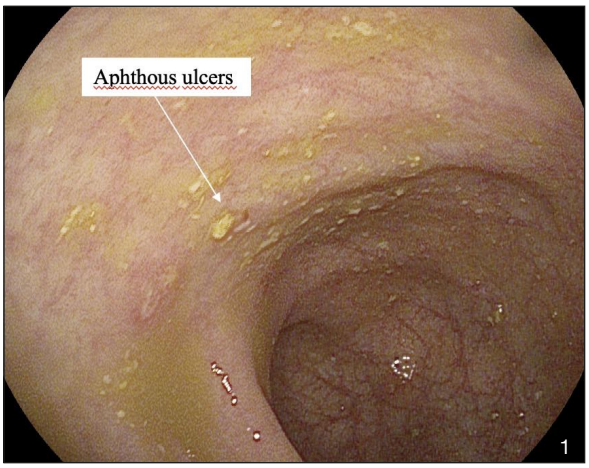

Figure 1 - Image of the colonoscopy- mucosal edema and aphthous ulcers along the colon.

of eosinophilic granulocytes. The eosinophils tended to cluster around the crypts, showing focal aspects of pericryptosis. Some non-necrotizing epithelioid microgranulomas were also present in the *lamina propria*. Some crypts harbored corpuscles morphologically compatible with parasites (Figure 2). Therefore, treatment with ivermectin 15 mg was undertaken with a first dose of the drug repeated one week apart.

The antibiotic therapy lasted 18 days, gradually improving patient health conditions. He was then discharged and followed later by the outpatient service. No relapse events were reported up to 30 days following his discharge.

LITERATURE REVIEW

We performed a literature review of *Strongyloides stercoralis* and enterococci co-infections in either immunocompetent or immunocompromised patients population. In May 2023, we searched PubMed using the following search strings: "MENINGITIS" AND "ENTEROCOCCUS". Overall 325 articles on this topic were identified. From these, we selected 27 articles in which the co-infection of *S. stercoralis* and enterococci was considered. Cases in which there was no microbiological isolation on CSF culture and, therefore no definite diagnosis of enterococcal meningitis were also included in the review. The paediatric population was not taken into consideration. Above all the 27 articles, 6 were excluded for unavailability of an English or Italian version, overall resulting in 21 papers included in the analysis. Inclusion and exclusion criteria are illustrated in the flow-chart reported below.

The cases of *S. stercoralis* and enterococci co-infections reported in the literature (Table 1) include mainly immunocompromised subjects; only 1 patient has no relevant pathological history. Alcoholism, HIV and HTLV-1 infection, hematologic malignancies, post-transplant status, and other im-

