Fatal Gastric Mucormycosis and Strongyloidiasis in a Patient with Dapsone Hypersensitivity Syndrome

Dear Editor,

Mucormycosis is an important and dangerous emerging opportunistic infection in diabetics and immunocompromised individuals.[1] Hyperinfected or disseminated strongyloidiasis is a relatively lesser-known complication following immunosuppressive therapy. In the literature, hyperinfection syndrome is usually defined as involvement of the skin, gastrointestinal tract, and/or pulmonary system (the usual pattern of natural infection). In contrast, diagnosis of disseminated strongyloidiasis is considered when there is involvement of organs other than the skin, gastrointestinal tract, or lungs (i.e., sites not generally included in the traditional life cycle).[2] Mucormycosis and strongyloidiasis co-infection in a single patient is rarely reported in medical literature.[3] Here, we report a 50-year-old woman with refractory erythroderma, who developed features of sepsis and gastric ulceration, that retrospectively was found to be due to co-infection with *Mucor* spp. and *Strongyloides stercoralis*.

A 50-year-old woman was admitted to our hospital with an erythroderma of 2 weeks duration due to dapsone hypersensitivity syndrome. Dapsone was prescribed by a local practitioner for a hypopigmented patch on the face, with a presumptive diagnosis of leprosy. Repeated skin biopsies revealed spongiotic dermatitis. She was managed with incremental doses of oral prednisolone up to 1 mg/kg/day and prophylactic oral proton pump inhibitors, (as she had a history suggestive of acid peptic disease), for three months. As there was no response, cyclosporine was added at a dose of 3 mg/kg/day. She subsequently developed isolated thrombocytopenia (37,000 cells/mm^3) and possibly steroid-induced diabetes mellitus. Her diabetes was managed with insulin and cyclosporine was withdrawn.

Lack of response to steroids and cyclosporine necessitated the use of intravenous immunoglobulin 2 g/kg given over 5 consecutive days; following which her erythroderma improved markedly. Subsequently, oral prednisolone was tapered to 0.5 mg/kg/day. Two weeks later the patient developed persistent abdominal pain with nausea and vomiting. Oral steroids were withheld, and she was managed with intravenous hydrocortisone 100 mg q12h and pantoprazole 40 mg once a day. An upper gastrointestinal endoscopy was performed that revealed a large ulcer of length 10 cm, with patchy blackish discoloration along the lesser curvature of stomach. This lesion was biopsied. Subsequently, the patient's general condition deteriorated, and she developed fever and hypotension. Work up for bacterial infections including blood cultures were sterile and she did not respond to broad spectrum antibiotics and other supportive management. Due to the progressive deterioration of her clinical condition, she was discharged against medical advice as per wish of her relatives. She passed away at home after 3 days. The histopathological report that was reported subsequently revealed fully eroded gastric mucosa invaded by broad obtuse angled ribbon shaped branching aseptate hyphae suggestive of mucormycosis [Figure 1] and sections of strongyloides larva with hooklets and suckers [Figure 2].

Mucormycosis, an opportunistic infection caused by *Mucorales* organisms, is reported to be the third most common invasive fungal infection after candidiasis and aspergillosis.[1] It is characterized by angioinvasion and tissue destruction in immunosuppressed patients.[1] The most common type of mucormycosis is rhino cerebral; others being gastrointestinal, pulmonary, and cutaneous.[4] Gastrointestinal infection is thought to occur by ingestion of spores through contaminated food,[4] which was probably the mode of entry in our patient. Mortality is high (98%)[4] and antemortem diagnosis is made only in 25-30% of patients.[1] The mainstays of treatment are control of underlying disease or immunosuppression, antifungal therapy, and aggressive surgical debridement. Among antifungals, amphotericin B and posaconazole are found to be effective, while the *mucor* fungus is intrinsically resistant to voriconazole.

Disseminated strongyloidiasis is one of the opportunistic infections reported in immunocompromised patients with dermatological diseases, because of its propensity for autoinfection.[5] Gastric strongyloidiasis is rare; however, achlorhydria due to prolonged use of proton pump inhibitors has been reported to increase the risk.[6] Our patient, belonging to a region endemic for strongyloidiasis,

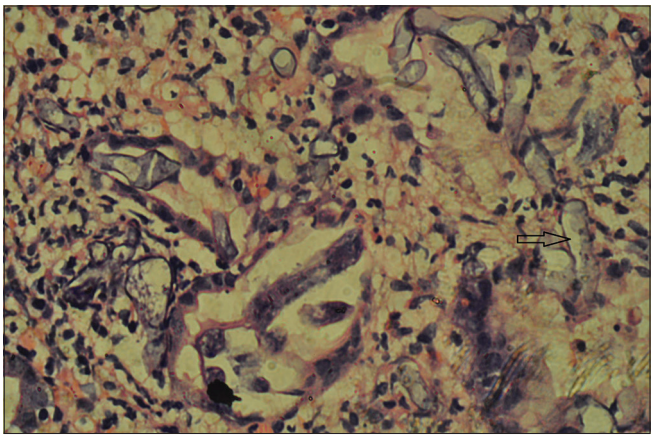

Figure 1: Section from gastric mucosa showing erosion with multiple broad obtuse-angled branching aseptate hyphae (arrow) (H and E, 40×)

