HTLV-1, HIV Co-Infection

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HIV/Human T-cell Lymphotropic Virus Coinfection Revisited: Impact on AIDS Progression

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Abstract

Human T-cell lymphotropic viruses type 1 and 2 are retroviruses that share the same routes of transmission as HIV-1. Since these agents are prevalent simultaneously in different parts of the world, coinfection is a frequently reported event. However, prevalence rates of coinfection differ for distinct populations and regions of the world or for each virus, with human T-cell lymphotropic virus type 1 being more prevalent among HIV-1-infected individuals in the Southern hemisphere, while type 2 is more frequently found in the Northern hemisphere. In common, they share the tropism for T-lymphocytes, although human T-cell lymphotropic virus type 1 and HIV-1 are predominantly $CD4^+$ T-cell tropic and human T-cell lymphotropic virus type 2 preferentially infects $CD8^+$ cells.
The biological properties of HIV-1 are distinct of those found in human T-cell lymphotropic virus 1/2. This fact makes possible an in vivo interaction between these agents, when coinfecting the same patients, with potentially relevant clinical implications.
The available evidence suggests a protective role for coinfection by human T-cell lymphotropic virus type 2 on AIDS progression. This hypothesis is supported by several laboratory evidences, as well as by a number of clinical studies that found no significant interaction between human T-cell lymphotropic virus type 2 and HIV-1, or even detected a protective effect on HIV-1 disease.

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The available evidence suggests a protective role for coinfection by human T-cell lymphotropic virus type 2 on AIDS progression. This hypothesis is supported by several laboratory evidences, as well as by a number of clinical studies that found no significant interaction between human T-cell lymphotropic virus type 2 and HIV-1, or even detected a protective effect on HIV-1 disease.

On the other hand, human T-cell lymphotropic virus type 1 seems to be a significant cofactor, with a potentially important role in HIV-1 infection. Although the clinical evidence is still controversial with regard to the real impact that coinfection exerts on clinical evolution, the majority of studies suggest it is associated with a modification of the natural history of HIV-1 infection, with a faster clinical progression and a shorter survival time. The main limitation of the available data is due to methodological problems in the majority of studies, which weaken the validity of their conclusions. A common finding in coinfection by both human T-cell lymphotropic virus type 1 and 2 is the increase in CD4+ cell count, but without any additional immune benefit for patients.

Due to the limited available data, we need more, larger studies, designed to respond to the pending questions on the real significance of coinfection by these retroviruses. (AIDS Rev. 2009;11:8-16)

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HIV and HTLV-1 Coinfection: The Need to Initiate Antiretroviral Therapy

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According to the latest Department of Health and Human Services guidelines for the use of antiretroviral therapy (ART) in HIV-1-infected persons, initiation of ART is recommended for all HIV-infected persons regardless of the CD4 count. In resource-limited settings where ART is not available for all patients, treatment should be prioritized for those with the following conditions: pregnancy, CD4 count <200 cells/mm^3 or AIDS-defining illness,

for all HIV-infected persons regardless of the CD4 count. In resource-limited settings where ART is not available for all patients, treatment should be prioritized for those with the following conditions: pregnancy, CD4 count <200 cells/mm^3 or AIDS-defining illness, HIV-associated nephropathy, HIV-associated dementia, hepatitis B virus coinfection, and acute HIV infection.[1] We suggest that coinfection with human T-cell lymphotropic virus type 1 (HTLV-1) should be added to this prioritized list for the following reasons:

The predictive value of CD4 count as a marker of HIV-related immunosuppression and disease stage for persons coinfected with HIV and HTLV-1 is likely not the same as for persons infected with HIV alone. Human T-cell lymphotropic virus type 1 promotes the clonal expansion of CD4-infected T lymphocytes, causing an artificially elevated CD4 count in coinfected persons.[2] Compared to HIV-infected patients with CD4 counts greater than 200 cells/mm^3, HIV/HTLV-1-coinfected individuals with similar CD4 counts are at increased risk for developing opportunistic infections.[3,4] Thus, a high CD4 count in coinfected persons does not necessarily reflect a competent immune system.

