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CLINICAL TRANSPLANTATION

Human T-cell leukemia virus type I infection in various recipients of transplants from the same donor

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Abstract

Background.

The human T-cell lymphotrophic virus (HTLV) causes adult T-cell leukemia-lymphoma, tropical spastic paraparesis-HTLV type I, and associated myelopathy.

Methods.

An analysis was performed of serum samples from a multiorgan donor and the five recipients. Also studied was the donor's family and the partner of one of the renal recipients. Serologic detection of anti-HTLV antibodies was carried out by enzyme immunoassay and Western blot to confirm and discriminate between HTLV types. Analysis of proviral DNA was performed by polymerase chain reaction and sequenced in the long terminal repeat region and the *env* gene. Peripheral blood

mononuclear cell samples from all the recipients of the HTLV-I–positive organs and the donor's mother were studied.

Results.

Two years after transplantation, three organ recipients positive for antibodies to HTLV-I were detected (two kidney transplants and one liver). All the recipients' serum samples were negative at the time of transplantation except those from the multiorgan donor. The donor's mother was born in Venezuela and was confirmed positive for antibodies to HTLV-I. The remaining family members were negative. HTLV-I DNA sequences were recovered, amplified, and sequenced from all the samples from the HTLV-I–positive recipients and the donor's mother. The homology of HTLV-I sequences was 100% in all cases.

Conclusions.

The authors are reporting the first documented case of HTLV-I infection in several transplant recipients sharing the same donor. The donor was infected by vertical transmission. HTLV-I infection has devastating consequences for some immunocompromised organ recipients. This emphasizes the need for a systematic survey of HTLV antibodies in all potential donors.

The human T-cell lymphotropic virus (HTLV) type I, isolated in 1980 ([1]), is the etiologic agent of human adult T-cell leukemia-lymphoma (ATLL) ([2]), tropical spastic paraparesis (TSP) and HTLV type I-associated myelopathy (HAM) ([3,4]), and some inflammatory disorders in a small proportion of infected individuals ([5]). ATLL is an aggressive, drug-resistant malignancy with a median survival of less than 12 months despite modern chemotherapeutic regimes ([6]). Subacute myelopathy (HAM-TSP) is the main neurologic manifestation of HTLV-I infection.

HTLV-I has a global distribution with high endemic foci in Japan, Central Africa, the Caribbean countries, certain areas of South America, and Melanesia and is found among immigrants from these regions in Europe. The prevalence of HTLV-I infection in the endemic areas is between 3% and 30%. In Western countries, it is less than 1%. Seroprevalence increases with age ([7]); in older age groups, rates are usually higher among women than men.

It is transmitted through sexual contact (especially from men to women), by blood transfusions, and after organ transplantation ([8]); in areas of high prevalence, breast-feeding is an important route of transmission, particularly if continued for over 6 months ([9]). Immunosuppression enhances the risk of infection, reduces latency, and accelerates clinical development ([10]).

The epidemiology of HTLV in Europe has been reviewed recently ([11]). The seroprevalence in Spain is 1.9 per 1,000 for HTLV ([12]). Some cases of myelopathy associated with HTLV-I have been reported in European countries ([13]), generally in immigrant patients, residents in endemic zones, or patients having received transfusions. The first patient in Spain was reported in 1991 ([14]). The authors report here the first documented case of HTLV-I infection in various transplant recipients sharing the same donor in a Western country.

MATERIALS AND METHODS

On 5 October 1998, a 44-year-old woman received a liver transplant and subsequently developed paraparesis after 2 years ([15]). Serologic tests for HTLV-I were positive in blood and cerebrospinal fluid, and the polymerase chain reaction test was positive in blood. The donor was an apparently

healthy young man who died after brain injury. He was a multiorgan donor (kidneys, liver, and corneas).

The authors have conducted a survey for HTLV-I in all the recipients before and after transplantation. Serum samples from the multiorgan donor and the five recipients were analyzed. The donor's family members (parents, sister, partner, and daughter) were also analyzed, as was the partner of one renal recipient.

