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Cardiovascular risk profile in patients with myelopathy associated with HTLV-1☆

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ABSTRACT

HAM/TSP (HTLV-1-associated myelopathy/tropical spastic paraparesis) is a slowly progressive disease, characterized by a chronic spastic paraparesis. It is not known if the disease carries an independent risk for cardiovascular disease. The objective of this study was to evaluate the cardiovascular risk profile related to HAM/TSP and compare it with the general population.

Methods: This was a cross-sectional study, with a control group. HAM/TSP patients were evaluated using cardiovascular risk scores (ASCVD RISK, SCORE and Framingham) and inflammatory markers (ultrasensitive CRP and IL-6), and compared with a control group of healthy individuals. We also evaluated the correlation between cardiovascular risk and the functional status of patients with HAM/TSP evaluated by the FIM scale.

Results: Eighty percent of patients in this study were females, mean age of 51 years (11.3). The control group showed an increased cardiovascular event risk in 10 years when ASCVD was analyzed (cardiovascular risk $\geq$7.5% in 10 years seen in 43% of patients in the control group vs. 23% of patients with HAM/TSP; $p = 0.037$). There was no difference in ultrasensitive CRP or IL-6 values between the groups, even when groups were stratified into low and high risk. There was no correlation between the functional status of HAM/TSP patients and the cardiovascular risk.

Conclusions: In this study, the cardiovascular risk profile of patients with HAM/TSP was better than the risk of the control group.

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Introduction

Human T-lymphotropic virus type 1 (HTLV-1) was the first known human retrovirus, described in 1979 by Poiesz.[1] It is estimated that 15–20 million people are affected worldwide.[2] The incidence of HTLV-1 infection is higher in Japan, Africa, and Caribbean Islands, where it may affect 5% or more of the population.[3] The virus was initially associated with adult T-cell leukemia; subsequently, myelopathy (HTLV-1-associated myelopathy/tropical spastic paraparesis – HAM/TSP), uveitis, and infective dermatitis were also associated with the virus.[4–7] Only 5–10% of patients, however, develop clinical manifestations.

HAM/TSP is a slowly progressive disease. It is characterized by a chronic spastic paraparesis, neurogenic bladder, neurogenic bowel, spasticity and neuropathic pain. Because these symptoms are similar to those found in patients with other spinal cord diseases (traumatic or non-traumatic), their treatment overlaps, consisting mostly of supportive measures and rehabilitation. There is currently no antiviral treatment for the disease. Once HAM/TSP develops, it is usually progressive and irreversible.

In the past, pulmonary and renal complications were the main causes of morbidity and mortality of patients with traumatic spinal cord injury.[8,9] However, cardiovascular disease is now one of the leading causes of death.[8,10,11] Risk factors such as diabetes, low HDL-cholesterol (HDL-C), sedentary life-style, and smoking have a higher prevalence among patients with spinal cord injury.[8,10,12–14]

Few studies describe the cardiovascular involvement related to HTLV-1. In 2014, Layegh et al.,[15] in a cross-sectional study assessed atherosclerosis in patients with HTLV-1. The authors found that HTLV -1 infected patients had greater carotid intima-media thickness than age-mached healthy controls. In 1996, Stuver et al. found increased incidence of cardiovascular diseases and EKG changes in patients infected with HTLV-1.[16] In 2013, Shabestari et al. found increased prevalence of HTLV-1 among patients undergoing angiography.[17]

The Human Immunodeficiency Virus (HIV), another retrovirus, is associated to increased cardiovascular risk. Freinberg et al. studied 82,459 patients with HIV during 5.9 years. After adjusting for comorbidities and cardiovascular risk scores, they found that HIV was an independent cardiovascular risk factor, raising the risk of a myocardial infarction by 50%. The mechanism by which HIV increases the risk of myocardial infarction is not known; it is thought that viral induced inflammation and endothelial dysfunction may play a role.[18] Anti-retroviral therapies, especially protease inhibitors are also related to higher cardiovascular risk.[19] As HIV and HTLV-1 are both retrovirus and share similar replication enzymes such as retroviral proteases,[20] one could hypothesize that HTLV-1 infection could also be an independent cardiovascular risk factor.

The objective of the present study was to evaluate the cardiovascular risk profile of patients with HAM/TSP and compare it with the general population.

Methods

Study design

This was a cross-sectional study, with a control group.

Patients and settings

The study group was comprised of HAM/TSP patients followed in a rehabilitation Hospital in the city of Salvador, Brazil from July 2014 to October 2015. All patients had the diagnosis of HAM/TSP defined according to the Castro–Costa criteria.[21] Patients with other possible causes for spinal cord lesions were excluded. Patients with possible causes for changes in inflammatory tests such as acute infections, rheumatologic diseases, inflammatory bowel disease, and use of corticosteroids or nonsteroidal anti-inflammatory drugs were also excluded.

Mid level employees of Hospital Sarah Salvador constituted the control group. They were paired in a 1:1 proportion, matching for sex and age ±5 years.

Socio-demographic data, physical examination and laboratory tests

Data were collected during individualized interviews, using a standardized questionnaire. Socio-demographic data and information about HAM/TSP disease (such as duration of the disease and comorbidities associated to the myelopathy) were collected.

Weight was obtained in a calibrated scale with patients using light clothing. For walking patients and in the control group, height was obtained using the metric measurement bar attached to an anthropometric scale. In wheelchair bound patients, height was obtained with the patient in the supine position, using a flexible inelastic tape, measuring segments of the body from the heel to head. Body mass index (BMI) was calculated by the Quetelet formula (weight/height^2).

An inelastic flexible tape was used to measure waist circumference. The measurement was obtained with the patient standing upright, with arms outstretched, after a normal expiration, at the midpoint between the last rib and the anterior superior iliac crest.[22] Wheelchair bound patients were assisted to a standing position with the use of a walker, parallel bars or a standing table for this measurement, whenever possible. The waist circumference was not measured if a standing position was not attainable. Two measurements were performed, and the average of the values was recorded.

Blood pressure was measured with aneroid sphygmomanometer with patients seated with supported upper extremity at the heart level. Patients were at rest for at least 10 min and had not smoked or drank coffee 30 min prior to the measurement.[23] Patients with neurogenic bladder underwent bladder catheterization before blood pressure measurement. Two measurements were performed, the second one minute after the first, and the average of the values was recorded.[23]