Seropositivity of a blood recipient from a donor with positive adult T-cell leukemia-associated antigens.

Kanji Miyamoto*  Norio Tomita†  Akio Ishii‡  Takeshi Nishizaki**  Ko-ichi Kitajima††  Toshio Tanaka‡‡

*Okayama Red Cross Blood Center,
†Okayama Red Cross Blood Center,
‡Okayama Red Cross Blood Center,
**Okayama Red Cross Blood Center,
††Okayama University,
‡‡Okayama University,
Seropositivity of a blood recipient from a donor with positive adult T-cell leukemia-associated antigens.*

Kanji Miyamoto, Norio Tomita, Akio Ishii, Takeshi Nishizaki, Ko-ichi Kitajima, and Toshio Tanaka

Abstract

A blood recipient, aged 66, was found to have positive adult T-cell leukemia-associated antigens (ATLA), approximately half a year after a transfusion. The donor’s ATLA-antibody titer was 1: 640. Routine screening of blood donors for ATLA antibody was proposed.

KEYWORDS: blood transfusion, adult T-cell leukemia virus, adult T-cell leukemia

*PMID: 6607585 [PubMed - indexed for MEDLINE]
Copyright (C) OKAYAMA UNIVERSITY MEDICAL SCHOOL
BRIEF NOTE

SEROPOSITIVITY OF A BLOOD RECIPIENT FROM A DONOR WITH POSITIVE ADULT T-CELL LEUKEMIA-ASSOCIATED ANTIGENS

Kanji Miyamoto, Noriko Tomita, Akio Ishii, Takeshi Nishizaki, Ko-ichi Kitajima* and Toshio Tanaka**

Okayama Red Cross Blood Center, Okayama 700, and *Department of Medicine (2nd Clinic), and **Pathology Section, Central Laboratories, Okayama University Medical School, Okayama 700, Japan

Received August 10, 1983

Abstract. A blood recipient, aged 66, was found to have positive adult T-cell leukemia-associated antigens (ATLA), approximately half a year after a transfusion. The donor’s ATLA-antibody titer was 1 : 640. Routine screening of blood donors for ATLA antibody was proposed.

Key words: blood transfusion, adult T-cell leukemia virus, adult T-cell leukemia

In 1982, Shimoyama et al. (1) followed by Miyoshi et al. (2) and Saxinger and Gallo (3) warned of the dangers of receiving blood from donors carrying adult T-cell leukemia-associated antigens (ATLA). In Okayama Prefecture, we recently experienced at least one blood recipient with positive ATLA, and found that the blood donor carried an unusually high titer of ATLA-antibody.

Sera and leukocytes were separated from ten ml of peripheral blood of the recipient on a Ficoll-Conray gradient. Sera were titered for ATLA-antibody by indirect immunofluorescence using KH-2 cells (4). Leukocytes were cultured with phytohemagglutinin for three days, and 50 cells in metaphase were analyzed for karyotypes using Q-bandining.

A 66-year-old female received a transfusion of 390 ml of type-A packed red cells in March 1982 at the time of radical surgery for tuberculous spondylitis. In mid-October, her ATLA-antibody titer was discovered to be 1 : 40 (normal range: less than 1 : 10) with normal karyotype; immunoglobulin then indicated high IgG (IgA 346, IgG 2200 and IgM 26). She was discharged from the hospital and has been reasonably well since. The blood donor, a 44-year-old female, had an ATLA-antibody titer of 1 : 640. Her leukocytes, cultured for 10 days with T-cell growth factor, contained 1 to 2% ATLA-positive cells when reacted by indirect immunofluorescence with two reference sera from adult T-cell leukemia (ATL) patients but not with three negative control sera.

C-type virus has been implicated as a possible pathogen for ATL (5).
C-type virus related to ATLA (ATLV) can be transmitted to blood recipients from donors with a certain titer of ATLA-antibody. According to Ōkōchi, the positive rate of ATLA-antibody titer was significantly higher in the patients who received packed red cells, as in this patient, than in those who received whole blood. Fluctuations in immunoglobulin, particularly IgG and IgM, after transfusion can forecast the positivity of ATLA-antibody titer (personal communication).

There were 22 healthy ATLA-antibody carriers in Okayama Prefecture out of 5835 individuals examined (0.4 %) (unpublished data). This figure is comparable with 2 % (2/105) in the ATL-nonendemic areas reported by Hinuma et al. (5), but much lower than 24 % (20/85) and 37 % (30/82), respectively, in Kagoshima and Nagasaki Prefectures, (both ATL-endemic areas) (6). Unfortunately, appropriate information on our patient as to ATLA-antibody titer or immunoglobulin levels prior to blood transfusion is not available. This case, however, does suggest a possible causal relationship between blood recipients acquiring ATLA-antibody and donors carrying a high titer of ATLA-antibody, because the possibility of "spontaneously positive" ATLA-antibody in Okayama Prefecture is extremely low.

To present, Shimoyama et al. (1) in the Kanto District, Ichimaru et al. (7) in the Kyushu District and Fujishita (8) in Kochi Prefecture have reported two, seven and one cases, respectively, of seroconversion after blood transfusion. In spite of the low positivity of ATLA antibody in Okayama Prefecture, this case points out the urgent necessity of routinely screening not only blood donors for the presence of ATLA antibody, but also, if possible, blood recipients before and after blood transfusion. As suggested by Tobinai et al. (9), "in ATL-nonendemic area, anti-ATLA is considered to be a very useful marker for identifying ATL and ATLV-related T-cell lymphoma".

According to Kinoshita et al. (10), retrospective analysis of stored sera from two ATLA-positive healthy adults showed the development of ATL within approximately five and ten year-intervals, respectively. "Prospective" clinical follow-up of this particular blood recipient is currently being made to check for possible progression of ATL after blood transfusion.

REFERENCES


