Original Article

Adenosine Deaminase (ADA) in Rheumatoid Arthritis in patients (RA)

Huda Th. Al-Marsomi* **PhD**

Summary:

Background: Rheumatoid Arthritis (RA) is heterogenous syndrome. Because the diversity of disease processes and formation of complex lymphoid microstructures that indicate the multiple T cell activation pathways are involved .affected patients have major abnormalities in the T cell pool with clonally expanded CD_4^+ T cell that lose expression of the $CD28^{null}$ molecule and lack the ability for profiliration. Adenosine deaminase (ADA) is an indicator of the proliferation and differenation of lymphocyte, in particularly the T cell subcells.

Patients and Methods: Total ADA levels were measured in the sera of RA patients and healthy group according to Giusti (1981).

Results: The mean value of ADA was lower in patients with RA than control group with no significant differences.

Conclusion: the lower value of ADA (which involved in the proliferation of lymphocyte) in RA patients may results from the predominance of CD_4^+ T cells in the peripheral blood

Fac Med Baghdad
2009 Vol. 51 No. 2
Received June 2008
Accepted Jan. 2009

Key words: RA, ADA, IR.

Introduction:

Rheumatoid Arthritis (RA) is a sever chronic inflammatory auto-immune disorder of mysterious etiology, characterized by inflammation of synovial membrane, principally affecting peripheral joints in asymmetric fashion , extra-articular manifestations also occur, so RA is a disease of an aberrant immunresponse (IR) in a genetically predisposed host, Both humoral and cellular IR are important in this disease(1,2).In RA patients have abnormalities in T cell function that are not restricted to the T cell participating in the synovial infiltrates . One aberration is the expansion of selected CD_4 T cell to large colonel population. In patients with sever RA, the $CD_4^+CD28^{null}$ T cells were initially identified(3,4) .Adenosine deaminase (ADA) (Adenosine aminohydrolase, EC (3.5.4.4)) is the enzyme that irreversibly catalyzes the deamination of adenosine and deoxyadenosine to inosine and deoxyinosine respectively and ammonia(5) . ADA is involved in the proliferation and differenation of lymphocyte, particularly the Tcell subtype, which was found to play a crucial role in the metabolism of the immune system cells and it is essential for the proper development of both T and B lymphocyte in mammals(6).

The aim of this study is to investigate the ADA level among RA patients as indicator of IR.

Patients and Methods:

The study included two groups:

a. Rheumatoid Arthritis (RA) patients: Blood samples were collected from thirty patients, their ages ranged from (30-60) years who were attending to AL – Kadhmia teaching hospital from march to august in 2005 and diagnosed by doctors to be infected with RA(before any treatment),all patients have symptoms for two to six months.

b .Healthy control group: fifteen individuals from blood bank donors, who have no history of clinical evidence of RA or other clinical disease.

Sera were separated and stored at -20 ºC until use. Total ADA levels were measured in the serum of each patient by the method described by Giusti (1981) (7). The method is based on measuring the rate of ammonia consumption at 620 nm following the reaction.

Results:

As shown in table 1 results indicated that their were no significant differences between RA patients (14.63±2.4 U/L) and control group (18.9± 2.15 U/L).

**Department of Microbiology, College of Medicine, Al-Nahrin University.*

Table-1: Mean± SD of serum adenosine deaminase (ADA) level in U/L among RA patients and control group.

Group	Mean ±SD
RA patients	14.63±2.4/L
Control group	18.9±2.15U/L

t=3.09, P<0.05, SD=Standard deviation

Discussion:

RA is the most common inflammatory arthritis, affecting about 1% of the general population world wide (8) . Pathogenesis of RA is till not fully understood , There is evidence that CD_4^+T cells play a central role in initiating , perpetuating and precipitating chronic inflammation in synovial tissue(1,9). Another role of activated CD_4^+T cells is stimulation of B cells to differentiate into plasma cells producing RF (Rheumatoid Factor) and other autoantibodies(2,10). ADA enzyme is one of the most essential immune enzymes. It is function gives a clear picture of the immune status of the body. It was found to play a critical role in proper development of the T- and B-lymphocytes in mammals(10,11). In this study, no significant difference was found regarding the mean value of serum ADA among RA patients when compared with the control group and this may be because those in RA patients had marked difficulties in repopulating the T cell compartment. In RA patients, Peripheral CD4+T cells counts remained depressed to lymphopenic levels for an extended period also T cell have a limited prolifirative life span and chronic immune activation could lead to accumulation of clonally expanded senescents cell (CD4+CD28null) which generally characterized by a limited or complete lack of proliferation that agreement with our resultas as that ADA a marker for cell proliferation. It will be of interest to measure ADA level in synovial tissue before and after treatment and compare it with ADA level in serum to get more information about association between the immune response and ADA level as parameter to response to treatment .

References:

1. Anderson, R.J; Rheumatoid arthritis, Clinical and laboratory Features.In: Keppel, J.H; Crofford, L.J; Ston, J.H.et al .Primer on the Rheumatic Disease.12th edit.Atlanta Georgia-Arthritis Foundation .2001:218-225.

2. Helen, ch. ,Mansel, H. ,Siraj, M. and Neil, S.:Joints and muscles. In: Essentials of clinical Immunology 5th ed.Blackwell publishing.U.S.A.;2006:p.180-183.

3. Abbe,N.V., Eduardo,D., Cornelia,M.W. and Jorg,J.G. :Biology of T lymphocytes. Rheum Dis Clin N Am ,2004;(30) 135-157.

4. Cornella,M.W.,Bryl,E.,Jorg, J.G.; The role of T cells in Rheumatoid Arthritis. Archivum immunologiae et therapiexperimentalis,2000;48:429-435 .

5.Dinjens,W.N.M., Tenkate,J. and Bosman,F.T.:Distribution of adenosine deaminase complexing protein (ADCP) in human tissue. J Histochem Cytochem ,1989;(37)1869-1875.

6.Ammann, A.J.:Immunodeficiency disease. In : Stites, D.P.; Stobo, J.and wells, J.V.editors. Basic and Clinical Immunology.6th ed. USA Appleton&Lange; 1987; p.339-341.

7. Giusti, G.:Adenosine deaminase.In: Bergmeyer, H.V. Ed. Methods of Enzymatic Analysis.2nd ed . Florid: Verlag Chemise International; 1981.2 p.1092-1099.

8. Al-Rawi, Z.S., AL-Azzawi, AJ., Al-Ajili, F.M., Al-WakilR.:Rheumatiod arthritis in population samples in Iraq. Ann Rheum Dis,1978; 37:73-75.

9.Kathleen ,B.and Mason,I,:Pathomechanisms in rheumatoid arthritis-time for a string theory? .The Journal of Clinical Investigation ,2006 ;116(4):869-871.

10. AL-Ubaide, A.H.; AL-Jeboori, T.I. &Juma, A, S.ADAlevel in patients with hydatid disease (Echinococcus granulosis).Iraqi J Med Sci, 2003; 2(1):25-28.

11.Juma, A., AL-Jeboori, T.I., Tawfiq, M.S. &Fadhil, R.S.:ADA Activity in the serum of patients with Schistosoma haematobium & those with bladder carcinoma. Iraqi J Med Sci, 2003; 2(3):24-28.