

Study of Serum Adenosine Deaminase Levels in FNAC Confirmed Cases of Tuberculous Lymphadenitis in South Karnataka Population – Retrospective Study

Jaya Kumar CK¹, Nandini GV², Manoj Kumar M³

¹Associate Professor, Department of Pathology, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur, Karnataka, India

²Associate Professor, Department of Pathology, Shri Siddhartha Institute of Medical Sciences and Research Centre, T Begur, Nelmangala, Bangalore Rural, Karnataka, India

³Assistant Professor, Department of Pathology, Shri Siddhartha Institute of Medical Sciences and Research Centre, T Begur, Nelmangala, Bangalore Rural, Karnataka, India

Received: 01-12-2025 / Revised: 15-01-2026 / Accepted: 21-02-2026

Corresponding author: Dr. Nandini GV

Conflict of interest: Nil

Abstract

Background: Tuberculosis is quite a common disease in underdeveloped countries like India. Hence, FNAC is an easy diagnostic tool to confirm tuberculous lymphadenitis and to determine sensitivity and specificity.

Method: 70 adults diagnosed with tuberculous lymphadenitis by FNAC, followed by measurement of elevated serum adenosine deaminase levels, were recorded.

Results: Out of 70 patients, 80% had granulomatous lymphadenitis and 20% had granulomas/20 HPE. The adenosine deaminase levels ranged between 31 to 40 IU/L.

Conclusion: The FNAC study of adenosine deaminase elevation is a confirmation of tuberculosis in the suspected enlargement of lymph nodes.

Keywords: serum adenosine deaminase, FNAC, lymphadenitis, undernutrition, AFB staining.

DOI: 10.25258/ijcpr.18.3.79

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Tuberculosis bacillus is one of the leading causes of morbidity and mortality in underdeveloped countries like India, Bangladesh, and Nepal. As per the WHO, approximately 16 million people are suffering from active TB globally, and 2 million mortalities are reported every year [1].

Tuberculosis is a social disease with medical aspects. It has been described as a barometer of social welfare. The social factors of poor quality of life (i.e., slum dwellers), under nutrition, and lack of awareness of causes of illness are implicated in the causation of tuberculosis [2]. Tuberculosis usually affects lungs, but extrapulmonary tuberculosis lymphadenitis is confirmed routinely by FNAC and AFB staining. Adenosine deaminase is an enzyme required for converting adenosine to inosine in the purine salvage pathway [3]. Its activity is involved in the differentiation and proliferation of lymphocytes and activation of macrophages. This enzyme is important in the rapid proliferation of cells to prevent the accumulation of toxic metabolite. ADA activity increases during cellular activation to detoxify the toxic metabolite;

hence, elevated ADA indicates severity of tuberculous disease [4]. Hence, an attempt was made to evaluate the ADA levels in the enlarged lymph nodes in TB patients to study the severity of disease.

Materials and Methods

70 (seventy) adult patients who visited the pathology department of Shridevi Institute of Medical Sciences and Research Hospital, Tumkur, Karnataka-572106 were studied.

Inclusion Criteria: Suspected tuberculosis patients with enlarged lymph nodes referred by the medicine department for FNAC, or patients on antitubercular therapy with peripheral lymphadenopathy. The patients who gave their consent in writing for serum adenosine deaminase estimation were selected for study.

Exclusion Criteria: The patients diagnosed with infectious mononucleosis, enteric fever, leprosy, hepatitis A & B, chickenpox, and hematopoietic malignancy were excluded.

Method: A complete history and socioeconomic status were studied in every patient. Fine needle aspiration cytology smears for the features of granulomas, multinucleated giant cells, and AFB, which suggest tubercular lymphadenitis, and serum adenosine deaminase levels were carried out by the calorimetric method.

The patients with elevated serum ADA and reactive FNAC lymphadenitis were subjected to lymph node biopsy for histopathological examination.

The duration of the study was from July 2022 to February 2024.

Statistical Analysis: Distribution of FNAC types of reactive patients' tubercular patterns, granuloma/20 HPE patients, and ADA levels were classified with percentage, and p value ($p < 0.001$) was recorded in significant studies. The statistical analysis was carried out using SPSS software. The ratio of males and females was 2:1.

