Transmission of *Trypanosoma cruzi* by Heart Transplantation

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**Background.** *Trypanosoma cruzi* infection (i.e., Chagas disease) is an unusual complication that can occur after solid-organ transplantation and that can result in severe illness or death. In 2006, there were 2 heart transplant recipients in Los Angeles, California, reported to have acute trypanosomiasis during the same month. We conducted an investigation to determine the source of these infections.

**Methods.** We reviewed the medical, organ procurement, and donor transfusion and transplantation records of these 2 heart transplant recipients. The 2 heart transplant recipients were interviewed regarding any kind of natural exposure and were screened for parasites by obtaining blood and other tissue samples for buffy coat, culture, and polymerase chain reaction. Serum samples from the heart transplant recipients, organ donors, and blood donors were tested for *T. cruzi* antibodies by use of immunofluorescence assay and radioimmunoprecipitation assay. Tissue samples from the organ donors were examined by use of polymerase chain reaction and immunohistochemical staining. Other recipients of organs from the same donors were monitored for *T. cruzi* infection by use of polymerase chain reaction and immunofluorescence assay.

**Results.** Both heart transplant recipients had no apparent risk factors for preexisting *T. cruzi* infection. Both were seronegative but tested positive for the parasite, indicating recent infection. Both recipients died despite medical treatment. The organ donors tested positive for *T. cruzi* antibodies by use of radioimmunoprecipitation assay; the blood donors were seronegative. Six other patients had received a liver or kidney from these organ donors. None showed evidence of *T. cruzi* infection.

**Conclusions.** To our knowledge, this is the first report of *T. cruzi* transmission associated with heart transplantation. Clinicians and public health authorities should be aware that manifestations of Chagas disease can occur after transplantation, requiring rapid evaluation, diagnosis, and treatment.

Chagas disease, which is also called American trypanosomiasis, is caused by the parasite *Trypanosoma cruzi*. An estimated 8–11 million persons throughout parts of Latin America are affected [1]. Most infections in endemic areas are transmitted by triatomine insects, but the parasite can be acquired through other routes, including blood transfusion [2, 3], organ transplantation [4, 5], from mother to child by congenital transmission [6], and oral ingestion [7]. This acute vectorborne infection is usually asymptomatic or mild. If left untreated, it is followed by chronic subclinical infection, with low levels of the parasite in the blood and tissues. Most infected persons are asymptomatic, but 20%–30% eventually develop cardiomyopathy or gastrointestinal manifestations (e.g., a toxic megaesophagus or megacolon) [8]. Transplant recipients may experience severe infection. Immunosuppression can result in the reactivation of chronic Chagas disease, and newly acquired *T. cruzi* is of special concern in transplant recipients because of their limited ability to control the infection.
We describe an investigation of the source of *T. cruzi* infection in 2 patients who received a heart transplant at Los Angeles County area hospitals. In February 2006, the Acute Communicable Disease Control Program of the Los Angeles Public Health Department and the Centers for Disease Control and Prevention (CDC) were notified of these cases and conducted the investigation.

**METHODS**

**Clinical and epidemiological investigation.** The 2 heart transplant recipients were interviewed regarding recent travel to or residence in regions where Chagas disease is endemic, place of birth, and whether they had a history of blood transfusions, to identify possible past exposure to *T. cruzi*. For the organ-donor part of the investigation, the dispositions of all organs and tissues was ascertained; common factors associated with organ procurement and handling were examined; and organ-donor demographic, travel, and medical histories were reviewed for risk factors for *T. cruzi* infection. Tissue samples obtained from the organ donors were examined for the presence of *T. cruzi* by use of polymerase chain reaction (PCR) and immunohistochemical staining. Serum samples obtained from the organ donors were tested for *T. cruzi* antibodies by use of immunofluorescence assay (IFA) and radioimmunoprecipitation assay (RIPA). Transplantation records were reviewed, and the physicians of other organ transplant recipients from these donors were notified. These organ transplant recipients’ serum samples were monitored by use of serologic testing and PCR of whole blood.

