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Adult T-cell leukemia occurring in mother and son.

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Abstract

We report two cases of adult T-cell leukemia in which the disease developed in a mother, aged 62 years, and her son, aged 41 years, less than four months apart. Both mother and son showed abnormal karyotypes and high titers of adult T-cell leukemia-associated antibody.

KEYWORDS: adult T-cell leukemia, familial occurrence, chromosome abnormality

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— BRIEF NOTE —

ADULT T-CELL LEUKEMIA OCCURRING IN MOTHER AND SON

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Abstract. We report two cases of adult T-cell leukemia in which the disease developed in a mother, aged 62 years, and her son, aged 41 years, less than four months apart. Both mother and son showed abnormal karyotypes and high titers of adult T-cell leukemia-associated antibody.

Key words : adult T-cell leukemia, familial occurrence, chromosome abnormality.

Adult T-cell leukemia (ATL) has a high incidence particularly among the Japanese, and has occurred in brothers (1), sisters (2), and brother and sister (3) indicating a familital tendency. No report, however, has been made concerning the occurrence in parents and their offspring. We report here two cases of ATL in which the desease developed in a mother and her son less than four months apart.

In late April 1981, a 62-year-old female started complaining of epigastralgia and was diagnosed as having multiple gastric ulcers. In the middle of September of the same year, she developed fever, cough and superficial lymphadenopathy ; the peripheral blood picture then indicated ATL. Laboratory data on admission to a hospital showed a white blood cell count (WBC) of 1.97×10^4 per ml, in which 70 % of the leukocytes were abnormally lobulated. The nucleated cell count (NCC) in the bone marrow was 6.17×10^4 per ml, of which 40.6 % were abnormal lymphocytes. Peripheral blood lymphocytes showed positive En-rosette formation, *i.e.*, the rosette formation with neuraminidase-treated sheep red cells, in 78.5 %, and their surface phenotypes consisted of OKT3, OKT4, OKT11 and Tac. ATL-associated antibody titer was $\times$ 80, and the karyotype in the peripheral blood was 50 % each of 46, XX and 46, XX, 6q-. The patient died of an infection in December of the same year.

The 41-year-old son of the patient described above had occacionally suffered from chronic bronchitis. Around December 1981, WBC in the peripheral blood

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were found to be 1.1×10^4 per ml, in which abnormal lympocytes consisted of 64 % and were characteristic of ATL. NCC in the bone marrow was 7.4×10^4 per ml with a few percent of abnormal lymphocytes ; En-rosette formation was 81.5 % ; ATL-associated antibody titer was $\times$ 160, and the karyotype was 46XY. He manifested no particular symptoms until late October 1982, when the patient developed fever and a thumb-sized lymph node swelling in the right side of the neck ; these, however, subsided spontaneously. The WBC (1.11×10^4 per ml) then showed 73 % abnormal lymphocytes, and the karyotype became abnormal. The modal chromosome number was near diploid, ranging from 45 to 47 ; 40 out of 50 cells had 46 chromosomes. Nine of 22 cells examined (41 %) were abnormal in karyotype with the following abnormalities : 46, XY, 3q-, 6q-, +18p+, -20.

Both mother and son developed a partial deletion of the long arm of chromosome 6, *i.e.*, 6q-, which is one of most common chromosome abnormalities in ATL (4). They had lived together for more than 40 years. The son developed an abnormal karyotype within 10 months of finding abnormal lymphcytes in December 1981. The higher percentage of cells with a normal karyotype than those with an abnormal one as determined in October 1982 suggests that the patient's prognosis is favorable (4). The possibility of C-type virus particles as an oncogen for ATL has been reported (5). The present cases may indicate a "vertical transmission" of oncogenic factor(s) during the son's fetal life, or, alternatively, the son became "genetically susceptible to the oncogenic factors" (1) because of an immature state of immunosurveillance in his early post-natal life or in his adult life.

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