ERYTHROCYTE ADENOSINE-DEAMINASE ACTIVITY IN GOUT AND HYPERURICEMIA

A. Carcassi, P. Macri, G. Chiaroni, S. Boschi

Institute of Clinical Medicine, University of Siena
Piazza Selva 7 53.100 Siena Italy

Adenosine-Deaminase (adenosine-aminohydrolase EC 3.5.4.4.) (ADA) catalyses the irreversible hydrolytic deamination of adenosine to produce inosine and ammonia. Inosine is then cleaved by Purine-Nucleoside-Phosphorylase (PNP) to hypoxanthine and oxidized to xanthine and uric acid by xanthine-oxidase.

ADA is an enzyme present in most mammalian tissues the activity being highest in organs containing many lymphoid cells (1,2). In erythrocytes ADA activity is present but lower of that observed in lymphoid cells.

ADA deficiency has been described in a group of patients with the autosomal recessive form of severe combined immunodeficiency (SCID), a disorder of infancy characterized by defects of both cellular and humoral immunity, with severe impairment of T and B lymphocytes (3).

Although the main involvement of this enzyme in man appears to be related to immune system, ADA activity can also be related to the catabolism of AMP, via the 5′-Nucleotidase pathway, alternatively with AMP-Deaminase, that is its main catabolic pathway (4).

The importance of this catabolic pathway in the production of uric acid in primary gout has not been proved, even if increased ADA activity in erythrocytes has already been reported in patients with primary gout (5).
To verify these data we have studied erythrocyte ADA activity in a group of patients with primary gout, in subjects with primary asymptomatic hyperuricemia and in patients with hyperuricemia secondary to chronic renal failure (on hemodialysis treatment).

MATERIALS AND METHODS

Studies were carried out on: 20 control subjects (15 males and 5 females), 16 patients with primary gout, 8 subjects with primary asymptomatic hyperuricemia and 10 patients with hyperuricemia secondary to chronic renal failure (on hemodialysis treatment) (6 males and 4 females).

Fructose load (0.5 g/Kg b.w./10 min') was administered intravenously to 5 control subjects and to 4 patients with primary hyperuricemia (all males). Erythrocyte ADA activity was studied before and 30, 60 and 120 min' following intravenous load.

All subjects were fasting for 12 hours. All gouty patients did not receive any drug affecting uric acid metabolism for at least 15 days.

Serum uric acid was determined by the method of Archibald (6).

ADA activity was determined by the method of Seligson and Seligson (7), and ammonia was evaluated with the colorimetric method of Chaney and Marbach (8).

RESULTS

The results obtained are reported in Tables I and II and in Figures 1 and 2.

Gouty patients and the subjects with the primary asymptomatic hyperuricemia showed slightly higher values in erythrocyte ADA activity than control subjects. The difference was not significant (p > 0.05).

No definite changes in ADA activity were observed in patients with hyperuricemia secondary to chronic renal failure (on hemodialysis treatment).