Investigators reviewed the blood transfusion histories of both the organ donors and the transplant recipients. Blood-collection agencies were notified to recall and quarantine all components of the identified units of blood. Blood donors were identified, contacted, asked about symptoms and risk factors associated with Chagas disease, and asked to provide new specimens of blood for IFA, RIPA, and PCR testing.

**Laboratory methods.** Serologic testing was performed by use of IFA (at the CDC in Atlanta, GA) and RIPA [9, 10] (at the American Red Cross Holland Laboratory in Rockville, MD). CDC IFA testing uses fixed *T. cruzi* epimastigotes (the vector-associated developmental stage of *Trypanosomatidae*) and has a cutoff value of $\geq 1:32$ dilution. Formalin-fixed tissues were examined by use of immunohistochemical stains, according to a previously described method [11]. For PCR testing, DNA was extracted from blood samples by use of the QIAamp DNA Blood Mini Kit (Qiagen), according to the manufacturer’s suggested protocols. Minicircle kinetoplast DNA was amplified with a modified 121 primer and S36 primer by use of *Taq* DNA polymerase. Amplified fragments were cloned into the pGEM-T Easy Vector Kit (Promega US). For each sample that tested positive by PCR, 10 clones were PCR-amplified and sequenced by use of the BigDye Terminator Version 1.1 Cycle Sequencing Kit (Applied Biosystems) and run by use of an ABI 3130 XL sequencer (Applied Biosystems).

**RESULTS**

**Case reports of the 2 heart transplant recipients.** One of the heart transplant recipients was a 64-year-old man (hereafter referred to as heart transplant recipient 1) who received an orthotopic heart transplant in December 2005. He was immunosuppressed immediately after his orthotopic heart transplant as a result of being treated with tacrolimus, mycophenolate, and prednisone. His posttransplant clinical course was complicated by his development of renal insufficiency and atrial flutter. During the next 6 weeks, he had a reduced ejection fraction of 35%–40% (with diffuse hypokinesis of the septum and inferomedial wall), as measured by echocardiography, and elevated troponin levels. He was empirically treated with corticosteroids for acute graft rejection, without apparent improvement. Test results for various infectious disease markers were negative, and no sources of infection were identified. He was readmitted 8 weeks after surgery for failure to thrive, anorexia, fever, and diarrhea of 2 weeks’ duration. A peripheral blood smear revealed *T. cruzi* (figure 1A). Blood culture results were positive for *T. cruzi* trypomastigotes, and review of endomyocardial biopsy specimens revealed amastigotes (figure 1B). He was seronegative for *T. cruzi* antibodies but positive for *T. cruzi* DNA by use of PCR. His parasitemia cleared by day 8 of nifurtimox chemotherapy, and his troponin levels returned to normal. No amastigotes were detected in specimens of endomyocardial biopsies performed 2 and 6 weeks after diagnosis, and PCR performed on blood and tissue samples resulted in negative test results. Immunosuppression was adjusted to maintain therapeutic efficacy on the basis of clinical, laboratory, and endomyocardial biopsy surveillance. While receiving nifurtimox, heart transplant recipient 1 developed tremors, which required that there be an adjustment to the dosage. He initially improved clinically but died 20 weeks after surgery. Postmortem examination revealed acute cellular rejection, primarily within the atrioventricular node and bundle of His. No parasites were noted by use of routine stains on cardiac tissue autopsy specimens.

The other heart transplant recipient was a 73-year-old man (hereafter referred to as heart transplant recipient 2) with ischemic cardiomyopathy. He received an orthotopic heart transplant in January 2006 and was placed on an immunosuppressive regimen of tacrolimus, mycophenolate, and prednisone. He had mild left-ventricular systolic dysfunction early in his recovery, but he improved and was discharged 2 weeks after surgery. He was readmitted to the hospital 7 weeks after surgery complaining of fever and fatigue. During a manual differential examination, a peripheral blood smear revealed trypomasti-
gotes. Blood culture results were positive for *T. cruzi*. Nifurtimox therapy resulted in the clearing of parasites from peripheral blood within 10 days. He suffered progressively worsening neurologic adverse effects, including tremors, weakness, diplopia, and confusion. These persisted, despite switching antitrypanosomal therapy to benznidazole after 4 weeks of therapy. Examination of cerebrospinal fluid revealed cytomegalovirus infection. His clinical course was complicated by aspiration pneumonia; he developed congestive heart failure as well, with suspected graft rejection, although biopsies were inconclusive. No change in his immunosuppressive regimen was made during his hospitalization. *T. cruzi* was not observed in any endomyocardial biopsy specimens, and his serological and PCR test results were negative. His condition continued to deteriorate, and he died of cardiac failure 25 weeks after surgery. No autopsy was performed.

