Human T-Lymphotrophic Virus Type-I: A Unique Association with Myelopathy in Sjögren’s Syndrome

Alexandria Voigt1 and Cuong Q Nguyen1,2*
1Department of Infectious Diseases and Pathology, College of Veterinary Medicine, University of Florida, 2015 SW 16th Ave, Gainesville, Florida, USA
2Center for Orphan Autoimmune Disorders, University of Florida College of Dentistry, 1600 SW Archer Rd, Gainesville, Florida, USA

Corresponding author: Cuong Q. Nguyen, PhD, Department of Infectious Diseases and Pathology, PO Box 110880, College of Veterinary Medicine, University of Florida, Gainesville, Florida 32611-0880 USA, Tel: 352-294-4180; Fax: 352-392-9704; E-mail: Nguyenc@ufl.edu

Rec date: Dec 15, 2014; Acc date: Dec 25, 2014; Pub date: Jan 05, 2015

Copyright: © 2014 Nguyen Q, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Editorial

Microbes play the most intricate and crucial role in the education of the immune system. Exposure to pathogens elicits specific immune response to clear the infection. After elimination of infectious pathogens, the immune cells undergo apoptosis during the contraction phase, and the remaining cells return to the quiescent memory phase [1]. During the primary and secondary responses, some of the immune cells fail to distinguish self and non-self. The failure to discriminate self and non-self entities triggers autoimmunity [2]. For decades, pathogens, the immune cells undergo apoptosis during the contraction phase, and the remaining cells return to the quiescent memory phase [1]. During the primary and secondary responses, some of the immune cells fail to distinguish self and non-self. The failure to discriminate self and non-self entities triggers autoimmunity [2]. For decades, pathogens, the immune cells undergo apoptosis during the contraction phase, and the remaining cells return to the quiescent memory phase [1]. During the primary and secondary responses, some of the immune cells fail to distinguish self and non-self. The failure to discriminate self and non-self entities triggers autoimmunity [2]. For decades, pathogens, the immune cells undergo apoptosis during the contraction phase, and the remaining cells return to the quiescent memory phase [1]. During the primary and secondary responses, some of the immune cells fail to distinguish self and non-self. The failure to discriminate self and non-self entities triggers autoimmunity [2]. For decades, pathogens, the immune cells undergo apoptosis during the contraction phase, and the remaining cells return to the quiescent memory phase [1]. During the primary and secondary responses, some of the immune cells fail to distinguish self and non-self. The failure to discriminate self and non-self entities triggers autoimmunity [2].

In cases of infection, onset of myelopathy has been acute with patients feeling neck pain followed by parathesia, numbness or pain in their extremities which in some cases is followed by paraplegia [13]. There is debate as to whether HTLV-1 infection can act as an environmental trigger to cause SJSS, however it remains unclear. Patients may not actually know that they have SJSS until they seek out medical attention for these symptoms and patients with SJSS can develop myelopathy without HTLV-1 infection. Ironically, patients with SJSS would not usually be tested for HTLV-1 unless myelopathy occurred; some patients with SJSS test negative for anti-SSA or anti-SSB autoantibodies and may lead to a negative diagnosis when SJSS is present.

In one study [11], of ten patients presenting with HTLV-1 associated myelopathy (HAM), six were diagnosed with SJSS and two were suspected to have it. Since all of the HAM patients also showed lymphocytic infiltration in the salivary glands (whether anti-SSA or anti-SSB autoantibodies were present), it becomes difficult to diagnose SJSS as that is one of the qualifying criteria. Either salivary gland infiltration is a symptom of HTLV-1 associated exclusively to HAM, or SJSS patients exclusively exhibit HAM. Further examination and larger cohorts of patients would be necessary to confirm which of these two options is correct.

Use of animal models is another viable option for studying this relationship. In one transgenic rat model (HTLV-1 env-pX), the env-pX gene was inserted into the WKAH strain [14] and rats developed SJSS-like symptoms along with an array of other symptoms characteristic of autoimmune diseases [15], but manifestation of myelopathy was not studied. Further examination of this line, focusing on myelopathy exclusively may yield an answer as to whether or not HAM is present only in SJSS patients, but this may be difficult as they are prone to developing other autoimmune disorders. Complexing SJSS with another autoimmune disorder may convolute the findings as they may either exacerbate or mask symptoms.

To further understand HAM moving forward, it will probably become necessary to correctly diagnose patients with or without SJSS. For HAM in SJSS patients combined treatment with a corticosteroid and anti-viral has shown improvement in the patients’ condition without resolving the lesion [16]; reversal of parathesia, numbness, pain or paraplegia may occur. Further examination of HAM both in a clinical setting and utilizing animal models may be necessary to fully understand if and why HAM presents exclusively in SJSS patients.

References