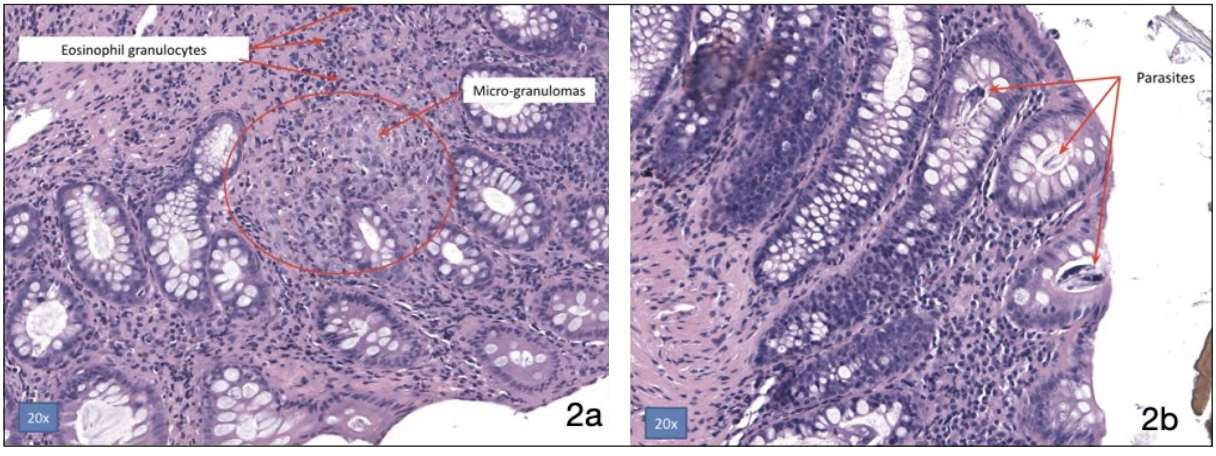

Figure 2 - **a)** Histology of the colon mucosa. haematoxylin and eosin (H&E), original magnification x 200. Three parasites are visible within the intestinal crypts. The lamina propria shows a polymorphous inflammatory infiltrate containing numerous eosinophils; **b)** - Histology of the colon mucosa. H&E, original magnification x 200. The lamina propria shows a moderate inflammatory infiltrate including numerous eosinophils and a small granuloma in formation.

Table 1 - Cases reported in the literature of enterococcal meningitis associated with infection with *Strongyloides*. (CSF: cerebrospinal fluid; CLD: chronic liver disease; HTLV-1: human t-lymphotropic virus; COPD: chronic obstructive pulmonary disease; HBV: hepatitis B virus).

Author	*Year*	*Country*	*Age (Yrs)*	*Gender*	*Co-morbidities*	*CSF (GB) (cells/µl)*	*CSF (glucose-protein) (mg/dL)*	*PCR*	*Blood culture*	*CSF culture*	*Stool culture*	*Microscopy*	*Treatment*	*Duration*	*Treatment (anthelmintic)*	*Outcome*
E Wee [4]	2020	Singapore	82	male	Hypertension; hyperlipidemia; atrial fibrillation	173		negative	*Enterococcus faecalis*	negative	*Strongyloides stercoralis*	no	cefepime, ampicillin	10 days	metronidazol	death
Zeana [5]	2001	USA	69	male	Diabetes mellitus; hypertension; coronary artery disease; autoimmune hemolytic anemia; long term corticosteroids	283	18-195	negative	negative	negative	*Strongyloides stercoralis*	no	linezolid	28 days	albendazole	alive
Tobin [6]	2021	USA	61	male	Obesity; alcohol abuse (10–12 beers per day); atrial fibrillation; COPD	3,375	/- 138	negative	negative	*Enterococcus faecium*	*Strongyloides stercoralis*	no	daptomycin, linezolid, meropenem	17 days	ivermectin	death
Shein [7]	2017	France	28	female	Cervical tumor responsible (HTLV-1) responsibile from dyspnea; weight loss; and asthenia	10	–	negative	*Enterococcus faecalis*	*Enterococcus faecalis; Strongyloides stercoralis*	negative	no	amoxicillin, linezolid	21 days	ivermectin	death
Shreshta [8]	2019	USA	46	male	Dermatomyositis; nephropaty; corticosteroids	–	–	–	*Enterococcus faecalis*	negative	*Strongyloides stercoralis*	no	uknown	uknown	ivermectin	alive
Shresha [8]	2019	USA	36	male	Acute lymphoblastic leukemia	–	–	–	negative	*Areococcus viridans*	*Strongyloides stercoralis*	no	vancomycin	21 days	ivermectin	alive
Einsiedel [9]	2007	Australia	39	female	CLD; HTLV-1	–	–	–	*Enterococcus faecium*	–	negative	larvae	not administered	–	ivermectin	death
Einsiedel [9]	2007	Australia	29	male	HBV; CLD; peripheral neuropathy			–	*Escherichia coli*	–	–	larvae	–	–	not administered	death
Einsiedel [9]	2007	Australia	32	male	CLD	–	–	–	negative	negative	negative	no	Penicillin, gentamicin	–	not administered	death
Einsiedel [9]	2007	Australia	41	male	diabetes type 2; CLD	–	–	–	negative	negative	negattive	larvae	–	–	not administered	death

