Delayed Seroconversion and Rapid Onset of Lymphoproliferative Disease After Transmission of Human T-Cell Lymphotropic Virus Type 1 From a Multiorgan Donor

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(See the Editorial Commentary by Taylor on pages 1425–6.)

Background. Human T-cell lymphotropic virus type 1 (HTLV-1) screening of blood and organ donors is not mandatory in Germany because of its low prevalence (about 7/100 000). An HTLV-1 transmission event caused by a multiple organ donor was investigated. Validity of diagnostic procedures and HTLV-1 disease association in immunosuppressed organ recipients were analyzed.

Methods. Two screening immunoassays and an immunoblot (confirmatory assay) were used for detection of HTLV-1/2 antibodies. Proviral DNA was quantified in blood and biopsies of organ recipients by HTLV-1 real-time polymerase chain reaction (PCR).

Results. Proviral HTLV-1-DNA was detected in all blood samples of 3 organ recipients (1–100 copies/10² cells), but seroconversion was delayed for up to 2 years in screening assays and >6 years in the confirmatory assay. In 2 of 3 organ recipients, a cutaneous T-cell lymphoma was diagnosed 2 and 3 years after infection, respectively. Proviral HTLV-1 DNA concentration was almost 100 copies/10² cells in cutaneous lymphoma biopsies whereas in biopsies of other tissues ≤3.0 copies/10² cells were found. The third organ recipient did not suffer from lymphoma, but detailed clinical data on this patient were not available to us.

Conclusions. Biopsy results support an etiological role for HTLV-1 in these cases of primary cutaneous T-cell lymphoma after solid organ transplant. HTLV-1–associated lymphoma can arise quickly in immunocompromised transplant recipients. The diagnosis of potentially HTLV-1–associated disease in organ recipients may require PCR because of delayed seroconversion.

Keywords. HTLV-1; cutaneous lymphoma; SOT; transmission; transplantation.
cases of transmission by transplantation have also been observed worldwide and only a single case in Europe [9–11].

Japan, as one of the endemic countries, implemented HTLV screening of blood donors in 1986 to prevent transmission of HTLV-1 by blood transfusions [12]. The United States started blood donor screening 2 years later [13]. Although Europe is an area of low prevalence, some European countries including France and the United Kingdom made HTLV antibody screening of blood donors mandatory [14]. In general, the seroprevalence rate in the European Union is between <1 per 100 000 and 30 per 100 000 in blood donors. These low values are due to self-exclusion of HTLV-1 high-risk groups by questionnaire and an underrepresentation of ethnic minorities among blood donors [15]. An extraordinarily high prevalence of 53.3 per 100 000 was found among first-time blood donors in Romania [14]. So far, >10 incidents of HTLV-1 transmission by blood transfusion have been reported in Europe, and only a single incident in 2002, in which the virus was transmitted by a solid organ donor [10]. This resulted in HTLV-1 infection of 3 organ recipients in Spain. Follow-up examination showed that the donor’s mother, born in Venezuela where seroprevalence is 390 per 100 000, was positive for HTLV-1 [16]. In nonendemic countries, including Spain, immigrants from endemic regions are the main source of HTLV transmission [15, 17, 18]. Therefore, since 2005, HTLV antibody screening has been mandatory in Spain for organ donors coming from areas where HTLV-1 is endemic or with a high suspicion of HTLV-1 infection [19]. In Germany, the testing of organ or blood donors is neither mandatory nor recommended because of a very low HTLV-1 seroprevalence (about 7/100 000; Table 1) [20–23].

Here, a detailed investigation on the second HTLV-1 transmission by solid organ transplant affecting 3 recipients in Europe is reported. Delayed seroconversion and rapid onset of primary cutaneous lymphoma was observed in 2 recipients, but all 3 recipients are alive (follow-up time, 6 years).

### METHODS

#### HTLV-1/2 Serology

For HTLV-1/2 antibody screening, the chemiluminescent immunoassay Architect rHTLV-1/2 (Abbott, Abbott Park, Illinois) and the enzyme immunoassay Murex HTLV I + II (Diasorin, Saluggia, Italy) were used for serum samples. The MPD HTLV BLOT 2.4 (MP Biomedicals, Santa Ana, California) was used for the confirmation. All diagnostic assays conformed to European Union regulations for in vitro medical devices (“CE marked”). All blood samples were stored at −20°C and were not thawed until testing for HTLV-1.