Neither of the 2 heart transplant recipients had apparent risk factors for previous exposure to *T. cruzi*. Both were seronegative for *T. cruzi* antibodies after surgery. Pretransplant serum samples were not available.

**Organ donors.** One of the organ donors was a 23-year-old Hispanic male construction worker (hereafter referred to as organ donor 1) who was hospitalized for a gunshot wound. He was declared brain dead, and the day after that his organs were recovered. A routine screening of organ donor 1 by the organ procurement organization revealed no symptoms or laboratory findings indicative of active infections. Organ donor 1 was born in the United States and had previously resided in Texas. He had stayed for several weeks in Guadalajara, Mexico, during the months before his death. Because travel-associated or autochthonous transmission of *T. cruzi* is rare, we attempted to contact the patient’s mother, who had been born in Mexico. However, she was unavailable for interview or testing to determine whether organ donor 1 could have been congenitally infected. Review of medical records and interviews with family members did not indicate other risk factors for *T. cruzi* infection. Lymph node tissue samples from organ donor 1 tested negative for *T. cruzi* by use of PCR and immunohistochemical staining. His serum samples tested borderline positive by use of IFA (1:32 dilution) and positive for antibodies to *T. cruzi* by use of RIPA (figure 2).

The other organ donor was a 25-year-old Hispanic male (hereafter referred to as organ donor 2) who was hospitalized for multiple traumatic head injuries. He was declared brain dead, and his organs were recovered and transplanted 4 days later. Screening of organ donor 2 by the organ procurement organization revealed no symptoms or laboratory findings indicative of infection before the fatal injury. He was born in El Salvador and moved to southern California in 2003. Medical records and interviews with family members did not suggest other risk factors for *T. cruzi* infection. His spleen tissue samples tested negative for *T. cruzi* DNA by use of PCR and immu-

**Figure 1.** *Trypanosoma cruzi* in heart transplant recipient 1. In human infection, *T. cruzi* is present in 2 forms: the bloodstream trypomastigote and the intracellular tissue amastigote. *A,* Peripheral blood smear displaying a typical trypomastigote. These are usually C or U shaped, with a centrally located nucleus and a kinetoplast at the posterior end. The arrow indicates the kinetoplast (Giemsa stain; original magnification, ×1000; oil immersion). *B,* Intracellular *T. cruzi* amastigotes within myocytes in an endomyocardial biopsy specimen. The arrow indicates the kinetoplast (hematoxylin and eosin stain; original magnification, ×1000; oil immersion).

**Figure 2.** Serologic testing of organ donors 1 and 2 by radioimmunoprecipitation assay. Organ donor 1 (lane 5) and organ donor 2 (lane 6) tested positive for *Trypanosoma cruzi* antibodies. Lanes 1–3 are negative controls; lanes 11–13 are positive controls. Diagnostic bands are 90 and 72 kilodaltons (kD).
Table 1. Summary of the results of laboratory testing for Trypanosoma cruzi infection in transplant recipients and organ donors.

