Case Report

HTLV Infection and Strongyloidal Hyperinfection Syndrome

Shobha M Itolikar*, Santosh B Salagre**, Sunil Kuyare***, Shailesh Bamburde****,

Abstract
Strongyloides stercoralis can affect humans in the form of asymptomatic infections, Strongyloidal hyperinfection syndrome and disseminated Strongyloidiasis depending on the immune response of the host. We report a case of Strongyloidal Hyperinfection syndrome that subsequently tested positive for HTLV infection.

Background
Before the advent of the HIV/AIDS era in 1983, the only known retroviruses were Human T-cell Lymphocytotropic Virus 1 and 2 (HTLV 1 and 2). Though these viruses have been found to cause multiple diseases, due to the low replicating nature and lower seroprevalence, they are rarely suspected. This article describes the case of a young boy with Strongyloidal hyperinfection syndrome who was subsequently detected to have HTLV infection in the absence of other associated diseases.

Case Report
A twelve year old boy presented to us with symptoms of easy fatigability, loss of appetite and pedal oedema since one month. He did not report any symptoms of abdominal pain, blood loss in stools, urinary complaints. He had stable vital parameters at presentation. He was markedly pale and had anasarca. His systemic examination was unremarkable except for a haemic murmur in the pulmonary area.

Further workup for microcytic hypochromic anemia revealed the presence of several rhabditiform larvae of the intestinal nematode Strongyloides stercoralis in three consecutive stool samples as shown below.

Since his stool samples showed severe infestation with strongyloid larvae, we suspected an immune-deficiency state. He denied any history of immunosuppressant drug intake like steroids in the past. We sent his blood HIV ELISA. It was negative. Further testing for HTLV 1 and 2 antibodies by ELISA yielded positive results. Thus he was diagnosed with ‘Severe Anemia with Hypoproteinemia due to malabsorption syndrome secondary to Strongyloidal Hyperinfection Syndrome with HTLV co-infection’.

He was treated with blood transfusions and Ivermectin in the dose of 200 μg/kg/day orally for two days. Repeat stool sampling after one week was negative for the presence of the larval forms.

His parents were explained about the nature of the disease and the risk of development of diseases like Adult T-cell Leukemia/Lymphoma (ATL) and Tropical Spastic paraparesis(HAM/TSP) in the future. Family members have been advised to undergo screening for HTLV infection.

Discussion
T-cell lymphocytotropic virus (HTLV-1) was first isolated from a patient with Cutaneous T-cell Lymphoma in 1979. This discovery, along with that of HTLV-2 (1981), HIV (1983) and HTLV 3and4 (2005) heralded the onset of the human retrovirus

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>3.3 gm%</td>
<td>Total Protein</td>
<td>4.7 g/dl</td>
</tr>
<tr>
<td>Leucocyte count</td>
<td>4000/cumm</td>
<td>Serum Albumin</td>
<td>2.3 g/dl</td>
</tr>
<tr>
<td>Platelet Count</td>
<td>2 lac/cumm</td>
<td>Serum calcium</td>
<td>7 mg/dl</td>
</tr>
<tr>
<td>MCV</td>
<td>56 fl</td>
<td>S.Alk phos</td>
<td>145 IU/ml</td>
</tr>
<tr>
<td>Retic Count</td>
<td>1.5 %</td>
<td>S.Creatinine</td>
<td>1.2 mg/dl</td>
</tr>
<tr>
<td>Smear</td>
<td>RBC</td>
<td>Urinalysis</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>Microcytosis and Usg Abdomen</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypochromia.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>Normal</td>
<td>ECG</td>
<td>Normal</td>
</tr>
</tbody>
</table>

*Assistant Professor, Department of Medicine, **Associate Professor, Department of Medicine, ***Assistant Professor, Department of Microbiology, ****Postgraduate Student, Department of Medicine, Seth G.S. Medical College and K.E.M. Hospital, Parel Mumbai-400012. Received: 04.05.2011; Accepted: 06.07.2011

Fig. 1: Light microscopy view of stool sample showing numerous rhabditiform larvae of Strongyloides stercoralis.
era. Human Immunodeficiency Virus (HIV) was originally named as the HTLV-III but subsequently it was reclassified as a Lentivirus.\(^1\)

It is transmitted through sexual contact, blood transfusion\(^2\) (risk of seroconversion 40%-60%), breastfeeding (20% risk), intravenous drug use and organ transplantation.

