Tropical Spastic Paraparesis

For several decades the term “tropical spastic paraparesis” (TSP) was used to describe a chronic and progressive clinical syndrome that affected adults living in equatorial areas of the world. This condition was initially thought to be associated with infectious agents (such as *Treponema pertenue* and *Treponema pallidum* which cause inflammation of the central nervous system) and with chronic nutritional deficiencies (such as *avitaminosis*) or exposure to potentially toxic foods (such as *bitter cassava*). Neurological and modern neuroepidemiological studies found that in some individuals no one cause could explain the progressive weakness, sensory disturbance, and sphincter dysfunction that affected individuals with TSP. In spite of public health programs created to eradicate the above-mentioned infectious and nutritional conditions in the tropics, large numbers of people continued to be affected. During the mid-1980's, an important association was established between the first human retrovirus-human T-cell lymphotrophic virus type 1 (also known as HTLV-1)-and idiopathic TSP (idiopathic means of unknown origin). Since then, this condition has been named *HTLV-1 associated myelopathy/ tropical spastic paraparesis* or *HAM/TSP* and scientists now understand that it is a condition caused by a virus that results in immune dysfunction. Patients with HAM/TSP may also exhibit uveitis (inflammation of the uveal tract of the eye), arthritis (inflammation of one or more joints), pulmonary lymphocytic alveolitis (inflammation of the lung tissues), polymyositis (an inflammatory muscle disease), keratoconjunctivitis sicca (persistent dryness of the cornea and conjunctiva), and infectious dermatitis (inflammation of the skin). Co-factors that may play a role in transmitting the disorder include being a recipient of transfusion blood products (especially before 1989), breastmilk feeding from a seropositive mother, intravenous drug use, or being the sexual partner of a seropositive individual for several years. Not every HTLV-1 seropositive carrier will become a HAM/TSP patient. Fewer than 5% will exhibit neurological dysfunction or, eventually, hematological malignancy such as adult T-cell leukemia or lymphoma.

TREATMENT

There is no established treatment program for HAM/TSP although some patients may be given steroids. Clinical studies using interferon alpha and plasmapheresis have not shown significant patient improvement. Spasticity may be treated with lioresal or tizanidine. Urinary dysfunction should be treated with self-catheterization or oxybutynin. A current trial with interferon beta 1a is underway.

WHAT WILL HAPPEN

HAM/TSP is usually a progressive neurological disorder but it is rarely fatal. Most patients live for several decades after the diagnosis. Their prognosis improves if they take steps to prevent urinary tract infection and skin sore formation, and if they enroll in physical and occupational therapy programs.

RESEARCH BEING DONE

The NINDS supports and conducts an extensive research program on disorders of the nervous system. The goals of this research are to find ways to prevent, treat, and, ultimately, cure these disorders

Research is continually being done to learn more about this problem.

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