Current Research in Microbiology

Chapter 1

Microsporidian Parasite Impact on Humans Health

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Abstract

Microsporidiosis is an emerging and opportunistic infection coupled with a broad range of clinical syndromes in humans. Microsporidian parasite infectivity has been identified in a wider range of human populations that includes persons with HIV infection, travellers, children, organ transplant recipients, and the elderly. Human immunodeficiency virus-positive patient with chronic diarrhoea, anorexia, and lethargy revealed the presence of numerous refractile bodies resembling microsporidian spores. Questions still exist about whether Microsporidia infections remain unrelenting in immune-competent individuals, re-activate during conditions of immune compromise, or may be transmitted to others at risk, such as during pregnancy or through organ donation. Therefore, this book chapter highlights the research on microsporidiosis in humans.

Key Words: Microsporidiasis; Emerging; Opportunistic; Immunodeficiency; Immune-competent

1. Introduction

The term microsporidia refers to a group of obligate intracellular protozoan parasites belonging to the phylum Microspora. Their host range is extensive, including most invertebrates and all classes of vertebrates [1]. More than 100 microsporidial genera and almost 1,000 species have now been identified [2]. The first human case of sufficiently substantiated microsporidial infection was reported in 1959 [3]. Yet, as part of the budding deadly disease of HIV infection, microsporidia have gained attention as opportunistic pathogens. To date, five genera (*Enterocytozoon* spp., *Encephalitozoon* spp., *Pleistophora* spp., and *Nosema* spp.), as

well as unclassified microsporidial organisms (referred to by the collective term Microsporidium), have been associated with human disease, which appears to predominantly affect immunocompromised persons [4]. *Enterocytozoon bieneusi* has been well documented as a cause of chronic diarrhoea in human immunodeficiency virus-infected patients [5]. The potential sources and means of transmission of human microsporidial infections are uncertain. Preliminary connections have indicated that microsporidial species first identified in patients with AIDS will not be restricted to this patient group.

2. Taxonomy and Biological Characteristics of Microsporidian parasite

The Microsporidia are ancient eukaryotes lacking mitochondria, current data suggests that they are linked to the Fungi [6,7]. With the completion of the *E. cuniculi* genome, the phylogenetic relationship between microsporidia and fungi has been further solidified by the presence of numerous genes that on phylogenetic analysis cluster the Microsporidia with the Fungi [8]. The developmental stages preceding the spore are structurally simple cells, a thick wall consisting of an electron-dense proteinaceous exospore, an electron-lucent chitinous endospore layer, and a plasma membrane renders the spores environmentally resistant. The spore wall encloses the uni-or binucleate infective spore content (sporoplasm), an exceptional extrusion apparatus (the polar tubule) for injecting the sporoplasm into new host cells, a complex membrane system (termed lamellar polaroplast) surrounding the straight section of the polar tubule, some rough endoplasmic reticulum, and free ribosomes. The extrusion apparatus consists of a polar tubule that lies coiled inside the spore and is attached to an anchoring disk. The tubule is averted when triggered by appropriate environmental stimuli, e.g., small-intestinal fluid, and is capable of penetrating a host cell to inoculate the sporoplasm into the host cell cytoplasm [9,10]. The life cycle of microsporidia includes three distinct phases: first, the infective phase, i.e., the spore stage, stimulation of the spore necessary to trigger the extrusion of the polar tubule, and inoculation of infective spore content (termed sporoplasm) into a host cell; second, the proliferative vegetative phase, termed merogony (schizogony), during which the parasites multiply intracellularly; and third, the intracellular sporogony, during which infective spores are formed [11,12].

3. Epidemiology

The epidemiology of human microsporidiosis may differ according to host immune status and the infecting species of microsporidia [13,14]. With antigens obtained from cultures of murine-derived strains of *Encephalitozoon cuniculi*, serologic surveys for human antibodies to *Encephalitozoon cuniculi* performed in the 1980s suggested that travellers and residents in tropical countries may have increased exposure to this organism, but clinical correlation and definitive epidemiologic data were lacking [15,16]. In humans, different host-parasite interactions may be observed depending on the microsporidial species and the competence of

the immune response. In immunocompetent and otherwise healthy persons, acute intestinal, self-limiting microsporidiosis may occur [17], but systemic microsporidiosis has not been satisfactorily documented in a previously healthy person (**Table 1**).

Table 1: Reports on Microsporidiasis infection in Humans

Microsporidial species	Detection of parasite	Clinical Manifestation	Reference No.
Nosema connori	Autopsy	Disseminated infection	Margileth *et al*, (1973) [18]
Pleistophora spp.	Histological examination	Myositis	Ledford *et al*, (1985) [19]
Enterocytozoon bieneusi	Stool specimen	Diarrheoa	Sandfort *et al*, (In press) [20]
Nosema corneum	Histological examination	Keratitis	Arison *et al*, (1966) [21]
Encephalitozoon cuniculi	Cerebrospinal fluid	Seizures	Matsubayashi *et al*,(1959) [3]

4. Conclusion

Microsporidia have also successfully adapted to the mammalian host. The long-term evolutionary host-parasite interactions have resulted in a "well-balanced" relationship and generally low pathogenicity of the parasite, manifesting in dormant or mildly symptomatic infection in mammals. Current data suggest that microsporidia are important pathogens capable of causing opportunistic infections in strictly immunodeficient HIV-infected patients. Sero-epidemiologic surveys have provided confirmation for the occurrence of latent microsporidial infection in healthy persons. Human microsporidiosis appears to be adventitious and chiefly related with an increasing centre of population of immune-deficient individuals. Altogether, a strong confirmation exists for an increasing commonness of microsporidiosis in animals and humans. Proper care has to be taken to further check its spread to HIV-infected persons. Awareness programmes should be launched to check its severe and large scale spread. More research has to be carrying out to further establish immunopathological Biochemistry, Microbiology and Molecular Biology.

5. References

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