

Opportunistic Parasitism: Parasitic Association with the Host that has Compromised Immune System

Fikresilasie Samuel

Department of Medicine, Ambo University, Ambo, Ethiopia

*Corresponding author: Fikresilasie Samuel, Department of Medicine, Ambo University, Ambo, Ethiopia, Tel: +251929050512; E-mail: fikre16sam@gmail.com

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Abstract

A symbiotic association between opportunistic parasites and immunocompromised hosts is known as opportunistic parasitism. The abnormality of immunity is caused by different factors: Aging enhances susceptibility to severe infections; malnutrition has significant depressing consequences for immune function; an immunosuppressant substance alcohol can interfere with the functions of many of the cells and molecules those are components of the immune system; infectious pathogens are also major causal agents of immunosuppression. Combined effect of these factors makes the host to be susceptible for opportunistic parasitic infections. In this review the promising factors which induce immune disorder and exemplary opportunistic parasitic infections which illustrate the real sense of opportunistic parasitism are well discussed.

Keywords: Opportunistic parasite; Immunity; Host; Parasitism

Introduction

Opportunistic parasitism can be defined as a symbiotic association between opportunistic parasites and immunocompromised hosts; so that the parasite causes opportunistic parasitic infection (OPI). OPI occur enthusiastically with organisms that are recognized pathogens, but are commonly caused by commensals or other normally non-pathogenic agents when host resistances are weakened by different environmental or natural factors [1].

OPI may not cause severe pathological changes in immunocompetent hosts as long as the immune system is functioning normally [2]. However when the immune system is weakened due to particular conditions, opportunistic parasites take this advantage to initiate an infection [3]. Many people have impaired immune systems as a result of many factors: diseases (like HIV infection or malignant diseases), medical procedures (such as organ transplants) [2], aging, chemotherapy and others. For these reasons, the system acquires different defects (humeral or cell mediated). This will establish a favorable condition for opportunistic parasites to flourish over the host system and cause a disease [4].

Review

The normal immune system task is depending on the interaction of enormous variety of cells and molecules. Which may involve many different mechanisms some nonspecific (i.e., generically applicable to many different pathogenic organisms) and others specific (i.e., their protective effect is directed to one single organism) to protect our body from infectious agents. Nevertheless, multiplicity of different defects as a result of diverse factors can diminish its competence badly; these deficiencies put in the same end- result, increased susceptibility to infection [5,6]. This review discusses the basic concept of opportunistic parasitism and provides a better definition of this association via explaining major factors which induce immune system impairment. As

well, three opportunistic parasitic infections are well discussed to exemplify others.

Major Risk Factors and Immune System Impairment

Age and stress

Resistance to the disease tends to be weaker in childhood and old age [7]. Aging enhances susceptibility to severe infections, which is related with a depression of cellular immunity. It has direct consequence on development of frequent and rigorous infection, which further increases morbidity, dependence and fatality in elderly population [8]. In addition to aging, the immuno-suppressive effect of stress contributes to the immune defects that give rise to the prevalence of infectious illnesses in older adults [9]. Studies on stress in older adults provide evidence that increased stress exposure, poor stress buffering, exaggerated stress reactivity, unlimited anxiety duration, and probably diminished restorative processes have effects that mimic, aggravate, and sometimes speed up the effects of aging on immunity [9].

Malnutrition

Nutrition has been clearly identified as a key factor for human development, not only as a conditioning factor for health but also as a determinant of quality of life throughout the lifecycle and of overall development. Specially, in order to function competently our immune system needs nourishment [10]. Calorie, protein, vitamins and minerals (such as iron, copper and zinc) intakes are very important [7].

The word 'malnutrition' expresses any disorder resulting from an insufficient or unbalanced diet, or a malfunctioning to absorb or assimilate nutritional elements. It could be protein-energy malnutrition or micronutrient deficiencies [10]. Usually, poor protein intake and insufficient amount of vitamins and minerals in the diet have significant depressing consequences for immune function [11]. All these conditions are associated with significant impairments of

cell-mediated immunity, antibody concentrations, phagocyte function, complement system, and cytokine production [10,12].

In conclusion, there is cyclical relationship between malnutrition and immunity. Poor nourishment consequences weight loss, growth faltering, lowered immunity and mucosal damage. These increase vulnerability of the host to communicable diseases and lead to immunological impairments. As a result, metabolic responses change nutritional status of the individual through loss of appetite, malabsorption and altered metabolism [13].

