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RESEARCH ARTICLE

ADA AND LDH LEVELS IN INFLAMMATORY AND NON-INFLAMMATORY JOINT DISEASES

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Key words:

ADA, LDH, Inflammatory, Non-Inflammatory, Synovial Fluid and Arthritis Aim of present study was to compare the adenosine deaminase (ADA) activity and Lactic Dehydrogenase (LDH) in various types of arthritis conditions with synovial effusion. No. significant difference was observed in ADA activity in synovial fluid of control and study groups in various arthritic conditions. The highest value of ADA activity and LDH was observed in synovial fluid of patients with tubercular arthritis followed by rheumatoid, septic, osteo and post traumatic arthritis. Thus measurement of ADA activity and LDH in synovial fluid can be used as a parameter of differential diagnosis of arthritis and monitoring the disease activity.

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INTRODUCTION

A comprehensive diagnostic approach is essential for early diagnosis of Arthritis and prompt management to prevent physical disability. Synovial fluid analysis helps in this aspect. Analysis of synovial fluid (SF) has long been recommended as a routine procedure to assist in the differential diagnosis of arthritis. Available synovial fluid biochemical tests like glucose, protein have limited value and lack specificity in differentiating inflammatory (INF) from non inflammatory arthritis (NON INF) Gyorgy Hasko and Bruce, 2004. Adenosine deaminase (ADA) and Lactic dehydrogenase (LDH) of the SF are among the important biomarkers in recent studies in differentiating INF from NON-INF arthritis. Studies have reported that SF ADA was higher in INF arthritis when compared to non INF arthritis. ADA and CRP level are indicators (Gyorgy Hasko and Bruce, 2004) of immune system activation. Aim of present study IS to estimate ADA, LDH levels in synovial fluid obtained from patients with arthritis and to assess the value of the investigation in differentiating between INF and NON-INF arthritis.

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MATERIALS AND METHODS

A total of 50 patients with knee arthritis with joint effusion who were admitted in King George Hospital and Andhra Medical College, Visakhapatnam during the period of July 2012 to March 2014 have been included in this study. The present study comprises 50 cases of Arthritis and 25 healthy age matched controls. No specific reference was made with reference to sex of the patient.

The Mean, SD, SEM of age of control versus test groups are:

 $58.8 \pm 2.97 \; Vs \; 53.83 \pm 4.65$

Synovial fluid was obtained from affected Knee joints of patients. The fluid is Centrifuged and cells are separated and the supernatant fluid is collected for analysis (Buhl and Jackson, 1970).

ADA and LDH levels are found to be higher in synovial fluid of cases with INF arthritis than those obtained in healthy individuals.

ADA: $25.346 \pm 5.761 \pm 1.1523$ Vs $4.936 \pm 1.292 \pm 0.2637$ LDH: $461.69 \pm 68.157 \pm 13.6314$ Vs $221.4 \pm 42.75 \pm 8.726$

Table 1. Categorization of patients into various groups

S. No	TYPES OF CASES	NO. OF PERSONS
1	Total No. of Patients	50
2	Total No. of controls	25
3	Patients/Cases with INF Arthritis	26
4	Patients/Cases with Non-INF Arthritis	24

Table 2. Various parameter values Mean, SD & SEM of control group

S.NO	VARIABLES	MEAN	SD	SEM
1	ADA	4.936	±1.292	±0.2637
2	LDH	221.4	±42.75	±8.726

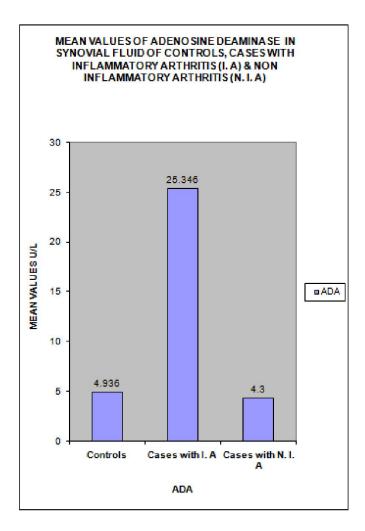
 Table 3. Different parameters values- Mean, SD, SEM and p