<table>
<thead>
<tr>
<th>Donor or recipient</th>
<th>Outcome or status</th>
<th>Peripheral blood smear</th>
<th>Culture</th>
<th>PCR</th>
<th>Immunohistochemical staining</th>
<th>Serologic testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organ donor 1a</td>
<td>No reported disease</td>
<td>NT</td>
<td>NT</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Heart transplant recipient 1b</td>
<td>Died 137 days after transplant</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Liver transplant recipient 1c</td>
<td>Clinically healthy</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>NT</td>
<td>NT</td>
</tr>
<tr>
<td>Kidney transplant recipient 1Ae</td>
<td>Clinically healthy</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>NT</td>
<td>NT</td>
</tr>
<tr>
<td>Kidney transplant recipient 1Be</td>
<td>Clinically healthy</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>NT</td>
<td>NT</td>
</tr>
<tr>
<td>Organ donor 2d</td>
<td>No reported disease</td>
<td>NT</td>
<td>NT</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Heart transplant recipient 2e</td>
<td>Died 177 days after transplant</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Liver transplant recipient 2f</td>
<td>Clinically healthy</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>NT</td>
<td>NT</td>
</tr>
<tr>
<td>Kidney transplant recipient 2Ae</td>
<td>Clinically healthy</td>
<td>–</td>
<td>–</td>
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<td>NT</td>
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<tr>
<td>Kidney transplant recipient 2Be</td>
<td>Clinically healthy</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>NT</td>
<td>NT</td>
</tr>
</tbody>
</table>

NOTE. IFA, immunofluorescence assay; NT, not tested; PCR polymerase chain reaction; RIPA radioimmunoprecipitation assay.

* Serum, lymph node, and spleen tissue samples were tested.  
b Peripheral blood and endomyocardial tissue samples were tested.  
c Serum samples were examined 28 weeks (for liver transplant recipient 1), 27 weeks (for kidney transplant recipient 1A), or 30 weeks (for kidney transplant recipient 1B) after transplant.  
d Serum and spleen tissue samples were tested.  
a Serum, lymph node, and spleen tissue samples were tested.  
b Serum and spleen tissue samples were tested.  
d Serum samples were examined 37 weeks (for liver transplant recipient 2), 29 weeks (for kidney transplant recipient 2A), or 24 weeks (for kidney transplant recipient 2B) after transplant.

Chagas Disease Transmission by Heart Transplantation • CID 2009:48 (1 June) • 1537

Blood transfusions for the heart transplant recipients and organ donors. Heart transplant recipient 1 received 16 units of blood components during the perioperative course (13 units of red blood cells, 1 unit of apheresed platelets, and 2 units of fresh frozen plasma). Organ donor 1 received 7 units of blood components (4 units of red blood cells and 3 units of fresh frozen plasma). All of the blood donors for heart transplant recipient 1 and organ donor 1 were identified, and blood samples were obtained from them. All tested negative for T. cruzi antibodies by use of both IFA and RIPA, and their whole-blood samples tested negative by use of PCR.

Heart transplant recipient 2 received 12 units of blood during the perioperative course (5 units of red blood cells, 3 units of platelets, and 4 units of fresh frozen plasma). All blood donors were identified, and blood samples were obtained from all donors but one. These donors tested negative for T. cruzi antibodies by use of IFA and RIPA, and their whole-blood samples tested negative by use of PCR. Organ donor 2 received 9 units of blood components (7 units of red blood cells and 2 units of platelets) before organ recovery. The platelet donors and 4 of the 7 red blood cell donors were contacted, and blood samples were obtained. These tested negative for T. cruzi antibodies by use of IFA and RIPA, and their whole-blood samples tested negative by use PCR. The laboratory test results for the transplant recipients and organ donors are summarized in table 1.

Recipients of other solid organs. In addition to the heart, 2 kidneys and a liver had been procured from each organ donor (i.e., from organ donors 1 and 2) and were transplanted. No conduit vessels, tissues, or other organs were used. The 2 liver transplant recipients and the 4 kidney transplant recipients were observed for 16 months after surgery and remained asymptomatic and clinically stable (e.g., no graft rejection or dysfunction). They were seronegative for T. cruzi antibodies by use of IFA, and their serum samples demonstrated no evidence of parasitemia by use of PCR 7–10 months after surgery.

DISCUSSION

To our knowledge, this is the first report of T. cruzi transmission by cardiac transplantation. The evidence suggests that these heart transplant recipients acquired the parasite from infected organ donors as independent events. The recipients were seronegative for T. cruzi antibodies, with no risk factors for pre-existing infection; the blood donors were seronegative; and the organ donors were seropositive. However, because 1 blood donor for heart transplant recipient 2 could not be reached for retesting, we cannot rule out the possibility that heart transplant recipient 2 could have also been exposed to T. cruzi as a result of blood transfusion.