HIV/HTLV-1 coinfection is associated with accelerated progression to AIDS and worse outcomes of HIV-related opportunistic infections.[5–7] Human T-cell lymphotropic virus type 1 induces HIV viral replication and the transition from M- to T-tropic HIV phenotype, which is often a marker of HIV disease progression.[8] In addition, compared to individuals infected with only HIV, HIV/HTLV-1-coinfected individuals have higher production of proinflammatory cytokines, most notably interleukin 2 and interferon-γ, suggesting that metabolic and cardiovascular complications mediated by chronic immune activation could be more pronounced among coin-fected individuals.[9]

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In countries with a high burden of tuberculosis, tuberculosis is more common in persons infected with both HIV and HTLV-1.[10] The risk of tuberculosis is about 2.5-fold higher in HIV/HTLV-1-coinfected patients compared to patients with HIV infection alone, and both morbidity and mortality are higher in patients with HIV/HTLV-1 coinfection.[11,12]

Using quantitative HTLV-1 proviral load (PVL) as an indicator of when to initiate ART in asymptomatic HIV/HTLV-1-coinfected individuals with preserved CD4 counts is often impractical due to limited laboratory capacity in countries where HTLV-1 infection is endemic. In addition, HTLV-1 PVL varies widely in asymptomatic carriers and in persons with HTLV-associated disease[13]; and the relationship between HTLV-1 PVL and HIV-associated manifestations in HIV/ HTLV-1-coinfection has not been well established. Hence, neither quantitative HTLV-1 PVL nor CD4 count is a clinically useful indicator of disease or immune status in the setting of HIV/HTLV-1 coinfection.

In contrast, limited evidence suggests that coinfection with HTLV-2, common among HIV-infected injection drug users in the United States and Europe, has a protective effect against progression of HIV infection to AIDS.[14] Human T-cell lymphotropic virus type 2 has been associated with increased CCL3L1 expression, a chemokine that inhibits HIV-1 cell entry by binding to CCR5.[6] Furthermore, HIV/HTLV-2-coinfection has been linked to a "long-term nonprogressor" phenotype. Therefore, the effect of HTLV-1 and HTLV-2 coinfection upon HIV infection may be different, and our recommendation for considering earlier initiation of ART in persons with HIV/ HTLV-1 coinfection may not apply to people with HIV/ HTLV-2 coinfection.

In conclusion, compared to a CD4 count greater than 200 cells/mm^3 in persons infected with

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In conclusion, compared to a CD4 count greater than 200 cells/mm^3 in persons infected with HIV alone, a similar CD4 count in persons coinfected with HIV and HTLV-1 may mask immunosuppression, and if used as an indicator for determining when to initiate ART in resource-limited settings, may delay initiation of ART, resulting in a missed opportunity to slow the progression to AIDS, decrease the risk of tuberculosis, and lessen complications related to chronic immune activation. Therefore, initiating ART earlier in HIV/HTLV-1-coinfected persons, independent of CD4 count, should be considered, especially in areas where tuberculosis is endemic.

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Retroviral Coinfections: HIV and HTLV: Taking Stock of More Than a Quarter Century of Research

Mark A. Beilke

Abstract

Retroviral coinfections with HIV-1 and HTLV-1 or with HIV-1 and HTLV-2 occur with variable frequencies throughout the world with the highest prevalence in large metropolitan areas in the Americas, Europe, and Africa. The recognition that retroviral coinfections exist dates back to the discovery of HIV-1 over 25 years ago. Despite the large body of published information regarding the biological and clinical significance of retroviral coinfections, controversy throughout several decades of research was fueled by several flawed epidemiologic studies and anecdotal reports that were not always supported with ample statistical and scientific evidence. However, the growing consensus obtained from recent systematic and well-devised research provides support for at least three conclusions: (1) HIV-1 and HTLV-1 coinfections are often seen in the context of patients with high CD4$^+$ T cell counts presenting with lymphoma or neurological complications; (2) HIV-1 and HTLV-2 coinfections have been linked in some cases to a "long term nonprogressor" phenotype; and (3) differential function and/or overexpression of the HTLV-1 and HTLV-2 Tax proteins likely play a pivotal role in the clinical and immunologic manifestations of HIV/HTLV-1 and -2 coinfections. This review will recount the chronology of work regarding retroviral coinfections from 1983 through the present.

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Case Report

HTLV-1 and HIV-1 co-infection: A case report and review of the literature

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ABSTRACT

HTLV type 1 and 2 are both involved in actively spreading epidemics, affecting over 15 million people worldwide. HTLV-1 has been described as the more clinically significant one, being associated with diseases such as adult T-cell leukemia and tropical spastic paraparesis. We report here a case of tropical spastic paraparesis in an HIV-positive patient who did not report any history of travel or residence in an HTLV endemic area.