#### HTLV-1 Real-Time Polymerase Chain Reaction

DNA was extracted from ethylenediaminetetraacetic acid (EDTA) blood samples with the QIAamp DNA Blood Kit (Qiagen, Hilden, Germany) and from biopsy samples with the DNeasy Blood and Tissue Kit (Qiagen). Proviral DNA of HTLV-1 was quantified by TaqMan polymerase chain reaction (PCR) using primers and a TaqMan probe (Eurogentec, Seraing, Belgium) as described previously [24] and a ready-made master mix with a HotStarTaq DNA polymerase (QuantiTect Probe PCR Kit, Qiagen). The cycling conditions on the Applied Biosystems 7500 Real-Time PCR System (Life Technologies, Carlsbad, California) were as follows: 15 minutes at 95°C, 40 cycles of 30 seconds at 95°C, 20 seconds at 55°C, and 1 minute at 65°C.

For the biopsy samples, a second real-time PCR for the human CRP gene was performed to normalize the cell number and to exclude PCR inhibition [25].

#### Histopathology and Immunohistochemistry

Formaldehyde-fixed (3.5%) and paraffin-embedded biopsies were stained with hematoxylin-eosin and Giemsa. Immunohistochemistry was done on the same material with antibodies for CD3, CD4, CD7, CD8, CD20, CD25, CD30, CD33, CD68, and Ki-67. Primary antibodies were purchased from Ventana.

### Table 1. Summary of Published Human T-Cell Lymphotropic Virus Seroprevalence Data in German Blood Donors, Pregnant Women, and Intravenous Drug Users, 1984–2000

<table>
<thead>
<tr>
<th>Population</th>
<th>Cohort Size</th>
<th>HTLV-1/HTLV-2 Positives</th>
<th>Rate per 100 000</th>
<th>Year(s)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood donors</td>
<td>248 000</td>
<td>53*</td>
<td>20</td>
<td>1990</td>
<td>[21]</td>
</tr>
<tr>
<td>Blood donors</td>
<td>76 000</td>
<td>2/0</td>
<td>3</td>
<td>1993–1994</td>
<td>[18]</td>
</tr>
<tr>
<td>Pregnant women</td>
<td>58 747</td>
<td>4/0</td>
<td>7</td>
<td>1997–2000</td>
<td>[23]</td>
</tr>
<tr>
<td>Pregnant women</td>
<td>30 000</td>
<td>2/0</td>
<td>7</td>
<td>1994–1997</td>
<td>[22]</td>
</tr>
<tr>
<td>IVDUs</td>
<td>3392</td>
<td>0/0</td>
<td>0</td>
<td>1984–1994</td>
<td>[20]</td>
</tr>
<tr>
<td>IVDUs</td>
<td>6000</td>
<td>0/0</td>
<td>0</td>
<td>1994–1997</td>
<td>[22]</td>
</tr>
</tbody>
</table>

Abbreviations: HTLV, human T-cell lymphotropic virus; IVDU, intravenous drug user.

* Screening in this study did not distinguish between HTLV-1 and HTLV-2.
DNA Fingerprinting of Lymphoma Cells
Laser microdissections and subsequent genetic fingerprinting for the identification of donor or recipient origin of dissected cells were performed essentially as described previously [26].

RESULTS

Index Case
In 2008, cutaneous lymphoma with laryngeal lesions was diagnosed in a liver-transplanted patient (recipient 1). This unusual cutaneous lymphoma in a liver transplant recipient was described as mycosis fungoides (MF) in a case report [27]. The transplant had been performed in 2006 as treatment for gastric neuroendocrine carcinoma with hepatic metastasis (Table 2).