The serologic detection of anti-HTLV antibodies was carried out by enzyme immunoassay (Murex HTLV I+II; Murex Biotech Limited, Dartford, United Kingdom). A Western blot was used to confirm and to discriminate between HTLV types (Bioblot HTLV; Biokit SA, Barcelona, Spain).

Analysis of proviral DNA was performed by polymerase chain reaction (PCR) and sequenced in the long terminal repeat (LTR) region and the a fragment of the *env* gene. Blood samples from all the HTLV-I–positive recipients, two kidneys recipients (ES-TRR1 and ES-TRR2), one liver recipient (ES-TRH), and the donor's mother (ES-TMD) were studied. The blood samples were collected in sterile EDTA-containing tubes. Peripheral blood mononuclear cells (PBMC) were extracted from 500 μL whole blood using the AMPLICOR Whole Blood Specimen Preparation Kit (Roche Diagnostic Systems, Inc., Branchburg, NJ). The pelleted cells were treated with proteinase K (100 μg/mL) in a solution containing 10 mM Tris-HCl (ph 8.3), 50 mM KCl, 2.5 mM $MgCl_2$, 1% gelatin (10 ng/mL), 0.45% Nonidet P-40, and 0.45% Tween 20 at 65°C for 1 hr 10 min at 95°C. Ten microliters of this DNA solution was amplified.

The following primers were used for amplification of the LTR region: HFL1 (5′-CCAAGCTTGACAATGACCATGAGC-3′) and HFL6 (5′-GTTAAGCCAGTG ATGAGCGGC-3′) for the first round. These primers amplify a fragment of the 878 base pairs (bp) for the HTLV-I–positive samples, corresponding to nucleotides 23 to 39 and 480 to 901 of the HTLV-I ATK isolate. A second round was carried out with nested primers LTR1 (5′-ACCATGAGCCCCAAATATCCC-3′) and LTR2 (5′-GAATAAAGGGGCGCTCGAATCC-3′), which amplify a 784-bp fragment of HTLV-I corresponding to nucleotides 31 to 51 and 794 to 815.

For amplification of the a fragment of the *env* gene, the authors used the following primers: 1E8 (5′-TCAAGAAGTTTCACACCTCAAT-3′) and ENV22 (5′-GGCGAGGTGGAGT CCTTGGAGGC-3′) for the first round. These primers amplify a fragment of the 1,168 bp for the HTLV-I–positive samples, corresponding to nucleotides 5,628 to 5,649 and 6,774 to 6,796 of the HTLV-I ATK isolate. A second round was carried out with nested primers ENV1 (5′-TCAAGCTATAGTCTCCTCCCCCTG-3′) and ENV2 (5′-GGGAGGTGTCGTAGCTGCAGGAGG-3′), which amplify a 569-bp fragment of HTLV-I corresponding to nucleotides 6,045 to 6,068 and 6,588 to 6,614.

An alternative second round was carried out with the nested primers 1E8 and 12E4 (5′-GCTAGGGCGGAGACAAGCC-3′), which amplify a fragment of 548 bp corresponding to nucleotides 5,628 to 5,649 and 6,158 to 6,176 of the HTLV-I ATK isolate. Extensive measures were used to prevent and detect carryover contamination. All PCR runs included positive controls consisting of dilutions of DNA HTLV-I MT2 isolate, and negative controls included uninfected PBMC and water.

Amplified DNA was purified from agarose gel using the Quiagen gel extraction kit (Qiagen, Chatsworth, CA). PCR products were directly sequenced with inner primers and were resolved using an automated DNA sequencer (Applied Biosystems 377 sequencer; PEBiosystems, Foster City, CA). The nucleotide sequences obtained were aligned with the reference sequences of HTLV-I subtypes from the GenBank sequence database using the Clustal X program ([16]).

After the regions containing gaps had been eliminated, the aligned sequences were used for

phylogenetic analysis, which was carried out using programs contained in the MEGA program, version 2.1 (http://www.megasoftware.net/text/downloads.sht). The phylogenetic trees were based on a Kimura two-parameter distance matrix using the neighbor-joining method. Bootstrap resampling (1,000 replications) was applied to neighbor-joining trees to place approximate confidence limits on individual branches.