The transmission of T. cruzi by allograft is an unusual occurrence. It has been reported in the United States and South America for only 13 kidney transplant recipients [1, 12–18] and 4 recipients [4, 5, 19] of other solid organs (liver and pancreas and/or kidney), and the CDC has been notified of 2 additional transplant recipients infected by this route. Acute
Chagas disease is usually a mild or asymptomatic infection, but it is of special concern in severely immunocompromised patients, who may develop high levels of parasitemia, rapidly progressive infection, or life-threatening complications. Recognition of Chagas disease in organ transplant recipients is challenging, not only because of the low index of suspicion but also because the clinical course of the disease can be similar to allograft rejection or to more common infections.

The clinical manifestations of acute *T. cruzi* infection in heart transplant recipients 1 and 2 were nonspecific. The diagnoses were made 7–9 weeks after surgery, when parasitemia was observed in both recipients, which underscores the importance of microscopic examination of peripheral blood smears or buffy coats in establishing this diagnosis. PCR methods, which detect infection before patent parasitemia develops, have been used to monitor for reactivated chronic Chagas disease in patients immunosuppressed by human immunodeficiency virus infection [20] or a drug regimen [21–23] and for acute infection in solid-organ transplant recipients who might have been exposed to *T. cruzi* (A.M., unpublished data). The parasite can also be found in other fluid and tissue samples (e.g., the *T. cruzi* amastigotes observed in endomyocardial biopsy specimens of the new heart of heart transplant recipient 1). Serologic testing, a mainstay for the diagnosis of Chagas disease, may be less useful for this type of patient, because seroconversion does not always occur in immunosuppressed patients [15]. The reported incubation periods for *T. cruzi* transmitted by transplantation are longer than those characteristic of vectorborne infection (i.e., 1–3 weeks). Patent parasitemia developed in most transplant recipients during the second or third month after transplant [4, 5], but longer incubation periods (e.g., up to 23 weeks) have been observed among kidney transplant recipients [15]. Clinicians should be aware that signs and symptoms of *T. cruzi* infection can appear later than those of more common infections conveyed by allograft [24].

Although both heart transplant recipients 1 and 2 described in our report received antitrypanosomal chemotherapy, they died within weeks of diagnosis. Chagas disease was not the primary cause of death in these patients, but it likely contributed to the outcome. The diagnosis was not made until high parasitemia levels were present, and the infection in heart transplant recipient 1 may have been exacerbated by the administration of antirejection therapy, which is associated with reactivation of *T. cruzi* infection in heart transplant recipients with chronic Chagas disease [25]. The effects of high parasite load and graft-infiltrating parasites may have depressed cardiac function. The adverse effects of antitrypanosomal drug therapy complicated posttransplant care. *T. cruzi* infection is associated with immunomodulatory effects [26], although the impact of these effects on patients treated with an immunosuppressive regimen is unknown. Among the small number of acutely infected transplant recipients who were symptomatic at the time of diagnosis, the outcome has been poor, with the exception of a liver transplant recipient who presented with an acute chagasic cardiopathy and responded favorably to chemotherapy [19]. However, limited data suggest that improved outcomes might be achieved in these cases if acute infection were diagnosed early and treatment were promptly administered. We are aware of 4 organ transplant recipients for whom the donor’s seropositive status became known after liver or kidney transplantation but before they developed patent parasitemia or symptoms of patent parasitemia. These patients’ blood samples were monitored by serial examinations for circulating parasites by use of PCR (A.M., unpublished data) or microscopy of buffy coat [5], and they were given antitrypanosomal therapy as soon as *T. cruzi* infection was detected. This approach resulted in apparently successful treatment, which was characterized by the absence of detectable parasitemia, lack of symptoms, and continued seronegative status with follow-up periods of months to several years.