In early 2011, a screening assay for HTLV-1/2 antibodies was requested by the treating physician and was found to be positive. HTLV-1 infection was confirmed by immunoblot assay (Figure 1). Proviral HTLV-1 DNA was detected in all available (archival) EDTA blood samples after transplant (Table 3). The patient had no epidemiological risk factors for HTLV-1 infection (e.g., intravenous drug use or migration from/travel to an endemic region). Retrospective analysis demonstrated that the patient was HTLV-1 seronegative in pretransplant serum samples from 2003 and from 2006, the latter sample having been obtained immediately before liver transplant (Table 2).

Analysis of Infection Chain
An archival serum sample of the liver donor was tested retrospectively by screening assays and a confirmatory assay (immunoblot) for HTLV-1/2 and found to be seropositive for HTLV-1 (Figure 1). A suitable sample for detection of proviral HTLV-1 DNA was not available from the donor. In the following traceback investigation by the organ procurement organization, 2 other organ recipients of the same organ donor were identified. In 1 of these 2 kidney recipients, 2 archived posttransplant blood samples were available. In the other recipient, only a single posttransplant blood sample was available. In the other recipient, only a single posttransplant blood sample was available (Table 3). Screening assays for HTLV-1/2 antibodies were positive from all 3 samples, and proviral HTLV-1-DNA was detected by PCR in both kidney recipients, thus confirming HTLV-1 infection (Table 3).

Delayed Seroconversion for HTLV-1
An archival serum sample from recipient 1 (index patient) obtained 3 days after transplant was positive in the HTLV screening assay, but showed only a very weak reaction in the immunoblot assay (Figure 1, lane 6). The band pattern was almost

<table>
<thead>
<tr>
<th>Table 2. Characteristics of Transplant Recipients</th>
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<tbody>
<tr>
<td>Characteristic</td>
</tr>
<tr>
<td>Age, y</td>
</tr>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Primary disease</td>
</tr>
<tr>
<td>SOT</td>
</tr>
<tr>
<td>HTLV-1 serostatus pretransplant</td>
</tr>
<tr>
<td>Outcome</td>
</tr>
</tbody>
</table>

Abbreviations: HTLV, human T-cell lymphotropic virus; SOT, solid organ transplant.
identical to that of a 100-fold prediluted donor serum (Figure 1, lane 5). A comparison of the reactivity of the recipient 1 serum 3 days after transplant with a dilution series of the donor serum in the HTLV screening assay showed that an equal reactivity was observed between the 1:200 and 1:400 dilution of the donor serum (Figure 2). This finding would be explained by the transmission of 5–10 mL of donor blood with the transplanted liver and is compatible with a low, donor-acquired HTLV-1 antibody titer in recipient 1 early after transplantation. This assumption of a transient, donor-derived seroreactivity was confirmed in the follow-up because the HTLV antibody screening test of recipient 1 was negative on day 108 after transplant (Table 3). As a first sign of seroconversion, a single p24 band was observed in the immunoblot assay (Figure 1, lane 7) on day 626 after transplant but the screening tests for HTLV antibodies were still negative (Table 3). Full seroconversion (positive screening tests and positive immunoblot) was observed as late as 5 years after infection, but no serum samples were available between day 626 and day 1832. However, type-specific antibodies to recombinant HTLV-1 rgp46 were still undetectable (Figure 1, lane 8). Recipient 2 was found to be reactive for HTLV antibodies in screening assays but remained negative in the immunoblot assay (about 6 years after transplant; Figure 1, lane 9; Table 3). Archival serum samples of recipient 3 were not available, but this recipient was found to be positive for HTLV antibodies and the immunoblot on day 1925 after transplant (Figure 1, lane 10) and by HTLV-1 PCR (Table 3). Interestingly, as in recipient 1, type-specific antibodies to recombinant HTLV-1 rgp46-1 were also lacking in recipient 3, whereas they were clearly detectable in the donor (Figure 1, lane 3).