A 57 year old African-American male was admitted to the hospital due to bilateral upper and lower extremity weakness associated with stiffness. He had recently been diagnosed with HIV. His physical examination showed mild to moderate decreased motor strength, in both upper extremities and marked loss in both lower extremities. This was associated with hyperreflexia and clonus. Sensory function was intact. He looked cachectic and had several psoriatic plaques on both lower and upper extremities. Laboratory work-up showed a CD4 count decreased to 94 cells/mm^3 and a HIV viral load of 273,000 copies/mL. Based on serum positivity for HTLV type 1 and the patient's clinical presentation suggestive of upper and lower motor neuron dysfunction, the diagnosis of tropical spastic paraparesis was made.

HTLV and HIV share the same routes of transmission and the same tropism for T-lymphocytes. Co-infection occurs probably more frequently than we are aware, since testing for HTLV is not routinely performed in outpatient HIV clinics.

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Background

Human T-cell lymphotropic virus (HTLV) was initially identified in 1979 and it represents the first human retrovirus ever described [1]. It belongs to the Retroviridae family, in the genus Deltaretrovirus, and predominantly affects T lymphocytes. Its genome is a positive single-stranded RNA [2]. So far, 4 types of HTLV have been established, but specific illnesses have been associated only with type 1 and 2. HTLV type 1 and 2 are both involved in actively spreading epidemics, affecting over 15 million people worldwide [3]. HTLV-1 has been described as the more clinically significant one, being associated with diseases such as adult T-cell leukemia and tropical spastic paraparesis. HTLV and HIV share the same routes of transmission and the same tropism for T-lymphocytes. Co-infection occurs probably more frequently than we are aware, since testing for HTLV is not routinely performed in outpatient HIV clinics [4]. We report here a case of tropical spastic paraparesis in an HIV-positive patient who did not report any history of travel or residence in an HTLV endemic area.

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Case report

A 57 year old African-American male was admitted to the hospital due to bilateral upper and lower extremity weakness associated with stiffness, more severe in his lower extremities. His symptoms had started several months prior to this presentation and had gradually worsened to the point that he was no longer able to ambulate. He denied any fever, chills, seizures or any history of trauma.

His past medical history was significant for psoriasis and HIV, diagnosed eight months prior. He was not enrolled in HIV care and he was not on any medications at home. He had no past surgical history. The patient denied tobacco use, alcohol abuse or any illicit drug use. He was born in Florida, United States, and had never

intravenous contrast, was interpreted as normal.

Laboratory work-up showed a CD4 count decreased to 94 cells/mm^3 and HIV viral load by PCR of 273,000 copies/mL. His white blood count was 4×10^9/mm^3 and hemoglobin was 11.6 g/dL. Platelets were within normal limits at a level of 186×10^9/mm^3. Renal, hepatic and thyroid function, as well as electrolytes, were all within normal limits. Cerebrospinal fluid analysis showed 102 red blood cells/mm^3, 42 white blood cells/mm^3 with 100% lymphocytes, normal protein, normal glucose and negative bacterial, fungal and mycobacterial cultures. VDRL, CMV PCR, JC PCR, HSV PCR, Cryptococcal antigen and West Nile virus IgM and IgG were all negative in the cerebrospinal fluid. HTLV-1 antibodies were negative in CSF, but positive in serum by enzyme-linked immunosorbent assay and confirmed by Western blot.

Based on serum positivity for HTLV type 1 and the patient's clinical presentation suggestive of upper and lower motor neuron dysfunction, the diagnosis of tropical spastic paraparesis was made. He was started on symptomatic treatment with muscle relaxants and gabapentin. He was also started on HIV treatment with tenofovir/emtricitabine, atazanavir and ritonavir. He was also started on opportunistic infection prophylaxis with daily trimethoprim-sulfamethoxazole and weekly azithromycin. He was discharged to a physical rehabilitation center. One year after initial presentation he had gained over 50 lbs and his lower extremity weakness and stiffness had improved. He was able to walk with a rolling walker. His CD4 count had increased to 169 cells/mm^3 and HIV viral load had decreased to less than 20 copies/mL.

Discussion

Human T-cell virus type 1 (HTLV-1) is well recognized as the cause of tropical spastic paraparesis (TSP), disease that is also known as HTLV-1-associated myelopathy (HAM) [5]. TSP/HAM is an upper motor neuron syndrome that primarily affects the lower extremities. It presents as a slowly progressive spastic paraparesis and the sensory signs are generally less evident. This syndrome can develop as early as three months after infection with HTLV-1 [6], but an average of three years of latency is more typical, and 20–30 years is possible [2]. An age of onset at 50 years or older is usually associated with a more rapid progression to a severe disability.