 Values of test group i.e. INF arthritis group

S.NO	VARIABLES	MEAN	SD	SEM	P - VALUE
1	ADA	25.346	±5.761	±1.1523	< 0.001
2	LDH	461.69	± 68.157	± 13.6314	< 0.001

Table 4. Study of different parameters mean, SD, SEM and pvalues in synovial fluid of NON-INF arthritis

S. NO	VARIABLES	MEAN	SD	SEM	P - VALUE
1	ADA	4.3	±0.66	±0.1376	< 0.020
2	LDH	315.29	±56.1724	±11.7127	< 0.001



ADA levels of non – inflammatory arthritis are found to be comparable in to that of healthy individuals. ADA: $4.30 \pm 0.66 \pm 0.1376$ vs. $4.936 \pm 1.292 \pm 0.2637$ LDH levels are found to be higher in Synovial Fluid of cases with non – inflammatory arthritis as compared with that of normal healthy individuals. LDH: $315.29 \pm 56.1724 \pm 11.7127$ vs. $221.4 \pm 42.75 \pm 8.726$

DISCUSSION

The present study included fifty (50) cases of Arthritis, out of which 26 patients are with INF type of arthritis, 24 patients are with NON-INF type of arthritis and 25 healthy age matched controls.

The possible mechanism for increased levels of ADA

It indicates the local release of ADA by cells within the points. Adenosine is an endogenous nucleoside released by a variety of normal, stimulated and injured cell types. It is a normal constituent of blood, where it has been detected at concentrations well below the concentrations in RA Synovial fluids, which is due to its rapid uptake by blood cells, primarily that, in the extra vascular compartment, i.e. in the absence of red cells, the rate of adenosine uptake could be slower and consequently the concentration of the autacoids could be higher. ADA is an enzyme involved in purine metabolism. It is needed for the breakdown of Adenosine from food and from the turnover of nucleic acids in tissues.

Adenosine cannot be deribosylated (removed from ribose) without first converting it to the related nucleoside inosine, which is then broken down further ADA converts Adenosine to inosine by the removal of an amino group. Pacheo R, Martine Z - Navio et al., studied, by a 3-fold reduction in the EC (50) for the antigen, ADA potentiated T cell proliferation in autologous co cultures with antigen-pulsed immature or mature dendritic cells. Costimulation was not due to the enzymatic activity but to the interaction of ADA-CD26 complexes in T cells with an ADA anchoring protein in dendritic cells. From co localizing with adenosine receptors on dendritic cells interact with CD26 expressed on lymphocytes. This co stimulatory signal in the immunological synapse leads to a marked increase (3 to 34 fold) in the production of T helper and pro inflammatory cytokines IFN-gamma, TNFalpha and IL-6. Increased LDH activity was observed in INF arthritis when compared to NON-INF arthritis. The synovial fluid activity of LDH is corresponding to disease activity of INF arthritis (Pejovic et al., 1992).

Increased LDH levels in INF arthritis as compared with that of control group: LDH: $461.69 \pm 68.157 \pm 13.6314$ Vs $221.4 \pm 42.75 \pm 8.726$ Increased Adenosine deaminase (ADA) enzyme levels in Synovial fluid of INF arthritis as compared with that of NON-INF arthritis.

ADA:25.346 \pm 5.761 \pm 1.1523 vs. 4.30 \pm 0.66 \pm 0.1376.

Conclusion

ADA activity was found significantly high in the synovial fluid of INF arthritis when compared to controls though the values are less raised in NON-INF arthritis. Synovial fluid did not show any increased ADA activity in synovial fluid of osteoarthritis (Yuksel and Akoglu, 1988). There is increased activity of ADA in in synovial fluid in INF arthritis when compared to NON-INF arthritis.

ADA activity in the synovial fluid is correlated with disease activity as measured by erythrocyte sedimentation rate.ADA activity may provide additional measure of degree of inflammation in joint diseases (Pettersson et al., 1988). Aim of present study was to compare the adenosine deaminase (ADA) activity in various types of arthritis conditions with synovial effusion. No. significant difference was observed in ADA activity in serum of control and study groups but results have shown a definite pattern of ADA activity in synovial fluid in various arthritic conditions. The highest value of ADA activity was observed in synovial fluid of patients with INF arthritis which include tubercular, rheumatoid, septic, osteo and post traumatic arthritis. Thus measurement of ADA activity in synovial fluid can be used as a parameter of differential diagnosis of arthritis especially tubercular in initial stages (Kumar, et al., 1994).

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