Alcohol

Alcohol is an immunosuppressant substance, which affects both cell mediated and humeral immunity, as does severe malnutrition [14]. It interferes with the functions of many of the cells and molecules those are components of the immune system. For example, alcohol slows down the functions of Phagocytes (i.e., neutrophils, Monocytes, and macrophages). It also changes the production of signaling molecules that facilitate to organize the immune response (i.e., cytokines) [15]. The dysfunction of the immune system makes the patient susceptible to enormous collection of infectious pathogens, resulting in biomedical consequences such as increased risk of infections after surgery, traumatic injury, or burns; of liver disease; of opportunistic infections in the lungs; and of accelerated development of HIV disease [14,16].

Infection and drug

Infectious pathogens are major causal agents of immunosuppression. Specially, acquired immunosuppression due to pathogens is primarily caused by viruses that invade the cellular compartment of the immune system [7]. For illustration, viral infection of cells naturally leads to the secretion of interferon alpha (IFN α) by that cell. It is protective for cells in the local setting. On the contrary, IFN α inhibits the G1 phase of the cell cycle, producing a momentary immunosuppression since cells responding to IFN α cannot clonally enlarge. In addition to immunosuppression induced by IFN α , the HIV itself induces immunosuppression in a number of ways, the destruction of CD4⁺ T cells, cells necessary for almost every part of immunity. Other viruses, such as measles, can too temporarily depress T cell function [7].

Besides to infections, drugs recommended for treatment contribute to impair the immune system. These cause immunodeficiency, either purposely or accidentally [7]. For example, cancerous diseases are often treated by administration of cytotoxic drugs intended to kill the tumor cells. These drugs are not cancer cell-specific and other proliferating cells are also destroyed. Cytotoxicity to immune cells causes nonspecific immunosuppression. In addition, to combat the risk of graft rejection, transplant patients are intentionally given immunosuppressive drugs, many of which inhibit the secretion of cytokines that enhance immune responses. These patients are thus at risk of the same infectious disorders seen in other immune defects [5].

Exemplary Opportunistic Parasitic Infections

Cryptosporidiosis

Species of the genus *Cryptosporidium* are protozoan parasites (Apicomplexa) that cause gastroenteritis in animals and humans. Of these *Cryptosporidium parvum* and *Cryptosporidium hominis* are the major causative agents of human cryptosporidiosis [17].

Cryptosporidium spp are well recognized as causes of diarrheal disease during waterborne epidemics and in immunocompromised hosts [18].

The degree of the disease is mostly dependent on the immune status of the host, whether the infection is self-limiting or continual. It is well known that, both branches of the immune system are required for complete improvement of the disease [19]. The studies on the mechanisms of immunity to cryptosporidiosis pointed out that, the spectrum and severity of disease in immunocompromised individuals with cryptosporidiosis reflect the importance of the T-cell response since the most severe disease is seen in individuals with defects in the T-cell response [20].

As a result, immunocompromised adults and children, especially those with AIDS, children in day care, travelers to endemic regions, dairy or cattle farm workers of their families or contacts, household contacts of cases or carriers, and possibly owners of infected dogs or cats or their neighbors are at greatest risk [21]. As well, individuals with neoplasm, severe combined immunodeficiency syndrome and acute leukemia (most commonly in children) are associated with increased risk of severe cryptosporidiosis disease [20]. To conclude, immunodeficiency increased the risk of having opportunistic parasites and diarrhea. In the immunocompromised host, infection is prolonged, sometimes asymptomatic, but may result in chronic debilitating diarrhea with dehydration, malabsorption, and wasting [22].

Public health measures to reduce contamination of water supplies and vigilant surveillance will reduce the risk to populations [23]. Besides, the widespread use of antiretroviral therapy does appear to be having a beneficial effect on recovery from cryptosporidiosis and on the frequency of infection in human immunodeficiency virus-positive patients. Therefore, increasing patient immune status and screening at least for those treatable parasites is important [24].

Toxoplasmosis

Toxoplasmosis is an opportunistic parasitic infection caused by obligate intracellular protozoan parasite *Toxoplasma gondii*, a parasite belonging to the phylum Apicomplexa. The disease is primarily transmitted by ingesting undercooked or raw meat containing tissue cysts, or by ingesting food or water contaminated with oocysts [6]. *Toxoplasma* invades any cell by an active invasion process involving motility and molecular secretion. Its success as an invasive organism resides on its high trans-epithelial migration capability reaching privileged organs such as brain, eye and placenta in pregnant women. Infection in pregnant women may lead to abortion, stillbirth or other serious consequences in newborns [25]. Toxoplasmosis affects up to one-third of the global human population. Sero-prevalence studies in different communities of Ethiopia showed that the general prevalence of the disease is about 80% in the adult population [6]. And up to 41% in children aged 1-5 years [26].