The diagnosis of TSP/HAM is based mainly on history and physical exam, in context of positive HTLV-1 serology. HTLV 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). Spine imaging studies are done mostly to rule out other causes of myelopathy. Magnetic resonance imaging of spine may show evidence of demyelination in patients with TSP/HAM and similar changes can be seen also in the periventricular white matter. However, these lesions also occur in patients with asymptomatic HTLV-1 infections, and comparisons with controls

HTLV-1 and HIV-1 co-infection

HTLV-1 predominantly affects CD4 lymphocytes. In vitro, HTLV-1 is also capable of infecting other cell types and this has been attributed to the fact that one of its host-receptors is GLUT-1, an ubiquitous glucose transporter.

HTLV-1/HIV-1 co-infection is more frequently reported in South America, the Caribbean and Africa [8–10]. Studies suggest that HTLV-1/HIV-1 co-infection is associated with a modification of the natural history of HIV-1, with a faster clinical progression to AIDS and a shorter survival time [11]. HIV-1 appears to up-regulate HTLV-1 expression, leading to a higher risk of HTLV-1 associated diseases, such as TSP/HAM and adult T-cell leukemia. However, clinical evidence remains controversial due to methodological problems in the majority of currently published studies [12].

An early study of HIV and HTLV-1 co-infection done by Leung et al. in 1988 [13] reported that co-infection with HTLV-1 led to increased production of specific host cell proteins which results in stimulation of HIV replication. In 1994 Schechter et al. published a case-control study of 27 patients with HIV/HTLV-1 co-infection [14] and concluded that co-infection was associated with higher CD4 counts, but at the same time with evidence of more advanced HIV clinical disease. A case–control, retrospective study, published in 2001 by Brites et al. [8] of 198 HIV-1 infected patients, of which 63 cases were co-infected with HTLV-1 concluded that HIV-1/HTLV-1 co-infected patients had a shorter mean survival than the HIV-1 mono-infected patients, regardless of sex or baseline CD4 cell count. Sobesky et al. found an increased risk of death for HIV-1/HTLV-1 co-infected patients from French Guiana, compared to HIV-1 mono-infected ones, but the power of their conclusion was limited by small sample size. Out of 151 HIV-infected patients that were included in this study, only 18 patients were co-infected [15].

In 2004 Beilke et al. published a longitudinal study, done in New Orleans, that looked at 62 patients with HIV/HTLV-1 co-infection and compared them to a group of 824 HIV-mono-infected patients. They found no significant differences in progression to AIDS, presence of opportunistic infections or death between these two groups [16]. There were though some methodological issues that may have influenced their results. This study included patients that were on antiretroviral medications, while some of the prior studies were done in the pre-HAART era. Beilke et al. mention that they adjusted the analysis for the use of antiretroviral drugs, but there is no description in the article on the duration of HAART therapy, patient follow-up or time when antiretroviral medication was started in HIV mono-infected patients, as compared to co-infected ones [17].

HTLV-2 and HIV-1 co-infection

HIV-1/HTLV-2 co-infection predominates in North America and Europe, especially among IV drug users [18]. The available evidence suggests a possible protective role of HTLV-2 co-infection

with a slowing of progression to AIDS [19]. HIV/HTLV-2 co-infected patients had lower levels of T-cell activation with lower rate of HIV replication [18]. In the retrospective study published by Beilke et al., 141 patients with HIV/HTLV-2 co-infection were compared to 824 patients who were HIV-mono-infected. Their conclusion was that HIV/HTLV-2 co-infection was statistically associated with delayed progression to both AIDS and death [16]. A longitudinal study by Turci and al. of 2371 HIV-1 infected Caucasian, intravenous drug users from Italy, of whom 6.7% were co-infected with HTLV-2, found that the co-infected patients were older aged, had higher baseline CD4 counts and delayed progression to AIDS [20].

Conclusions

HIV and HTLV-1/2 co-infection may occur more frequent than we are aware of, since routine testing for HTLV-1/2 in outpatient HIV clinics is not currently recommended. HTLV-1 and HTLV-2 co-infection seem to have different effects on HIV infected individuals. It appears that HTLV-1 may accelerate clinical progression to AIDS and the HIV virus may promote a higher risk of HTLV-1 associated diseases. However, some of the available data is contradictory. HTLV-2 co-infection seems to have a protective role, decreasing progression to AIDS. One common denominator between HTLV-1 and HTLV-2 co-infection in HIV patients, is that both have been linked to higher CD4 counts. The higher CD4 counts may have resulted in a delay in introduction of antiretroviral therapy in these co-infected patients [12].