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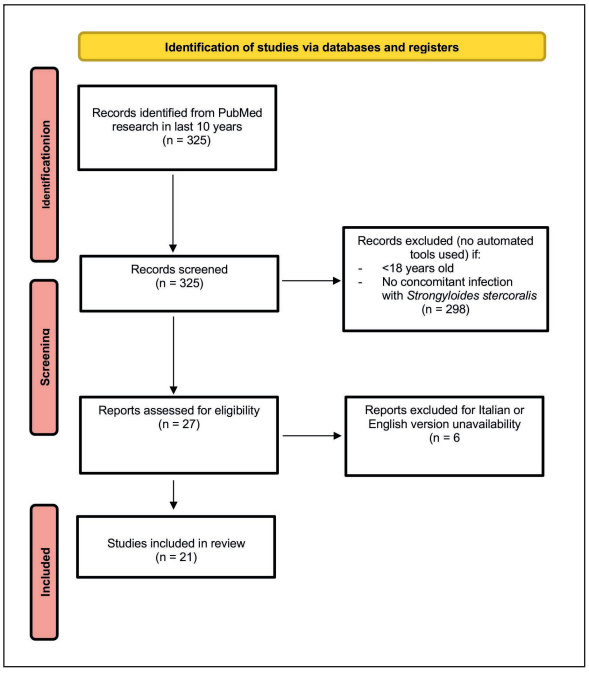
Author	*Year*	*Country*	*Age (Yrs)*	*Gender*	*Co-morbidities*	*CSF (GB) (cells/µl)*	*CSF (glucose-protein) (mg/dL)*	*PCR*	*Blood culture*	*CSF culture*	*Stool culture*	*Microscopy*	*Treatment*	*Duration*	*Treatment (anthelmintic)*	*Outcome*
Einsiedel [9]	2007	Australia	45	male	CLD	–	–	–	*Haemophilus influenzae*	negative	negative	larvae	–	–	albendazole	death
Bamias [10]	2010	Greece	62	male	Long-term use of high-dose methylpred-nisolone; retroperitoneal fibrosis	1500	low/high	negative	negative	*Enterococcus faecalis*	*Strongyloides stercoralis*	larvae	ampicillin, gentamicin	10 days	albendazole	alive
Link [11]	1994	Canada	64	male	Osteoarthritis, COPD	–	–	–	*Enterococcus faecalis*	*Streptococcus bovis*	*Strongyloides stercoralis*	no	vancomycin, gentamicin	28 days	thiabendazole	alive
Somin [12]	2007	Israel	80	male	–	1800	15 -high	Mycobacterium tuberculosis	negative	*Enterococcus faecium*	*Strongyloides stercoralis*	no	ceftriaxone, vancomycin, ampicillin	21 days	not administered	death
Ortega [13]	2019	Spain	52	male	Hypertension; dyslipidemia; corticosteroids	–	–	–	negative	*Enterococcus faecium*	*Strongyloides stercoralis*	no	meropenem, vancomicin	–	ivermectin, albendazole	death
Sugiura [14]	2006	Japan	40	male	HTLV1	pleocystosis	–	–	*Enterococcus faecalis*	*Enterococcus faecalis*	*Strongyloides stercoralis*	no	ampicillin, ceftriaxone	–	ivermectin, albendazol	alive
Khadka [15]	2018	Nepal	56	male	COPD, systemic hypertension; formerly treated pulmonary tuberculosis; corticosteroids	–	–	–	VREfm	–	*Strongyloides stercoralis*	no	linezolid, vancomycin	6 days	ivermectin, albendazol	death
Sahu [16]	2020	Germany	67	male	Mantle cell lymphoma; hematopoietic stem cell transplantation	–	–	–	VREfm	–	*Strongyloides stercoralis*	no	daptomycin	–	ivermectin, albendazol	alive
Zahar [17]	2004	France	52	male	T-lymphoma	41	400-600	–	*Enterococcus faecalis*	*Enterococcus faecalis*	*Strongyloides stercoralis*	no	amoxicillin, gentamicin	–	ivermectin, albendazol	alive
Panosian [18]	1986	USA	58	male	–	–		–	negative	–	*Strongyloides stercoralis*	no	–	–	ivermectin, albendazol	death
Panosian [18]	1986	USA	67	male	Oropharingeal carcinoma	–	–	–	*Streptococcus faecalis*	–	*Strongyloides stercoralis*			–	ivermectin, albendazol	alive

munocompromising therapies are known risk factors for persistent *S. stercoralis* infection and also increase the risk of hyperinfection syndrome and disseminated strongyloidiasis. Many of the patients appear to have been exposed to prolonged corticosteroid treatment prior to hospitalization due to infection-related symptoms, which could be considered as an additional risk factor. A higher frequency of SEC is also observed in male than female subjects.