**Cutaneous HTLV-1–Associated T-Cell Lymphoma**

Cutaneous lymphoma was diagnosed in recipient 1 two years after liver transplant, and in recipient 2 three years after kidney transplant. Symptoms were skin and laryngeal lesions in

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**Table 3. Human T-Cell Lymphotropic Virus (HTLV) Types 1 and 2 Serology and Proviral HTLV-1 DNA Load of Transplant Recipients in Blood Samples**

<table>
<thead>
<tr>
<th>Days After Transplant</th>
<th>CMIA</th>
<th>EIA</th>
<th>Blot</th>
<th>PCR (Copies/10⁷ Cells)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recipient 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3a</td>
<td>Positive</td>
<td>Positive</td>
<td>Ambiguousᵇ</td>
<td>ND</td>
</tr>
<tr>
<td>108ᵇ</td>
<td>Negative</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>613ᶜ</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>Positive (10.0)</td>
</tr>
<tr>
<td>626ᵃ</td>
<td>Negative</td>
<td>Negative</td>
<td>Ambiguousᵈ</td>
<td>ND</td>
</tr>
<tr>
<td>1832ᵃ</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
<td>ND</td>
</tr>
<tr>
<td>1858ᶜ</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>Positive (8.4)</td>
</tr>
<tr>
<td>Recipient 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>224</td>
<td>Positive</td>
<td>Positive</td>
<td>Negative</td>
<td>Positive (2.5)</td>
</tr>
<tr>
<td>2111</td>
<td>Positive</td>
<td>Positive</td>
<td>Negative</td>
<td>Positive (44.0)</td>
</tr>
<tr>
<td>Recipient 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1925</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
<td>Positiveᵉ</td>
</tr>
</tbody>
</table>

Abbreviations: CMIA, chemiluminescent microparticle immunoassay; EIA, enzyme immunoassay; ND, not determined; PCR, polymerase chain reaction.

ᵃ Only serum available.
ᵇ Detection only of donor-acquired p19 and GD21 antibodies (see Figure 1).
ᶜ Only EDTA blood available.
ᵈ Detection only of p24 antibodies (see Figure 1).
ᵉ Quantitative data not available.

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**Figure 2.** Serial dilutions of the donor serum were tested by a human T-cell lymphotropic virus chemiluminescent microparticle immunoassay. The dilution factor of the donor serum was calculated, which showed the same reactivity in comparison to the antibody titer of the recipient 3 days after transplant. Abbreviations: HTLV-1/2, human T-cell lymphotropic virus types 1 and 2; S/CO: sample value/calibration value.
recipient 1 and painful acral ulcers of the skin in recipient 2. Skin biopsies of both recipients revealed a cutaneous infiltration of T lymphocytes consistent with a pleomorphic, cutaneous T-cell lymphoma. In recipient 1, the dermis and submucosa contained a dense infiltrate of CD3-, CD4-, CD8-, CD25-, and Ki-67 (80%)-positive, CD7-, CD30-, CD20-, CD33-, CD68-negative, predominantly small-sized T lymphocytes with a narrow cytoplasm and folded and angulated nuclei with a dense chromatin in close vicinity to the surface epithelium (Figure 3A and 3C). Only occasionally larger blasts were intermingled with folded nuclei resembling MF cells (Figure 3C). There was intense invasion of the squamous epithelium with formation of so-called microabscesses within the epithelium. In recipient 2, the intradermal infiltrate also consisted of small- to medium-sized lymphocytes with expression of CD3, CD4, CD25, and Ki-67 (70%) with folded nuclei (Figure 3B). The gamma and beta chains of the T-cell receptor gene were not found to be rearranged in either patient (data not shown). Systemic evaluation did not reveal any extracutaneous manifestation of a lymphoma or leukemic-type disease in either patient. The third recipient did not develop a lymphoproliferative disease, but detailed clinical data on this recipient were not available.

The proviral HTLV-1 DNA concentration in the cutaneous lymphoma biopsies was in the range of almost 100 copies/10² cells (40 in recipient 1, 99.0 in recipient 2). For comparison, multiple archival biopsies taken after transplant (liver, appendix, duodenum, gallbladder, lymph node) of both recipient 1 and 2 were analyzed. In these nonlymphoma tissues, the HTLV-1 DNA concentration was >10-fold lower (range, 0.3–3.0 copies/10² cells).

Genetic fingerprinting of the lymphoma cells of both patients indicated the recipient origin of these tumor cells and excluded transmission of a donor-derived lymphoma with the transplanted organ (data not shown).