There is definitely a need for larger, well designed studies in order to clearly determine the impact of HIV/HTLV-1/2 co-infection.

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HTLV-1/-2 and HIV-1 co-infections: retroviral interference on host immune status.

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Abstract

The human retroviruses **HIV**-1 and **HTLV-1**/HTLV-2 share similar routes of transmission but cause significantly different diseases. In this review we have outlined the immune mediated mechanisms by which HTLVs affect **HIV**-1 disease in **co**-infected hosts. During **co-infection** with **HIV**-1, HTLV-2 modulates the cellular microenvironment favoring its own viability and inhibiting **HIV**-1 progression. This is achieved when the HTLV-2 proviral load is higher than that of **HIV**-1, and thanks to the ability of HTLV-2 to: (i) up-regulate viral suppressive CCL3L1 chemokine expression; (ii) overcome **HIV**-1 capacity to activate the JAK/STAT pathway; (iii) reduce the activation of T and NK cells; (iv) modulate the host miRNA profiles. These alterations of immune functions have been mainly attributed to the effects of the HTLV-2 regulatory protein Tax and suggest that HTLV-2 exerts a protective role against **HIV-1 infection**. Contrary to **HIV**-1/HTLV-2, the effect of **HIV-1/HTLV-1 co-infection** on immunological and pathological conditions is still controversial. There is evidence that indicates a worsening of **HIV-1 infection**, while other evidence does not show clinically relevant effects in **HIV**-positive people. Possible differences on innate immune mechanisms and a particularly impact on NK cells are becoming evident. The differences between the two **HIV-1**/**HTLV-1** and **HIV**-1/HTLV-2 **co-infections** are highlighted and further discussed.

KEYWORDS: **HIV**-1; HTLV; JAK/STAT; chemokines; **co-infection**; cytokines; miRNA; natural killer cells

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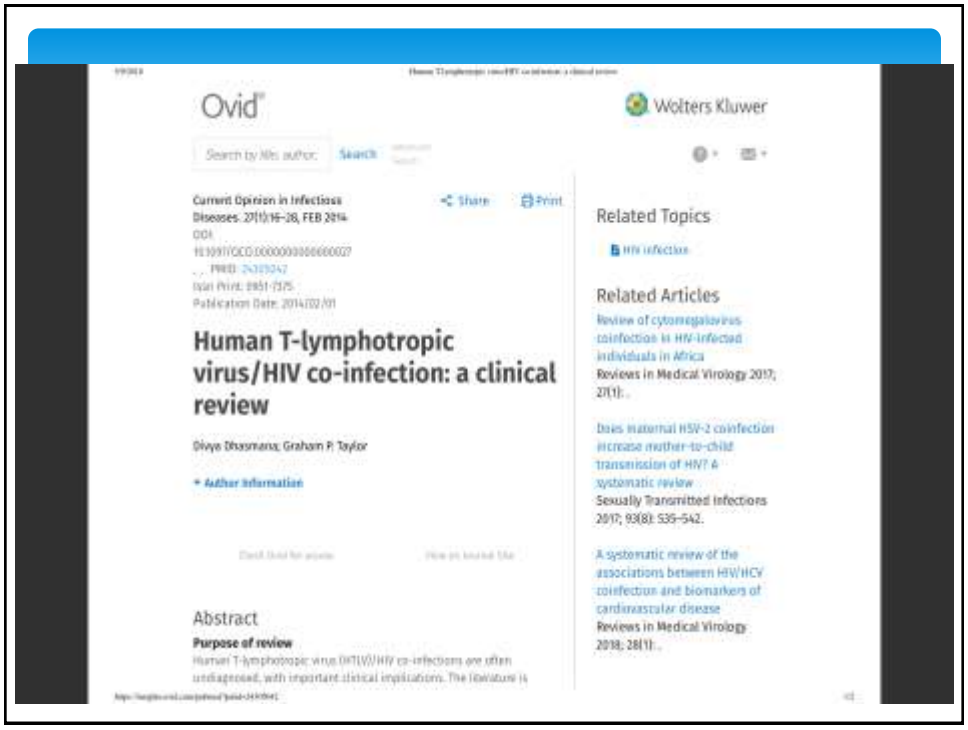
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relatively sparse with key observations derived in the pre-highly-active antiretroviral therapy era.
Recent findings
The epidemiology of co-infection, the impact of each virus on the other, with particular reference to clinical manifestations and the impact of antiretroviral therapy on HTLV are described.
Summary
Important clinical effects of HTLV/HIV co-infection include the higher rates of myelopathy and other neurological disorders and the poor predictive value of CD4+ cell counts as a surrogate for immune suppression. Current antiretroviral therapies in isolation have no proven effect on HTLV-1/2 proviral load.
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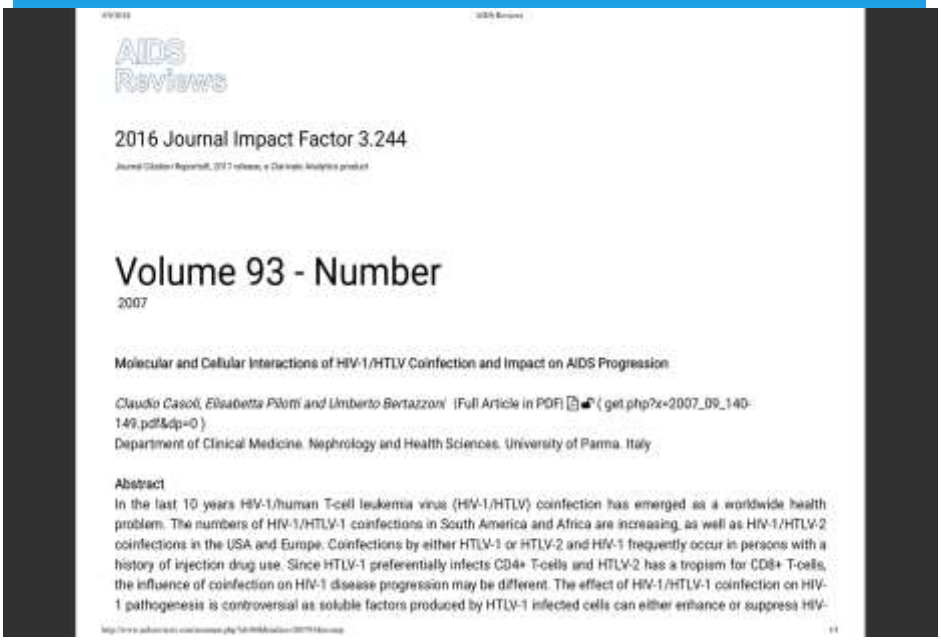

