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Accepted: February 17, 1986

Acta haemat. 1986;75:122

T Activation in a Patient with T Cell Chronic Lymphatic Leukemia

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T polyagglutination of red cells is known to have been caused in vivo and in vitro by various microorganisms which produce the enzyme neuraminidase [1]. T receptors are also exposed on malignant tissue [2]. Tn polyagglutination has been demonstrated in only 4 cases of AML [3], but no cases of T polyagglutination have been described in patients with hemato-poietic malignancies.

A patient with chronic lymphocytic leukemia (CLL) and T antigen exposure is described here. An 84-year-old woman of Roumanian Jewish origin was diagnosed in April 1983 as suffering from T cell CLL stage IV (Rai). In January 1984, she was hospitalized with fever, chills and septic arthritis of the right knee. Cultures from the blood and fluid aspirated from the right knee grew *Escherichia coli* 07: H(-). The patient was treated with appropriate antibiotics and responded well. Repeated cultures from blood and knee aspirations were negative. The patient's blood was screened with lectins for polyagglutination as part of a study in patients with malignancy and sepsis. T antigen exposure and possible T polyagglutination was diagnosed. This finding has persisted in repeated examination of blood samples over a period of 7 months with no evidence of hemolysis.

The patient's red blood cells were examined with a lectin panel and they agglutinated with: *Arachis hypogaea*, *Glycine soja*, and *Vicia cratica*. They did not

Table I. Reaction of patient's red blood cells with lectins

Date Lectins and strength of polyaggluti- Type of poly-
nation agglutination

Arachis *Glycine soja* *Vicia cratica* *hypogaea*

agglutinate with *Salvia horminum*, *Salvia sclarea*, *Bandeiraea simplicifolia* (BS) I, BSII, or *Hibiscus sab-dariffa* (Sorrel). These results are compatible with exposure of the T antigen (see table I). The patient's red cells were not agglutinated by 3 samples of AB cord sera. They aggregated with Polybrene. Direct and indirect antiglobulin tests were negative. *E. coli* produces neuraminidase and can cause T polyagglutination. The supernatant of the cultures of *E. coli* 07:H(-) which was grown from the blood cultures and knee aspirate did not cause T antigen receptor exposure when incubated with normal red blood cells, incubated according to a known method (J.J. Moulds, Gamma Biologicals Inc., personal communication). One would have

expected the potential polyagglutination to disappear with the resolution of the infection, but the T antigen exposure persisted and even became stronger during the 7 months of follow-up despite repeated negative blood and aspirate cultures. The other possible cause of this phenomenon is the malignancy, the CLL. This could suggest a direct connection between the CLL and the T antigen exposure in this case, a finding which to the best of our knowledge has not been described before in the literature.

References

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4+

weak T1

2–3 +

weak

2–3 +

weak

2 +

weak

+

weak

Confirmed by G.W.G. Bird, Birmingham, England.

Accepted: February 17, 1986