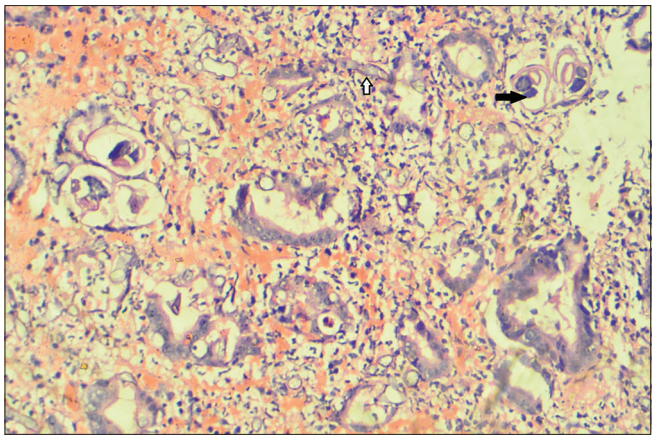

Figure 2: Section from gastric mucosa shows a cut section of *Strongyloides stercoralis* with hooklets and suckers (black arrow) and a few fragments of broad aseptate hyphae (white arrow) (H and E, 40×)

was on immunosuppressants and proton pump inhibitors and had developed diabetes mellitus, thus significantly increasing her risk of acquiring both these infections. The mortality of disseminated strongyloidiasis is high, ranging from 60-70%.[3] The prognosis of the co-infection is not known as there are only few reports in the literature. The usual reported causes of fatality in disseminated strongyloidiasis are Gram-negative sepsis, respiratory failure, and gastrointestinal bleeding.[5]

These relatively rare opportunistic infections are not usually considered by dermatologists while managing patients with high dose steroids. In a rare coincidence, our patient seems to have been affected simultaneously by both these potentially dangerous pathogens. The importance of regular deworming of patients in endemic areas on immunosuppressants should not be underestimated. Therefore, this report serves to stress that clinicians, and dermatologists in particular, need to have a high index of suspicion when faced with a chronic patient on immunosuppresants having persistent upper abdominal symptoms.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Saritha Mohanan, Laxmisha Chandrashekhar[1], Debdatta Basu[2], Devinder Mohan Thappa[1]

Department of DVL, IGMC and RI, Pondicherry, [1]Department of Dermatology, JIPMER, Pondicherry, [2]Department of Pathology, Mahatma Gandhi Medical College and Research Institute, Pondicherry, India

Address for correspondence:
Dr. Laxmisha Chandrashekhar, Department of Dermatology, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry, India.
E-mail: laxmishac@gmail.com

References

1. Lo OSH, Law WL. Ileocolonic mucormycosis in adult immunocompromised patients: A surgeon's perspective. World J Gastroenterol 2010;16:1165-70.
2. Abad CLR, Bhaimia E, Schuetz AN, Razonable RR. A comprehensive review of *Strongyloides stercoralis* infection after solid organ and hematopoietic stem cell transplantation. Clin Transplant 2022;36:e14795.
3. Autier B, Chesnay A, Mayence C, Houcke S, Guégan H, Demar M, *et al*. From strongyloidiasis to disseminated mucormycosis: The story of an HTLV-1-infected migrant. J Travel Med 2021;28:taaa229.
4. Prasad N, Ram R, Satti Reddy V, Dakshinamurty K. Non-fatal gastric mucormycosis in a renal transplant patient and review of the literature. Transpl Infect Dis 2006;8:237-41.
5. Sirka CS, Rout AN, Patra S, Nayak HK. Strongyloides hyperinfection syndrome in dermatology in-patients: A case series. Indian Dermatol Online J 2022;13:94-7.
6. Lam CS, Tong MKH, Chan KM, Siu YP. Disseminated strongyloidiasis: A retrospective study of clinical course and outcome. Eur J Clin Microbiol Infect Dis 2006;25:14-8.

Access this article online

Website: https://journals.lww.com/idoj

DOI: 10.4103/idoj.idoj_209_24

Quick Response Code

How to cite this article: Mohanan S, Chandrashekhar L, Basu D, Thappa DM. Fatal gastric mucormycosis and strongyloidiasis in a patient with dapsone hypersensitivity syndrome. Indian Dermatol Online J 2025;16:469-70.

Received: 05-Mar-2024. **Revised:** 14-Jun-2024.
Accepted: 15-Jun-2024. **Published:** 20-Jan-2025.