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Molecular and Cellular Interactions of HIV-1/HTLV Coinfection and Impact on AIDS Progression

Claudio Casoli, Elisabetta Pilotti and Umberto Bertazzoni (Full Article in PDF) (get.php?x=2007_09_140-149.pdf&dp=0)
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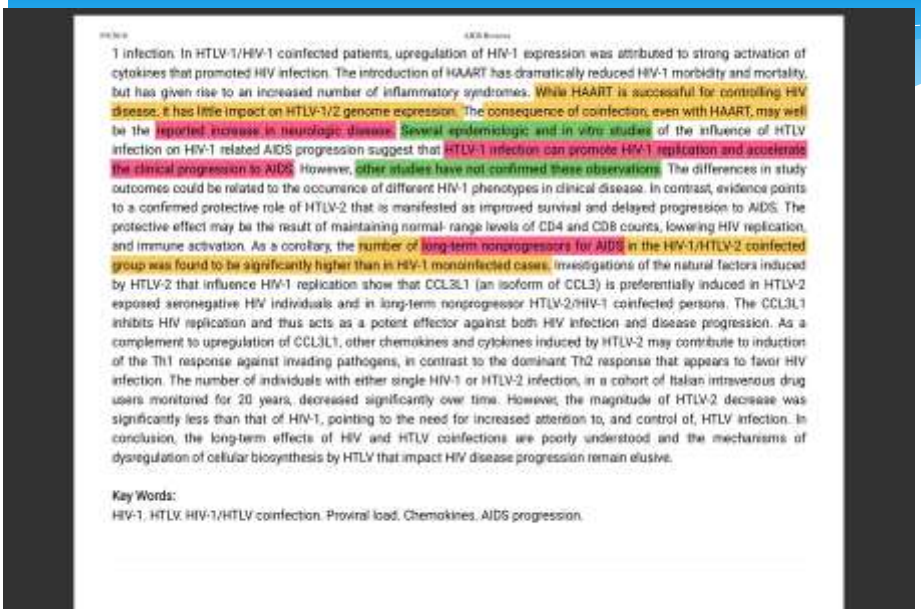
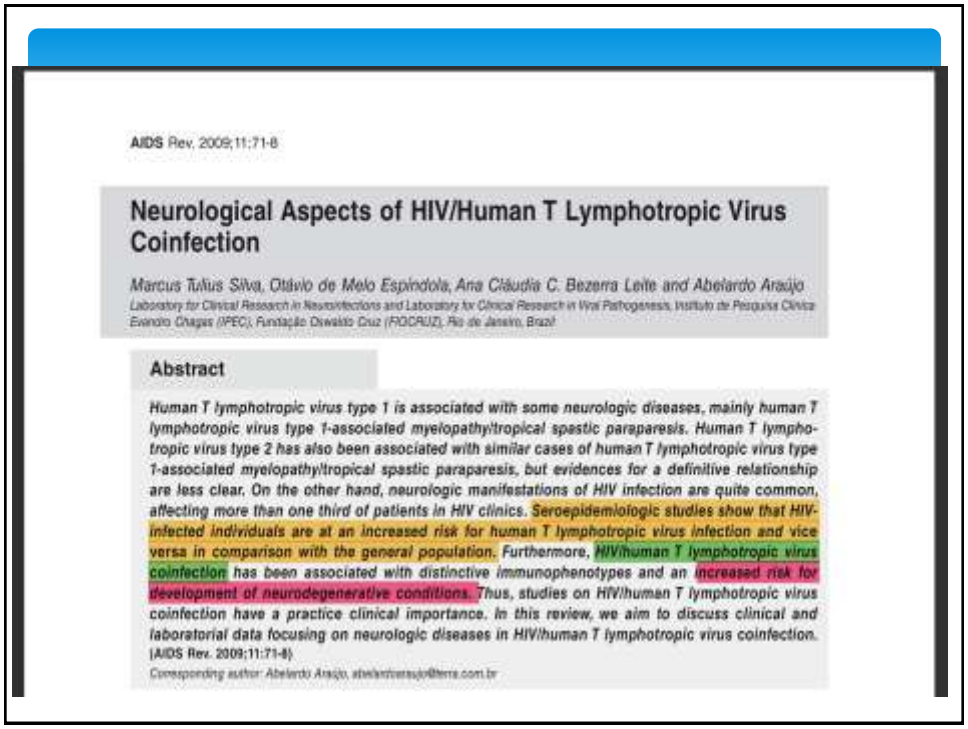
Abstract
In the last 10 years HIV-1/human T-cell leukemia virus (HIV-1/HTLV) coinfection has emerged as a worldwide health problem. The numbers of HIV-1/HTLV-1 coinfections in South America and Africa are increasing, as well as HIV-1/HTLV-2 coinfections in the USA and Europe. Coinfections by either HTLV-1 or HTLV-2 and HIV-1 frequently occur in persons with a history of injection drug use. Since HTLV-1 preferentially infects CD4+ T-cells and HTLV-2 has a tropism for CD8+ T-cells, the influence of coinfection on HIV-1 disease progression may be different. The effect of HIV-1/HTLV-1 coinfection on HIV-1 pathogenesis is controversial as soluble factors produced by HTLV-1 infected cells can either enhance or suppress HIV-



1 infection. In HTLV-1/HIV-1 coinfected patients, upregulation of HIV-1 expression was attributed to strong activation of cytokines that promoted HIV infection. The introduction of HAART has dramatically reduced HIV-1 morbidity and mortality, but has given rise to an increased number of inflammatory syndromes. While HAART is successful for controlling HIV disease, it has little impact on HTLV-1/2 genome expression. The consequence of coinfection, even with HAART, may well be the reported increase in neurologic disease. Several epidemiologic and in vitro studies of the influence of HTLV infection on HIV-1 related AIDS progression suggest that HTLV-1 infection can promote HIV-1 replication and accelerate the clinical