Immunosuppression due to treatment after transplantation or related with HIV infection raises exposure to numerous opportunistic infections, counting toxoplasmosis. The disease central nervous system (CNS) toxoplasmosis affects the brain and occurs almost exclusively due to reactivation of latent infection [27]. It is very important opportunistic infection in AIDS patients. Abnormal cytokine profile and impaired cytotoxic T cell functionality are responsible for easy susceptibility to this infection [28]. The lung is also a major site of infection after the CNS toxoplasmosis in immunocompromised peoples. AIDS patients having CD4⁺ T cell count <100 cells/ μ l of blood

can have reactivation from asymptomatic latent infection. However, most vulnerable patients are those having CD4⁺ T cell count <50 cells/ μ l of blood [28].

In transplant recipients toxoplasmosis is common and clearly stated in different studies. It occurs by reactivation of latent infection or is a primary infection if a donor organ containing encysted *T. gondii* was transplanted into a seronegative recipient [29]. In addition, studies in Korea have announced that toxoplasmosis is a rare but can be fatal complication in hematopoietic stem cell transplant recipients, usually associated with allogeneic hematopoietic stem cell transplantation (HSCT) [30].

Educating the public about the risks associated with unhealthy food and life style habits, tracking serological examinations to special populations and measures to strengthen food and occupational safety are recommended to avoid toxoplasmosis [25]. Finally, ART treatment lowers the incidence of toxoplasmosis in HIV patients [28].

Strongyloidiasis

Strongyloidiasis is an infection caused by an intestinal nematode, *Strongyloides stercoralis*, which has a cosmopolitan distribution in tropical and subtropical regions. Infection is via trans-cutaneous by filariform larvae. This usually leads to cutaneous, gastrointestinal, or pulmonary symptoms depending on the host immune status [31]. It is endemic world-wide, yet more prevalent in hot and humid climates as well as resource poor countries with inadequate sanitary conditions [32].

S. stercoralis is unique among intestinal nematodes in its ability to complete its life cycle within the host through an asexual auto-infective cycle, allowing the infection to persist in the host indefinitely. Under some conditions associated with immunocompromised, this autoinfective cycle can become amplified into a potentially fatal hyperinfection syndrome, characterized by gastrointestinal bleeding and respiratory distress [33]. It makes *S. stercoralis* opportunistic parasite, which occurs frequently in immunocompromised patients, especially in those with a defect in cell-mediated immunity [34].

A variety of underlying conditions appear to predispose to severe infections. Including immunodeficiency due to defective T-lymphocyte function, therapeutic regimens consisting of corticosteroids or other immunosuppressive medication, and chronic illness or malnutrition, predispose to systemic strongyloidiasis. Studies suggested that this infection incorporate unexplained gram-negative bacillary bacteremia in a compromised host [32].

Measurement like screening for Strongyloides infection before the initiation of immunosuppressive therapy should be considered in patients with unexplained eosinophilia, serpiginous skin lesions, or pulmonary or gastrointestinal symptoms [31,35]. Furthermore, transplantation centers drawing patients from areas with endemic Strongyloides should evaluate potential recipients closely for occult strongyloides infection prior to initiating immunosuppressive therapy [34]. To conclude, after immunocompromised patients develop hyperinfection syndrome, diagnosis is often delayed and mortality is high, as a result emphasis should be placed on testing and treatment prior to transplantation

Conclusion

OPIs are well flourished in immunocompromised individuals, whose immune system has defects to function normally. There are

factors which provoke the human immune system to be impaired. Such as: age, infection, alcohol etc. So that opportunistic organisms use the advantage to initiate an infection.