The 23 LTR strains were aligned using a 725-nucleotide sequence. The 20 *env* HTLV-I strains were aligned using a 519-nucleotide sequence corresponding to the gp21 protein. The HTLV-I strains used for LTR and *env* phylogenetic analysis were ATK (Japan, J02029), HS35 (Jamaica, D13784), EL (USA, M67514), GAB7 (Gabon, L76311), PH236 (Gabon, L76307), MEL5 (Melanesia, L02534), H23 (Cameroon, L46645, L76312), pyg19 (C.A.R., L76310), Efe1 (D. R. Congo, Y17014, Y17021), Lib2 (Gabon, Y17017, Y17022), Ni1.Peru (Peru, Y16484, Y16495), Ni2.Peru (Peru, Y16487, Y16496), Ni3.Peru (Peru, Y16485, Y16497), Bl1.Peru (Peru, Y16481, Y16492), Bl2.Peru (Peru, Y16482, Y16493), Bl3.Peru (Peru, Y16483, Y16494), Me2.Peru (Peru, Y16479, Y16490), Qu1.Peru (Peru, Y16475, Y16486), and BOI (France, L36905).

RESULTS

The authors found that the solid organ recipients were positive for antibodies to HTLV-I, two kidney transplants (ES-TRR1 and ES-TRR2) and one liver (ES-TRH). Both cornea recipients were negative for antibodies to HTLV. All the recipients' serum samples were negative at the time of transplantation, except those from the multiorgan donor (kidneys, liver, and corneas).

Two years after transplantation, the three positive infected organ recipients (ES-TRR1, ES-TRR2, and ES-TRH) developed neurologic manifestations associated with severe spastic paraparesis of HTLV-I infection. The donor's mother (ES-TMD) was confirmed positive for antibodies to HTLV-I. She was born in Venezuela and was asymptomatic. The remaining members of the family were negative for HTLV-I. Figure 1 shows the detection of anti-HTLV antibodies in all the serum samples analyzed. HTLV-I DNA sequences were successfully recovered, amplified, and sequenced from the samples from all the HTLV-I–positive recipients and the donor's mother.

F1-17
Figure 1:
Analysis by Western blot. Lane 1, negative control; lane 2, positive HTLV-I control; lane 3, positive HTLV-II control; lane 4, serum donor; lane 5, serum liver recipient before transplantation; lane 6, serum liver recipient after transplantation (ES-TRH); lane 7, serum kidney A recipient before transplantation; lane 8, serum kidney A recipient after transplantation (ES-TRR1); lane 9, serum kidney B recipient before transplantation; lane 10, serum kidney B recipient after transplantation (ES-TRR2); lane 11, serum cornea A recipient after transplantation; lane 12, serum cornea B recipient after transplantation; lane 13, serum donor's father; lane 14, serum donor's mother (ES-TMD); lane 15, serum donor's sister; lane 16, serum donor's partner; lane 17, serum donor's daughter; lane 18, serum kidney A recipient's partner.

The phylogenetic analysis performed in the LTR region and a fragment of the *env* gene are shown in Figures 2 and 3, respectively. The donor's mother and the recipients were infected by an identical genotype. The homology of HTLV-I sequences recovered from the recipients and the mother was 100% in all four cases. The nucleotide sequence data reported in this study have been deposited in the GenBank sequence database under the access numbers AF494238 to AF494245.

F2-17
Figure 2:
Phylogenetic analysis of HTLV-I isolates. LTR sequences were aligned using the

CLUSTAL X program with previously reported strains (geographic origins are given in parentheses); their accession numbers are listed in the text (see *Methods*). Phylogenetic trees were constructed by the neighbor-joining method, using the nucleotide distance data set obtained by the Kimura two-parameter approach. Horizontal branch lengths are to scale. Bar = 0.01 of genetic distance. Vertical branches are for clarity only. Values at nodes are percentages of 1,000 bootstrap replicates (>50%). (*Within boxes*) Sequences of HTLV-I isolates from Spain obtained in 2001 (ES-TMD, ES-TRR1, ES-TRR2, and ES-TRH).