progression to AIDS. However, other studies have not confirmed these observations. The differences in study outcomes could be related to the occurrence of different HIV-1 phenotypes in clinical disease. In contrast, evidence points to a confirmed protective role of HTLV-2 that is manifested as improved survival and delayed progression to AIDS. The protective effect may be the result of maintaining normal- range levels of CD4 and CD8 counts, lowering HIV replication, and immune activation. As a corollary, the number of long-term nonprogressors for AIDS in the HIV-1/HTLV-2 coinfected group was found to be significantly higher than in HIV-1 monoinfected cases. Investigations of the natural factors induced by HTLV-2 that influence HIV-1 replication show that CCL3L1 (an isoform of CCL3) is preferentially induced in HTLV-2 exposed seronegative HIV individuals and in long-term nonprogressor HTLV-2/HIV-1 coinfected persons. The CCL3L1 inhibits HIV replication and thus acts as a potent effector against both HIV infection and disease progression. As a complement to upregulation of CCL3L1, other chemokines and cytokines induced by HTLV-2 may contribute to induction of the Th1 response against invading pathogens, in contrast to the dominant Th2 response that appears to favor HIV infection. The number of individuals with either single HIV-1 or HTLV-2 infection, in a cohort of Italian intravenous drug users monitored for 20 years, decreased significantly over time. However, the magnitude of HTLV-2 decrease was significantly less than that of HIV-1, pointing to the need for increased attention to, and control of, HTLV infection. In conclusion, the long-term effects of HIV and HTLV coinfections are poorly understood and the mechanisms of dysregulation of cellular biosynthesis by HTLV that impact HIV disease progression remain elusive.