Patients with Chagas disease should be treated early in the course of the disease, when chemotherapy is thought to be most effective. The 2 existing antitrypanosomal drugs, nifurtimox and benznidazole, achieve cure in ∼70% of patients with acute infection [27]. A cure is more difficult to document for chronic disease, because seroreactivity usually persists for years in persons treated for a long-standing infection. However, the treatment of chronic disease suppresses parasitemia, might improve prognosis, and is recommended for certain patients [28]. The effectiveness of these drugs in immunosuppressed patients has not been studied systematically. Adverse effects are common, especially neurological toxicities. Neither drug has been approved for use in the United States. They can be obtained from the CDC Drug Service (telephone 404-639-3670 [weekdays] or 770-639-2888 [off-hour emergencies]) for use under investigational protocols.

The heart, gastrointestinal tract, and central nervous system are the main target organs in cases of *T. cruzi* infection; however, in humans and animal models, the parasite can be found in other organs [29, 30] and tissues, including bone [31], cartilage, and cornea [32]. Therefore, any organ or tissue from a seropositive donor must be regarded as potentially infectious. However, the transplantation of an organ from such a donor does not always result in the transmission of the infection; the risk may depend on such factors as donor parasite load and the specific organ or tissue involved. In addition to the heart, organ donors 1 and 2 described in this report also provided a liver or kidney to 6 other transplant recipients. These patients’ blood samples have been monitored by serial serologic testing and PCR examination and have shown no evidence of infection. These findings are consistent with data from case series in which *T. cruzi* transmission occurred in 12 (35%) of 34 of initially
seronegative patients who received a kidney from a seropositive donor [5]. Chagas disease is not considered an absolute contraindication to kidney donation in some South American hospitals [5]. No transmission of T. cruzi was observed in 3 allogeneic bone marrow transplants from seropositive donors [33]. Even heart transplantation may not always transmit the infection. In the single previous report of cardiac transplantation from a seropositive donor, the recipient showed no evidence of infection during 3 months of follow-up [34].

The best approach for the management of solid-organ transplant recipients who are at risk for graft-associated T. cruzi infection has not been identified. Chemoprophylaxis has not been formally evaluated. Serial monitoring and early treatment of confirmed cases of infection, rather than prophylaxis, have been viewed as the more prudent approach, given the substantial drug toxicity and the fact that transmission by transplantation does not always occur. However, prophylaxis has been used with apparent success for a small number of patients [35]. An estimated 100,000 persons in the United States are asymptomatically infected with undiagnosed T. cruzi [36]. Previously, data about the prevalence of T. cruzi infection, which varies by region, have been limited. However, in January 2007, the American Red Cross and Blood Systems Laboratories began screening donated blood for T. cruzi antibodies with a recently licensed assay [37]. In the first 18 months of screening, ~20 million blood-donation specimens were tested. The screening identified 2455 repeatedly reactive specimens, and 639 (26%) of these 2455 specimens tested positive for T. cruzi antibodies by use of RIPA [38]. Confirmed seropositive donors have been found in 40 states, with the highest rates in Florida (1:3800 donors) and California (1:8300 donors) [39]. The presence of T. cruzi antibodies in prospective organ donors in southern California has been documented [40]. No test is licensed yet for screening potential cadaveric donors, and the best strategies for performing screening, which may vary with location and infection prevalence, have not been defined. However, several organ procurement organizations are considering the use of commercial tests that have been approved for diagnostic use to perform selective screening of potential donors who have demographic risk factors for T. cruzi infection. There is a need for improved serologic assays for screening organ donors. High sensitivity is crucial, because organ donors have often received multiple blood transfusions before organ procurement, resulting in hemodilution of T. cruzi antibodies and a loss of sensitivity, as we observed with IFA serologic testing. High specificity is needed, to avoid discarding uninfected organs or unnecessarily subjecting recipients to lengthy and burdensome monitoring for infection. Although existing assays are not optimal, transplant recipients potentially would benefit from their use in the screening of organ donors. Even if test results are not available until after transplantation, knowledge of possible exposure to T. cruzi will allow clinicians to monitor recipients for the infection and may improve patient outcomes.

**CHAGAS DISEASE IN TRANSPLANT RECIPIENTS INVESTIGATION TEAM**


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