The frequency among the population of blood donors is 0.013 in America, 0.001 in Sweden, 0.002 in Netherlands, 0.003 in Denmark, 0.004 in France, 0.007 in Italy, 0.056 in Saudi Arabia and 0.05 in Argentina.\(^3\)

**Disease Associations:**

**HTLV-I:** It is associated with the following diseases.

1. **Adult T-cell Lymphoma/Leukemia (ATL):** Four clinical subtypes have been described-acute, lymphomatous, chronic, and smoldering.
2. **HTLV-1-associated myelopathy/tropical spastic paraparesis (HAM/TSP) which is a chronic progressive non-compressive myelopathy resembling multiple sclerosis.**
3. **Others:** Uveitis, dermatitis, pneumonitis, rheumatoid arthritis, and polymyositis.

**HTLV-2:** This virus has not been consistently associated with a particular disease and in fact has been thought of as ‘a virus searching for a disease’. But there have been case reports linking HTLV-2 infection with pneumonia, bronchitis, arthritis, asthma and dermatitis.\(^4\)

**Diagnosis:** HTLV-1 and HTLV-2 infections are detected with enzyme-linked immunosorbent assay (ELISA), which then must be confirmed with Western blot, immunofluorescence assay (IFA), or polymerase chain reaction (PCR). HTLV ELISA may yield false-positive rates in areas of low prevalence.\(^5\) Thorough neurological and ophthalmologic examinations, in addition to a complete physical examination, should be performed in patients infected with HTLV.

Patients diagnosed with HTLV-1 or HTLV-2 infection should also be screened for HIV, Hepatitis B and C and syphilis and their stools samples should be examined for ova/parasites of Strongyloides stercoralis. Likewise, patients whose have intestinal / systemic disease due to Strongyloides stercoralis infection should undergo testing for HIV and if necessary, HTLV infection.

**HTLV and Strongyloidiasis:** HTLV-1 predominantly infects T cells and induces spontaneous lymphocyte proliferation and secretion of high levels cytokines. Strongyloides stercoralis patients with HTLV-1 co-infection have modified immunological responses against parasite antigens and co-infection has clinical implications for strongyloidiasis. The high production of IFN-gamma observed in patients co-infected with HTLV-1 and Strongyloides stercoralis decreases the production of IL-4, IL-5, IL-13 and IgE, molecules that participate in the host defence mechanism against helminths. Moreover, there is a decrease in the efficacy of treatment of Strongyloides stercoralis in patients co-infected with HTLV-1. Alterations in the immune response against Strongyloides stercoralis and the decrease in the efficacy of anti-parasitic drugs are responsible for the increased prevalence of Strongyloides stercoralis among HTLV-1 infected subjects and make HTLV-1 infection the most important risk factor for disseminated strongyloidiasis.\(^6\)

**Treatment:** No treatment intervention exists for acute or chronic human T-cell lymphotropic virus (HTLV) infection. Antiretroviral regimens have not shown to be effective.\(^7\)

Chemotherapy is the primary treatment approach for ATL. HTLV-1–associated myelopathy/tropical spastic paraparesis (HAM/TSP) treatment options are limited and focus on symptomatic therapy.

All patients with HTLV-1 or HTLV-2 infection should be counselled extensively on the lifelong implications of their infection and advised on avoidance of needle sharing, adherence to safe-sex practices and avoidance of breast-feeding by infected women as well as lifetime risk of developing ATL and HAM/TSP.

In conclusion, we would like to state that unusual diseases like HTLV infection should be suspected in patients presenting with infections with opportunistic pathogens like Strongyloides stercoralis, Pneumocystis jirovecii, Cytomegalovirus in the absence of HIV seropositivity and other conditions predisposing to immunocompromised state.

**Acknowledgement**

Authors are grateful to Dr. Preeti Mehta (Professor and Head, Department of Microbiology) and Dr. Avni Kotecha, (Associate Professor in Microbiology) for their valuable inputs.

**References**