References

1. Peter JD, Ivan MR (1998) Encyclopedia of immunology (2nd edn). Volume 1.
2. Zope A, Pai A, De A, Baveja SM (2014) Opportunistic Intestinal Parasites in HIV Infected Individuals and Its Correlation with the Cd4 Counts. Research and Reviews: Journal of Medical and Health Sciences.
3. Kashyap A, Singh MP, Ghoshal U (2013) Occurrence of Gastrointestinal Opportunistic Parasites in Immunocompromised Patients in Northern India. Journal of Biology 1: 77-80.
4. FMOH (2008) Guidelines for management of opportunistic infections and Anti-Retroviral Treatment in adolescents and adults in Ethiopia. Addis Ababa, Ethiopia.
5. Gorczynski R, Stanley J (1999) Clinical immunology. Landes Bioscience, Texas.
6. Eales LJ (2003) Immunology for Life Scientists. John Wiley & Sons Ltd, England.
7. Playfair JHL, Chain BM (2001) Immunology at a glance. Blackwell Publishing, USA.
8. Dey AB, Chatterjee P, Das PC (2012) Immune Status in the Elderly. Medicine Update.
9. Hawkey LC, Cacioppo JT (2004) Stress and the aging immune system. Brain Behav Immun 18: 114-119.
10. Duggal S, Chugh TD, Duggal AK (2012) HIV and malnutrition: effects on immune system. Clin Dev Immunol 2012: 784740.
11. Sicotte M, Langlois EV, Aho J, Ziegler D, Zunzunegui, MV (2014) Association between nutritional status and the immune response in HIV + patients under HAART: protocol for a systematic review. Systematic Reviews 3: 9.
12. Huhhes S, Kelly P (2006) Interaction of malnutrition and immune impairment, with specific reference to immunity against parasites. Parasite immunology 28: 577-588.
13. Keusch GT (2003) The history of nutrition: malnutrition, infection and immunity. J Nutr 133: 336S-340S.
14. Molina PE, Happel KI, Zhang P, Kolls JK, Nelson S (2010) Focus on: Alcohol and the immune system. Alcohol Res Health 33: 97-108.
15. Szabo G (1997) Alcohol's contribution to compromised immunity. Alcohol Health Res World 21: 30-41.
16. Greenfield TK, Ye Y, Bond J, Kerr WC, Nayak MB, et al. (2014) Risks of alcohol use disorders related to drinking patterns in the U.S. general population. J Stud Alcohol Drugs 75: 319-327.
17. Rossle NE, Latif B (2013) Cryptosporidiosis as threatening health problem: A review. Asian Pac J Trop Biomed 3: 916-924.
18. Checkley W, White AC Jr, Jaganath D, Arrowood MJ, Chalmers RM, et al. (2015) A review of the global burden, novel diagnostics, therapeutics, and vaccine targets for cryptosporidium. Lancet Infect Dis 15: 85-94.
19. McNair NN, Mead JR (2013) CD4⁺ effector and memory cell populations protect against Cryptosporidium parvum infection. Microbes Infect 15: 599-606.
20. Hunter PR, Nichols G (2002) Epidemiology and clinical features of Cryptosporidium infection in immunocompromised patients. Clin Microbiol Rev 15: 145-154.
21. Desai NT, Sarkar R, Kang G (2012) Cryptosporidiosis: An under-recognized public health problem. Trop Parasitol 2: 91-98.
22. Mead JR (2014) Prospects for immunotherapy and vaccines against Cryptosporidium. Hum Vaccin Immunother 10: 1505-1513.
23. Peletz R, Mahin T, Elliott M, Montgomery M, Clasen T (2013) Preventing cryptosporidiosis: the need for safe drinking water. Bull World Health Organ 91: 238.

24. Marcos LA, Gotuzzo E (2013) Intestinal protozoan infections in the immunocompromised host. *Curr Opin Infect Dis* 26: 295-301.
25. Zhou P, Chen Z, Li HL, Zheng H, He S, et al. (2011) *Toxoplasma gondii* infection in humans in China. *Parasit Vectors* 4: 165.
26. Dubey JP, Tiao N, Gebreyes WA, Jones JL (2012) A Review of Toxoplasmosis in Humans and Animals in Ethiopia. *Epidemiol Infect* 140: 1935-1938.
27. Halonen SK (2004) Immune Response to *Toxoplasma Gondii* in the Central Nervous System. *World Class Parasites* 9: 67-88.
28. Sandhu A, Samra AK (2013) Opportunistic infections and disease implications in HIV/AIDS. *International Journal of Pharmaceutical Science Invention* 2: 47-54.
29. Ahmadpour E, Daryani A, Sharif M, Sarvi S, Aarabi M, et al. (2014) Toxoplasmosis in immunocompromised patients in Iran: a systematic review and meta-analysis. *J Infect Dev Ctries*. 8: 1503-1510.
30. Kim KH, Song KH, Jeon JH, Park WB, Park SW, et al. (2010) A Case of Cerebral Toxoplasmosis Following Tandem Autologous Stem Cell Transplantation in a Multiple Myeloma Patient. *Infect Chemother* 42: 181-186.
31. Ardiç N (2009) An overview of *Strongyloides stercoralis* and its infections. *Mikrobiyol Bul* 43: 169-177.
32. Schar F, Trostorf U, Giardina F, Khieu V, Muth S, et al. (2013) *Strongyloides stercoralis*: Global Distribution and Risk Factors. *PLoS Negl Trop Dis* 7: e2288.
33. Keiser PB, Nutman TB (2004) *Strongyloides stercoralis* in the Immunocompromised Population. *Clin Microbiol Rev* 17: 208-217.
34. Roxby AC, Gottlieb GS, Limaye AP (2009) Strongyloidiasis in transplant patients. *Clin Infect Dis* 49: 1411-1423.
35. Lam CS, Tong MK, Chan KM, Siu YP (2006) Disseminated strongyloidiasis: a retrospective study of clinical course and outcome. *Eur J Clin Microbiol Infect Dis* 25: 14-18.