F3-17
Figure 3:
Phylogenetic analysis of HTLV-I isolates. The a fragment of the *env* gene sequences were aligned using the CLUSTAL X program with previously reported strains (geographic origins are given in parentheses); their accession numbers are listed in the text (see *Methods*). Phylogenetic trees were constructed by the neighbor-joining method, using the nucleotide distance data set obtained by the Kimura two-parameter approach. Horizontal branch lengths are to scale. Bar = 0.01 of genetic distance. Vertical branches are for clarity only. Values at nodes are percentages of 1,000 bootstrap replicates (>50%). (*Within boxes*) Sequences of HTLV-I isolates from Spain obtained in 2001 (ES-TMD, ES-TRR1, ES-TRR2, and ES-TRH).

The subtype classifications in both regions were concordant, and the four samples (donor's mother, both kidney recipients, and the liver recipient) clustered with subtype A and close to the strains came from patients born in American countries and Japan with a bootstrap value of 97% of the LTR region and 83% of the a fragment of the *env* gene region of the HTLV-I. The analysis of the LTR region of the four HTLV-I strains (mother and recipients) demonstrated a divergence of 1.82% with respect to the sequence of the ATK-1 prototype (Japan); a divergence of 2.68% to the strain HS-35 (Caribbean); and a divergence of 0.55% to 0.97% to strains Bl2, Qu1, and Me2 from Peru and Boi from France. These divergences show a greater relation to the American strains than to those from Japan in these regions.

The analysis of the a fragment of the *env* gene region of the four HTLV-I strains (mother and three recipients) demonstrated a divergence of 1.76% with respect to the sequence of the ATK-1 prototype (Japan), a divergence of 1.95% to the strain HS-35 (Caribbean), and a divergence of 0.77% to 0.97% to strains Qu1 and Me2 (Peru), respectively. All the strains used in the authors' analysis are cosmopolitan and are clearly distant from the HTLV-Ib (Central Africa), HTLV-Ic (Melanesia), HTLV-Id (Central Africa), HTLV-Ie (D. R. Congo), and HTLV-If (Central Africa) strains.

DISCUSSION

Phylogenetic trees based on nucleotide sequencing have cast further light on the worldwide distribution of HTLV. The decision was made to determine the sequences of the proviral DNA and to conduct a phylogenetic analysis to elucidate the origin of these HTLV-I strains, ES-TMD, ES-TRR1, ES-TRR2, and ES-TRH. The LTR (especially the U3 and U5 regions) is a more variable fragment and is thus more informative for genetic comparison and phylogenetic analysis.

The subtype classifications in the LTR region of the four samples clustered with subtype A was closer to the strains from patients born in American countries than those from Japan. These results are in agreement with a phylogenetic analysis of the *env* gene based on a 519-bp fragment of gp21. However, because of limited sequence variation within the *env* region of the cosmopolitan HTLV-I, this tree was less informative than the LTR tree and did not provide additional information. They are clearly distant from HTLV-I strains classified in the other two major lineages: Melanesian and Central African.

The donor's mother was born in Venezuela and moved to Spain after marrying a local man. She was later shown to be an asymptomatic carrier of HTLV-I by the authors' study. In Venezuela, Zabaleta et al. ([17]) reported in 1994 a general HTLV-I seroprevalence of 0.39% among mestizos (admixture of whites, Indians, and Negroes). The main route of mother-to-child transmission is postnatal breast-feeding. Refraining from breast-feeding or limiting the duration of breast-feeding can reduce the risk of mother-to-child transmission ([18]). Antenatal screening for HTLV, with a view to recommending formula feeding, must be taken into consideration in countries with high seroprevalence to prevent the vertical transmission of HTLV through breast-feeding.