**Therapeutic Intervention and Outcome**

Because the HTLV-1 infection was still unknown in both patients at the time of lymphoma diagnosis, tacrolimus was suspected as an etiological factor for the lymphoma in recipient 1 [27]. Therefore, tacrolimus was terminated and recipient 1 was treated with high-dose corticosteroids [27]. The cutaneous lymphoma of recipient 2 was also treated with high-dose intravenous corticosteroids (250 mg prednisolone daily). Immunosuppression with tacrolimus was tapered to 1.5 mg daily and immunosuppression with mycophenolate mofetil was continued with 360 mg daily. Complete remission of the cutaneous lymphoma was achieved in both recipients. Both patients are currently alive and well.

**DISCUSSION**

This reported incident of HTLV-1 transmission by solid organ transplant is remarkable for 2 reasons. The first is the development of HTLV-1-positive skin lymphomas with a similar histology in 2 recipients only 2 and 3 years after infection, respectively. The second is the delayed seroconversion and lack of type-specific HTLV-1 gp-46 antibodies observed in all 3 recipients.

In the general population, the time from HTLV-1 infection to the manifestation of disease has been estimated to be approximately 20 years or more, but immunosuppression in transplanted patients might possibly lead to a more rapid development of disease [4]. A delayed seroconversion, as observed in all 3 recipients, and impaired cellular immunity may promote the clonal proliferation of HTLV-1-transformed cells. For example,

**Figure 3.** Histopathology of skin infiltrates. The squamous epithelium and subepithelial tissue were infiltrated by a population of uniform, predominantly small-sized T lymphocytes with folded nuclei. Recipients 1 (A) and 2 (B) revealed a very similar atypical infiltrate. B, An aberrant immunophenotype with CD7 negativity and CD25 expression. C, Bizarre cells with folded nuclei resembling mycosis fungoides cells are obvious (arrow; recipient 1). A, Hematoxylin-eosin staining (×200 magnification). B, CD25 immunohistochemistry (×200 magnification). C, Giemsa staining (×400 magnification).
the skin lesions of recipient 1 and 2 consisted of lymphoma cells carrying the HTLV-1 genome, because (almost) 100 copies of proviral HTLV-1 DNA per 10² cells was detected. For comparison, in other tissues only ≤3.0 copies proviral HTLV-1 DNA per 10² cells was found, which may be related to a low number of HTLV-1–infected lymphocytes infiltrating these tissues.

Delayed seroconversion may impede the diagnosis of HTLV-1 infection by serological screening tests in solid organ transplant recipients. As a first sign of seroconversion in recipient 1, a single p24 band was observed in the immunoblot assay on day 626 after transplant (Table 3). For comparison, HTLV-1 seroconversion was detected within 30–60 days after transfusion of HTLV-1–positive blood with anti-p24 generally appearing first [28]. HTLV-1 PCR proved to be reliable in transplant recipients, but HTLV-1 PCR is not performed as a routine diagnostic procedure, and is only available in a small number of specialized laboratories. As early manifestations of HTLV-1–associated diseases may present with atypical symptoms (eg, cutaneous lymphocyte infiltration), both HTLV-1 infections and diseases may be underdiagnosed in transplant recipients. In recipient 1, the atypical symptoms of HTLV-1 infection had been described as MF before the HTLV-1 infection was diagnosed [27]. Although an association of HTLV-1 with MF had been reported in a few case reports [29, 30], the majority of MF patients proved to be HTLV-1–seronegative and MF is not considered to be an HTLV-1–associated disease [31]. A second detailed immunohistopathological analysis of the skin biopsies revealed that a polyclonal expansion of HTLV-1–transformed lymphocytes mimicked the symptoms and histopathology of MF. In recipient 2, a similar immunohistopathological diagnosis of the skin lesions was made. These findings supported a common etiology, HTLV-1 infection plus immunosuppression, for the cutaneous lymphoproliferative disease observed in both recipients. In contrast to MF, and in contrast to ATLL caused by HTLV-1, the lymphoproliferative disease observed in this study was found to be benign because it responded well to treatment with high-dose corticosteroids and tapering of other immunosuppressive drugs. Furthermore, screening for HTLV may be considered in the workup of patients with T-cell lymphoproliferative disease, in particular immunocompromised ones, regardless of the perceived risk of HTLV infection.