Key Words:
HIV-1, HTLV, HIV-1/HTLV coinfection, Proviral load, Chemokines, AIDS progression.



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Neurological Aspects of HIV/Human T Lymphotropic Virus Coinfection

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Abstract

Human T lymphotropic virus type 1 is associated with some neurologic diseases, mainly human T lymphotropic virus type 1-associated myelopathy/tropical spastic paraparesis. Human T lymphotropic virus type 2 has also been associated with similar cases of human T lymphotropic virus type 1-associated myelopathy/tropical spastic paraparesis, but evidences for a definitive relationship are less clear. On the other hand, neurologic manifestations of HIV infection are quite common, affecting more than one third of patients in HIV clinics. Seroepidemiologic studies show that HIV-infected individuals are at an increased risk for human T lymphotropic virus infection and vice versa in comparison with the general population. Furthermore, HIV/human T lymphotropic virus coinfection has been associated with distinctive immunophenotypes and an increased risk for development of neurodegenerative conditions. Thus, studies on HIV/human T lymphotropic virus coinfection have a practice clinical importance. In this review, we aim to discuss clinical and laboratorial data focusing on neurologic diseases in HIV/human T lymphotropic virus coinfection.
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بررسی مقایسه ای سیر بیماری HIV دربیماران مبتلابه HIV و HTLV-1 در مرکز مشاوره مشهد

* ***مقدمه:*** مطالعات قبلی نشان دهنده اثرات متناقضی از عفونت همزمان با **HTLV-I** روی پاتوژنز **HIV** می باشد. روش کسب این دو ویروس یکسان است اما اثر انها روی سلول های **CD4** متفاوت است. این مطالعه به مقایسه سیر بالینی و یافته های ازمایشگاهی بیماران با عفونت همزمان **HIV/HTLV-I** و مقایسه ان با بیماران **HIV** پرداخته است.
* ***روش انجام کار***: این مطالعه به روش هیستوریکال کوهورت در مرکز مشاوره بیماریهای رفتاری- عفونی مشهد انجام شد. میزان پلاکت , گلبول های سفید, نوتروفیل, سطح **CD4** , **stage** و شدت بیماری در زمان تشخیص وزمان شروع درمان **ART** در پایان مطالعه مورد بررسی قرار گرفت
* و داده ها توسط **SPSS** تجزیه وتحلیل شد.

بررسی مقایسه ای سیر بیماری HIV دربیماران مبتلابه HIV و HTLV-1 در مرکز مشاوره مشهد

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* ***نتایج:*** ٦٤ بیمار شامل ٦١ مرد و ٣ زن بررسی شدند که ٤٢ نفر از انها **HIV** مثبت بودند(٣٥ نفر از انها **HCV** مثبت هم بودند.) و٢٢ نفر از انها **HIV/HTLV-I** مثبت بودند(١٨ نفر از انها **HCV** مثبت هم بودند.) یافته های دموگرافیک شامل سن ومیزان تحصیلات و وضعیت شغلی و وضعیت تآهل و سابقه رفتارهای پر خطر بررسی شدند.

* **بحث :**افراد با عفونت همزمان سابقه رفتارهای پرخطر بیشتری داشتند به خصوص مصرف مواد مخدر تزریقی.مرگ در گروهی که مبتلا به **HTLV-I** بودند شایعتر بود و عفونتهای فرصت طلب در هر دو گروه تقریبا یکسان بود. تمام یافته های ازمایشگاهی به جز **CD4** در دو گروه تقریبا یکسان بود. **Stage** و شدت بیماری در دو گروه تفاوتی نداشت.