In many 15 of the cases mentioned, data on the chemical-physical characteristics of the cerebrospinal fluid were not reported. Among the cases mentioned, 13 of them were found to be positive to culture isolations (blood cultures; CSF) for enterococci, attesting to the invasiveness of the infection by this etiological agent. Almost all reported cases are characterized by the identification of *S. stercoralis* through culture, microscopic, or histological testing. Unfortunately, 12 of the reported cases result in early death despite timely administration of broad-spectrum antibiotic therapy.



DISCUSSION

Strongyloidiasis has been recently considered a neglected tropical disease by the World Health Organization due to its global distribution and high burden all over the world. Since the parasite can be continuously auto-infecting the host, many patients remain asymptomatic or have mild symptoms for decades, even after leaving the endemic area and going unnoticed. Rhabditiform larvae are excreted in the stools of infected individuals and can develop into infective filariform larvae in warm, moist soil, which can pierce intact skin and affect a new host, invade the lungs via the bloodstream, migrate to the small intestine to settle, reach maturity, and replicate. *S. stercoralis* has two ways of auto-infection: the rhabditiform larvae de-

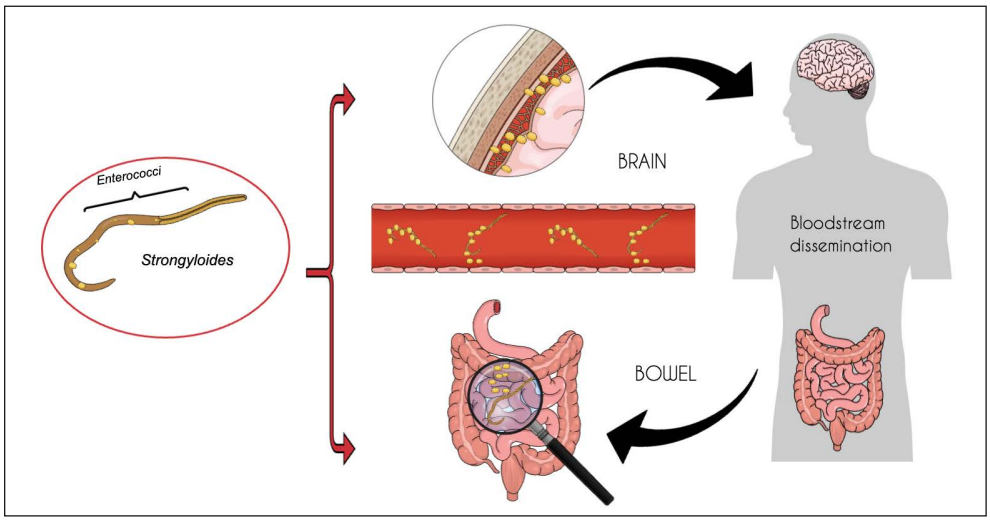


Figure 3 Disseminated strongyloidiasis is characterized by diffusion of gut flora into the bloodstream, leading to bacteremia.

velop into filariform larvae and then enter the bloodstream from the intestine, or they do not develop into filariform larvae but enter the bloodstream directly from the intestinal mucosa (endo auto-infection) [4].

The clinical presentation of strongyloidiasis is variable in terms of both symptoms and severity. Strongyloidiasis can be acute, chronic, persistent, or severe disseminated infection, which can involve multiple organs [19-21]. The enteric bacteria are also carried by invasive larvae on their outer surfaces, this can result in septicemia and in meningitis, which represent a disseminated bacterial infection. Disseminated disease is characterized by the presence of parasites in organs outside the traditional life cycle sites, such as the central nervous system (Figure 3) [8, 9].

An analysis of the cases reported by the literature result that most of them are characterised by a relevant clinical history: only two of the reported cases did not show a clinical history suggestive of immunosuppression. This aspect reinforces the peculiarity of this clinical case suggesting the hypothesis of investigating the presence of a co-infection in those cases where intestinal translocation is suspected. The presence of a chronic strongyloidiasis could intercur when a diagnosis of enterococcal meningitis is carried-out by clinicians. The presence of *S. stercoralis* infection in fact may predispose immunocompromised population as either patients presenting just the epidemiological criterion without other apparent risks factors.

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