Reports on HTLV-1 transmission by solid organ transplant are rare. Only a single other incident, also affecting 3 recipients, had been reported in Europe. In these recipients, the rapid development of subacute myelopathy >2 years after transplant was observed [10]. These clinical findings supported a hypothesis that transmission of HTLV-1 by organ transplant and subsequent immunosuppression leads to a rapid onset of HTLV-1–associated diseases. Furthermore, a multitude of case reports described HTLV-1–associated disease in transplant recipients with an unknown route of infection; some of these may have been infected by an HTLV-1–positive organ donor [32]. In contrast, a rapid onset of HTLV-1 diseases was not observed in 6 HTLV-1–naïve recipients of HTLV-1–positive organs (follow-up time, 4–10 years) [33]. Based on these results, the transplantation of HTLV-1–positive allograft was proposed for selected recipients under extended donor criteria [34]. This proposal may be questioned, considering our present results regarding 2 recipients of HTLV-positive organs who developed cutaneous lymphoma lesions only 2–3 years after transplant. However, it should also be taken into account that the lesions were reversible in both patients and there was no relapse in a follow-up period of currently >6 years. Thus, especially recipient 1 clearly benefited from liver transplant in spite of HTLV-1 infection, as a 6-year survival was very unlikely [27]. The same may be true for heart and lung transplant recipients of HTLV-positive donors.

On the contrary, the onset of HTLV-1–associated diseases was not found to be more rapid in patients who had been HTLV-1 infected prior to organ transplant. In 2 studies carried out with a total of 24 HTLV-1–positive kidney allograft recipients (average follow-up of 5 and 8 years, respectively) the HTLV-1–related diseases such as ATLL or HAM/TSP were not observed [33, 35]. Patient and graft survival were not significantly different from HTLV-1–negative kidney recipients [35]. HTLV-1–associated disease (ATLL) was described so far only in 2 solid organ transplant recipients who were HTLV-1–infected prior to transplant [36, 37]. Thus, HTLV-1 infection is not considered to be a contraindication for solid organ transplant.

In nonendemic areas (eg, Europe), the risk of HTLV-1 transmission is enhanced in selected donor populations. For example, 6 of 1100 Afro-Caribbean blood donors living in the United Kingdom tested positive for HTLV-1 whereas 0 of 1100 white donors were HTLV-1 infected [38]. In Germany, testing of organ or blood donors is neither mandatory nor recommended because of a very low HTLV-1 seroprevalence (about 7/100 000). In Germany, even screening tests with a high specificity of 99.95% would give rise to a positive predictive value of only 12% with the consequence of the unjustified exclusion of a much higher number of uninfected organ donors compared to the truly infected ones. Therefore, confirmatory testing is essential in any case of a positive HTLV-1/2 screening assay but due to time restriction hardly feasible in the organ donation setting. For the same reason, the HTLV-1/2 screening of organ donors was terminated in the United States [32] despite a >40-year-old epidemic of HTLV-2 due to injection drug use [39]. Testing for HTLV may be proposed for groups of donors with higher HTLV prevalence, for example, migrants from HTLV endemic areas, donors with a history of travel to these regions, or intravenous drug users. This strategy may increase the safety of solid organ transplant and limit the risk of losing organ donors due to false-positive screening results. Unfortunately, none of the potential risk factors for HTLV-1 infection had been reported in
our donor. However, this does not preclude the possibility that risk factors for HTLV-1 infection were present in our donor, because obtaining medical and social history is difficult and probably incomplete in case of deceased organ donors. Another limitation of the study was that pretransplant samples and clinical data for recipient 3 were not available.

In conclusion, HTLV-1 infection should be considered in the differential diagnosis of T-cell lymphoproliferative and neurological diseases in solid organ transplant recipients, even in cases with an early onset of disease after transplant. Laboratory diagnosis of HTLV-1 infection may require testing by PCR because of delayed seroconversion.

**Notes**

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All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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