



## Fungi caused by Neuroinfections Diseases

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### Letter to the Editor

Fungal infections of the central nervous system (FIs-CNS) have become significantly more common over the past 2 decades. Invasion of the CNS largely depends on the immune status of the host and the virulence of the fungal strain. Infections with fungi cause a big morbidity in immune compromised hosts, and therefore the involvement of the CNS may cause fatal consequences [1].

While it's estimated that 1.5 million fungal species exist, only about 70,000 are formally described. Of the described species, 300 may show virulence to humans, and only 10-15% of these could influence the CNS. Clinically relevant fungi being etiological agents of fungal infections of the CNS include yeasts, filamentous fungi, and dimorphic fungi. Yeasts are unicellular organisms and include the cosmopolitan fungal species of genera *Candida* and *Cryptococcus*, and less common fungi such as *Trichosporon* spp. The filamentous fungi, which are characterized by branching hyphae, include moniliaceous (light-colored) moulds with septate hyphae (*Aspergillus* spp., *Fusarium* spp.) and Mucoromycetes with non-septate hyphae (*Rhizopus*, *Rhizomucor*, and *Mucor*). They have worldwide distribution and are common causes of fungal CNS infections. Pigmented moulds (darkly pigmented) are seen less common and include species which are considered as true neurotropic fungi such as *Cladophialophora bantiana*, *Exophiala dermatitidis* (encountered worldwide, common in East Asia), *Rhinocladella mackenziei*, and *Verruconis gallopava* (syn. *Ochroconis gallopava*, worldwide). The dimorphic fungi with two morphological stages: mould in environment (25°C) and yeast in tissue (37°C) (*Blastomyces*, *Histoplasma*, *Coccidioides*, and *Paracoccidioides*) are geographically restricted to specific endemic areas (see part Dimorphic fungi) [2].

The incidence of fungal infections is increasing per annum, with greater numbers of infections noted among patients belonging to high-risk groups like HIV-infected persons and AIDS patients, transplant recipients, and immunosuppressed patients treated with chemotherapeutics or corticosteroids, also as those affected by haematological disorders and chronically ill patients. Certain conditions may predispose the patient to the event of a selected etiological agent: disease/treatment-associated and genetic factors (prolonged antibiotic therapy, neutropenia, steroid therapy, transplantation, chronic granulomatous disease, CARD9 deficiency, neurosurgery, contaminated devices, and prematurity in infants *Candida*; diabetic ketoacidosis, necrotic burns, kidney failure, and intravenous drug use—Mucoromycetes; contact with birds—*Cryptococcus* and *Histoplasma*; deferoxamine therapy and iron overload—Mucoromycetes.

However, some fungi, like *Cryptococcus*, *Coccidioides*, and *Histoplasma*, also can cause infection in immunocompetent patients. In USA, it had been estimated that invasive mycoses caused by *Candida* spp. are responsible for 72 to 228 infections per million populations annually, while *Cryptococcus neoformans* is responsible for 30–66 infections and *Aspergillus* spp., 12–34 infections. The most common CNS fungal infection worldwide is cryptococcal meningoencephalitis [3].

The FIs-CNS can have various clinical presentations, mainly meningitis, encephalitis, hydrocephalus, cerebral abscesses, and

stroke syndromes. The etiologic factors of neuroinfectious are yeasts (*Cryptococcus neoformans*, *Candida* spp., *Trichosporon* spp.), moniliaceous moulds (*Aspergillus* spp., *Fusarium* spp.), Mucoromycetes (*Mucor* spp., *Rhizopus* spp.), dimorphic fungi (*Blastomyces dermatitidis*, *Coccidioides* spp., *Histoplasma capsulatum*), and dematiaceous fungi (*Cladophialophora bantiana*, *Exophiala dermatitidis*). Their common route of transmission is inhalation or inoculation from trauma or surgery, with subsequent hematogenous or contiguous spread. As the manifestations of FIs-CNS are often non-specific, their diagnosis is extremely difficult. A fast identification of the etiological factor of neuroinfection and therefore the application of appropriate therapy are crucial in preventing an often fatal outcome [4].

Although the amount of fungal species causing CNS mycosis is increasing, just some possess well-defined treatment standards (e.g., cryptococcal meningitis and CNS aspergillosis). The early diagnosis of mycosis, amid identification of the etiological factor, is required to permit the choice of effective therapy in patients with FIs-CNS and limit their high mortality [5].

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