Blood or organ donors are not currently screened for HTLV in Spain. This policy is based possibly on a relatively low prevalence of HTLV among the Spanish population and a low risk of developing the disease after infection. However, the consequences of such a policy may be tragic for patients who are infected. New evidence should prompt a reappraisal of this policy. Because travel is now more common, several investigators ([19]) have suggested since 1995 the need to introduce testing for HTLV antibodies in potential organ donors and in volunteer blood donors. The virus has generally been considered to be of little relevance in Europe, although HTLV-I seropositivity and acute T-cell leukemia-lymphoma have been widely documented in Europe among Afro-Caribbean immigrants ([20,21]) with histories of sexual exposure to partners at risk of HTLV infection ([22]) and now among white Europeans with no apparently identifiable risk factors. In Spain, it is now recommended that individuals presenting a risk of transmission for HTLV-I and HTLV-II virus, such as intravenous drug users, people from endemic countries, and their sexual partners, be refused as potential organ donors. The authors' multiorgan donor was an apparently healthy young Spanish man who died after brain injury.

Tosswill et al. ([13]) suggested that HTLV-I infection is more widespread in the population than previous studies have estimated. Information on the prevalence of HTLV-I infection in Europe ([11,23]) derives mainly from studies of blood donors and studies of antenatal patients. The lower prevalence of HTLV infection in blood donors than in antenatal patients can be at least partly explained by two factors: first, there was a higher proportion of white people among the blood donors; second, blood donors are screened to exclude high-risk groups. Data from metropolitan areas of the United Kingdom and France suggest that the seroprevalence of HTLV-I in pregnant women is up to 100 times higher than in blood donors. HTLV-I infection is also more common in patients attending sexually transmitted disease clinics.

Blood donor screening for HTLV was introduced first in Japan in the mid-1980s, followed by the United States in 1988, Canada in 1989, and France in 1991. Furthermore, in France, the test for HTLV infection is mandatory in all organ donors. Subsequently, a number of other countries (the French West Indies, Portugal, Holland, Sweden, Denmark, Luxembourg, Finland, and The Netherlands) have started to perform blood donor testing, varying from regular screening of all donations to testing of new donors only or surveillance of proportions of blood donors ([21,24]). The prevalence rates of HTLV are insufficiently known in many regions of the world ([25]). The data acquired from blood donor screening constitute an important body of knowledge on the spread of HTLV.

The risk of developing disease after infection with HTLV-I has previously been assumed to be low because ATLL and tropical spastic paraparesis develop in only 2.5% to 4.0% of cases in which infection is not acquired through blood transfusion, and the incubation period is long, ranging from 10 to 30 years. Between 12.8% and 63.4% of people receiving blood infected with HTLV-I will seroconvert ([26,27]). Fresh blood products (those <6 days old) have a transmission efficiency of 80% ([26]). The incubation period between infection and the onset of myelopathy is substantially shorter in patients infected by blood transfusion than in patients who acquire the infection through breast-feeding or by the venereal route ([4,28]).

Several investigators ([23]) have suggested that immunocompromised individuals may be more at risk than healthy carriers of the virus ([4]). The time between transfusion and the development of myelopathy was short in an immunosuppressed patient who received an organ transplant, suggesting a relationship between viral load, the immune system, and the development of HTLV-I myelopathy ([8,14,28,29]). Nakamura et al. ([30]) have suggested that in nonimmunocompromised persons, HTLV-I infection is a relatively insignificant problem, and occurrence of ATLL in the setting of immunosuppression is unknown because the HTLV-I status of the recipient before transplant was not known. In the authors' study, all the recipients' serum samples were negative at the time of transplantation, except those from the multiorgan donor.

The authors are reporting the first documented case of HTLV-I infection in various transplant recipients sharing the same donor who was infected by vertical transmission. Two years after transplantation, the three positive infected organ recipients developed neurologic manifestations associated with severe spastic paraparesis of HTLV-I infection, a progressive, unremitting myelopathy for which there is no specific treatment. This aggressive condition responds poorly to chemotherapy.

Two points should be emphasized in this report. The first is that HTLV-I infection has devastating consequences for some immunocompromised organ recipients. The second is the need to consider a systematic survey of HTLV antibodies in all potential donors, despite the low current prevalence of HTLV-I infection in Western countries. The presence of HTLV-I must be taken into consideration in diagnostic tests.

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