

Normal Serum Adenosine Deaminase Activity in Mycosis Fungoides

Sir,

Adenosine deaminase (ADA) is an enzyme that catalyzes hydrolytic and irreversible deamination of deoxyadenosine into deoxyinosine and of adenosine into inosine (1). The activity of ADA is very high in lymphocytes, especially in immature and undifferentiated T-lymphocytes. Therefore, some authors consider ADA as a marker of cell-mediated immunity. Some studies have reported an increased ADA activity in lymphocytic tissues and leukemic cells, especially tumours of T-cell origin (2).

The aim of this study was to investigate serum ADA activity in patients with mycosis fungoides (MF) at different stages and the significance of serum ADA activity in determining the course of the disease.

MATERIALS AND METHOD

A total of 25 patients with MF (11 males, 14 females), aged between 18–83 years (median 41), were included. Of these, 3 were in the tumoural, 8 in the plaque and 14 in the patchy stage. All patients were newly diagnosed and neither systemic chemotherapy nor radiation therapy had been administered to the patients. Visceral involvement and Sézary syndrome were not detected clinically or with laboratory studies. Twenty-five sex- and age-matched healthy subjects were included as a control group.

ADA assay

Venous blood of about 2 ml was drawn for ADA estimation, and after centrifugation the serum was stored at -20°C . ADA activity

was measured in serum samples within 10 days, according to the colorimetric method described by Giusti (3).

The Mann Whitney U-test and Students' *t*-test were used for statistical analysis, as appropriate.

RESULTS

The serum ADA level (mean 7.5 IU/ml) was not significantly different in the control group (mean 7.0 IU/ml) and in the patients in the tumoural, plaque and patchy stages; the mean was 8.0 IU/ml, 7.5 IU/ml and 7.4 IU/ml, respectively.

DISCUSSION

Koizumi & Ohkawara reported an increase in ADA activity in the sera of patients with MF and adult T-cell leukemia (4). The results in the present study conflict with this study. No difference was found between the patients at different stages of MF and the control group. The normal range of ADA in the serum in our laboratory is 5–20 IU/ml. In the present study no patient had a serum ADA activity above this limit. As none of the patients presented with Sézary syndrome in our study, the results we obtained are consistent with the natural course of the disease. Since MF is a primary lymphoma of the skin, originating from helper T-cells, circulating atypical lymphocytes are not generally encountered and serum lymphocyte count alterations are not usually observed. This may be

the reason for the normal ADA activity in the study group. The high ADA activity in the serum is explained by these authors as the result of a leakage of ADA to the systemic circulation, without any discrete evidence. In conclusion, our study clearly demonstrates that measurement of serum ADA activity is not a diagnostic or prognostic marker in the follow-up of patients with MF without any visceral involvement or Sézary syndrome.

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Response to the Letter by B. Yalçın et al.

Sir,

Adenosine deaminase is one of the key enzymes in purine nucleotide degradation. This enzyme exists in most of the human tissues and the activity is high in lymphatic tissues, especially in T-lymphocytes. Elevated adenosine deaminase activity in T-cell leukemia has been reported, and its inhibitor, deoxycoformycin, has been developed as an anti-tumor agent. In some types of leukemia, serum adenosine deaminase activity increases in accordance with the severity of the disease. Although mycosis fungoides rarely involves peripheral blood, tumor cells do invade the skin. An elevated adenosine deaminase activity was observed in 7 of the 8 patients with mycosis fungoides (1). Thus, in order to evaluate the clinical significance of adenosine deaminase in mycosis fungoides, adenosine deaminase activity was measured in sera of 15 patients with mycosis fungoides at various stages (2). The adenosine deaminase activity in the sera of 299 healthy humans was 10.59 + 3.19 IU/l (mean + 1 S.D.) (range 5.3–17.8). The mean adenosine deaminase activity in the sera of the patients in the plaque stage (T2N0M0, IB), was as high as 19.0 IU/l (range 13.7–21.4), with statistical significance compared with healthy controls ($p < 0.001$). Three tumor stage patients without visceral involvement (T3N0M0, IIB) showed higher levels of adenosine deaminase activity (19.7, 21.5, 24.4 IU/l). An erythrodermic patient (T4N0M0, III), who did not have

Sézary syndrom, also had a high adenosine deaminase activity, 28.4 IU/l. Two tumor stage patients with organ involvement (T3N0M1, IVB) exhibited an extremely high adenosine deaminase activity (60.9, 32.2 IU/l). The enzyme activity in the plaque stage was from within the normal range to a slightly higher level. The data of Dr. Yalçın, which showed no difference between patient and control group, might be due to the sensitivity of the measurement. The adenosine deaminase activity in sera showed a tendency to become higher with the extension of the stages. From the results obtained, it is suggested that serum adenosine deaminase activity may reflect the tumor progress in mycosis fungoides.

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