AIDS/HIV

General Description

• Acquired immune deficiency syndrome (AIDS) is a disease that acts by lowering the victim’s immunity, allowing secondary, unrelated infections to produce fatal symptoms.
• In 1983, researchers in France and the United States isolated the virus that produces AIDS. This virus is known as the human immunodeficiency virus (HIV).

Causes

• HIV causes depression in immunity by primarily affecting T-cells, which are responsible for the body’s ability to defend itself against foreign organisms (acquired immunity).
• HIV appears to preferentially target helper T cells over other cells in the body, although it has been shown to also be capable of attacking other T cells and monocytes as well. Once it enters the body, it initially infects helper T cells, causing flu-like symptoms that last for several weeks. The virus then enters a dormant period which can range from 6 months to 10 years. Following this dormant phase, HIV begins its reproductive phase. The virus is released and invades other helper T cells, destroying them to produce new viruses. This reproductive cycle is capable of destroying 60 - 90% of the helper T cells in the body within several months. The result of helper T-cell destruction is a suppression of cell-mediated immunity.
• Once immunity has been suppressed, infectious agents take advantage of the opportunity to cause secondary diseases. The opportunistic pathogens cause many of the diseases which are used to diagnose AIDS: pneumonia from *Pneumocystis carinii*, a type of skin cancer called Kaposi’s sarcoma, a liver disease called hepatitis B, a systemic bacterial infection know as toxoplasmosis, a loss of weight caused by chromic diarrheas known as HIV wasting syndrome, and a degeneration of functional brain tissue called AIDS dementia. These secondary diseases are the usual causes of death among AIDS sufferers.

At Risk

• Initially, AIDS was primarily confined to certain population sub-sets with high risk for interchanging blood or other HIV-infected bodily fluids. Two groups in particular, homosexual males and intravenous drug users, were at highest risk.
Today, the prevalence of HIV has made caution the by-word for all individuals exposed to blood or other bodily fluids. Stringent pre-testing and handling precautions are indicated because the ultimate symptoms produced by HIV which ultimately lead to AIDS can take several years to develop. There are potentially many individuals who are unaware that they are HIV positive.

**Prevention and Management**

- At the present time, there is no vaccine or drug to prevent AIDS. The best way to reduce the risk of HIV exposure is to reduce or eliminate exposure to potentially infected blood.
- Sexual transmission of HIV, which is the most common mode of infection, can be prevented by use of barriers such as latex condoms.
- Health care professionals are well aware of the precautions that must be taken whenever blood is handled. The best course is to assume that the blood is HIV positive, and to take appropriate measures, such as latex gloves, gowns, and eye protections, to prevent exposure.
- Advanced cases of AIDS are accompanied by a general wasting of the body (AIDS wasting syndrome). Many individuals with AIDS also have deficiencies in many vitamins and minerals.
- Nutrition and nutritional status can have profound effects on immune functions. Some AIDS sufferers have shown temporary improvement in their symptoms when put on a strict nutritional regimen which also included vitamin supplements.
- Recent evidence also suggests that HIV infected individuals are under chronic oxidative stress which may contribute to many aspects of the pathogenesis of AIDS. This has lead to the suggestion that the progression of AIDS may be retarded by supplementing natural antioxidant defenses. However, well controlled prospective clinical trials of the efficacy of antioxidants for AIDS remain to be performed.
- Cognitive and hematopoietic dysfunctions of some AIDS patients is reversed by vitamin B-12 therapy.

**Abstracts**


Acquired immune deficiency syndrome (AIDS) is a clinical disorder caused by a retrovirus infection and represents the end point in a progressive sequence of immunosuppressive changes. Vitamins can enhance disease resistance in animals and humans. As such they are important co-factors in optimal functioning of the immune systems. In this article, the immunological and nutritional modifications caused by AIDS are summarized. The effects of murine and human retrovirus infection on vitamin status are analyzed as co-factors in the development of severe immune dysfunction, AIDS. The properties of immunoenhancing antioxidative vitamins, vitamin A, B6, B12, C, E, and beta-carotene, which are frequently low in AIDS patients, are evaluated relative to the development of immunodeficiency during retrovirus infection. Vitamin A, E, and B12 deficiency accelerated the development of AIDS with low T cells, whereas their normalization retarded the development of immune dysfunction. The interactions between these vitamins and the immune system in human AIDS patients and animal models of AIDS are reviewed. Our purpose is to provide data on how retrovirus infection can cause nutritional deficiencies that accentuate immune damage and to evaluate the potential therapeutic role of vitamins in the treatment of immune dysfunctions in AIDS patients.
Pace GW, Leaf CD. The role of oxidative stress in HIV disease. Free Radic Biol Med 1995 19(4):523-8. Evidence has accumulated suggesting that HIV-infected patients are under chronic oxidative stress. Perturbations to the antioxidant defense system, including changes in levels of ascorbic acid, tocopherols, carotenoids, selenium, superoxide dismutase, and glutathione, have been observed in various tissues of these patients. Elevated serum levels of hydroperoxides and malondialdehyde also have been noted and are indicative of oxidative stress during HIV infection. Indications of oxidative stress are observed in asymptomatic HIV-infected patients early in the course of the disease. Oxidative stress may contribute to several aspects of HIV disease pathogenesis, including viral replication, inflammatory response, decreased immune cell proliferation, loss of immune function, apoptosis, chronic weight loss, and increased sensitivity to drug toxicities. Glutathione may play a role in these processes, and thus, agents that replete glutathione may offer a promising treatment for HIV-infected patients. Clinical studies are underway to evaluate the efficacy of the glutathione-repleting agents, L-2-oxothiazolidine-4-carboxylic acid (OTC) and N-acetylcysteine (NAC), in HIV-infected patients.

References