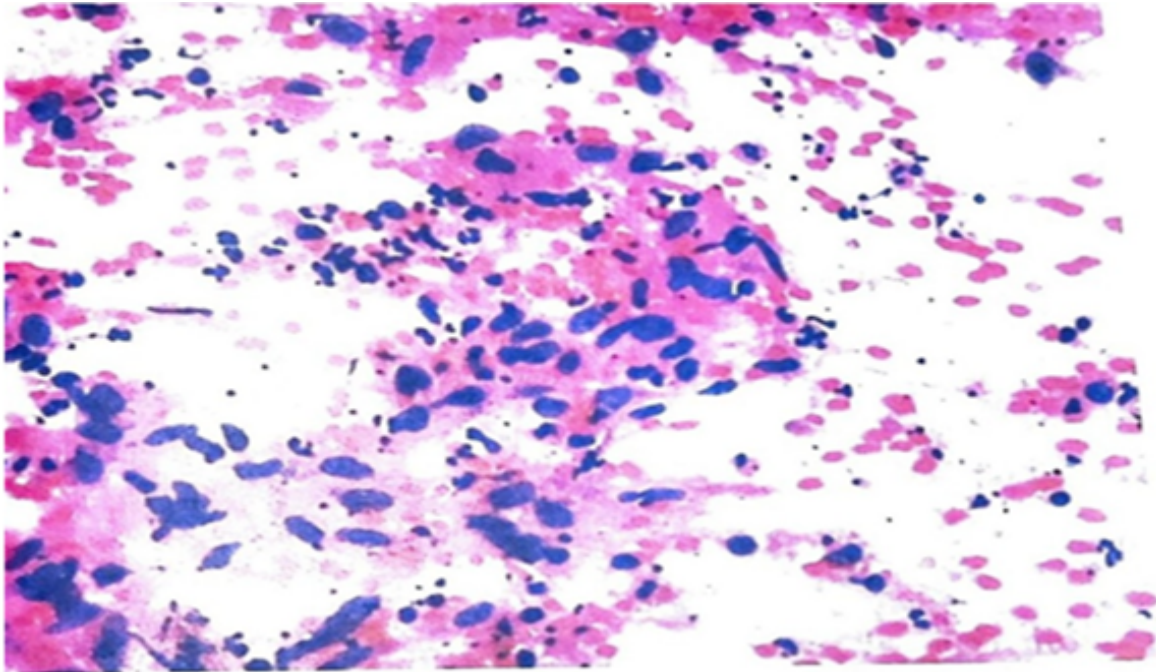


Figure-1: Photo micro graph showing epithelioid granuloma 40x(H&E)

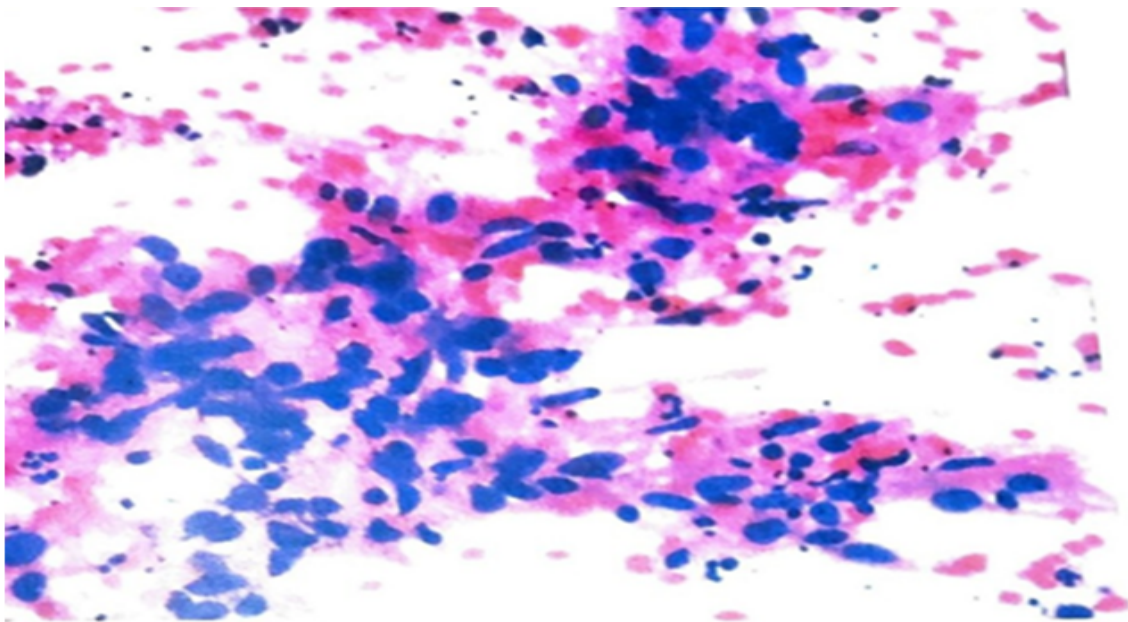


Figure-2: Photo micrograph showing the old granuloma; 40x (H&E)

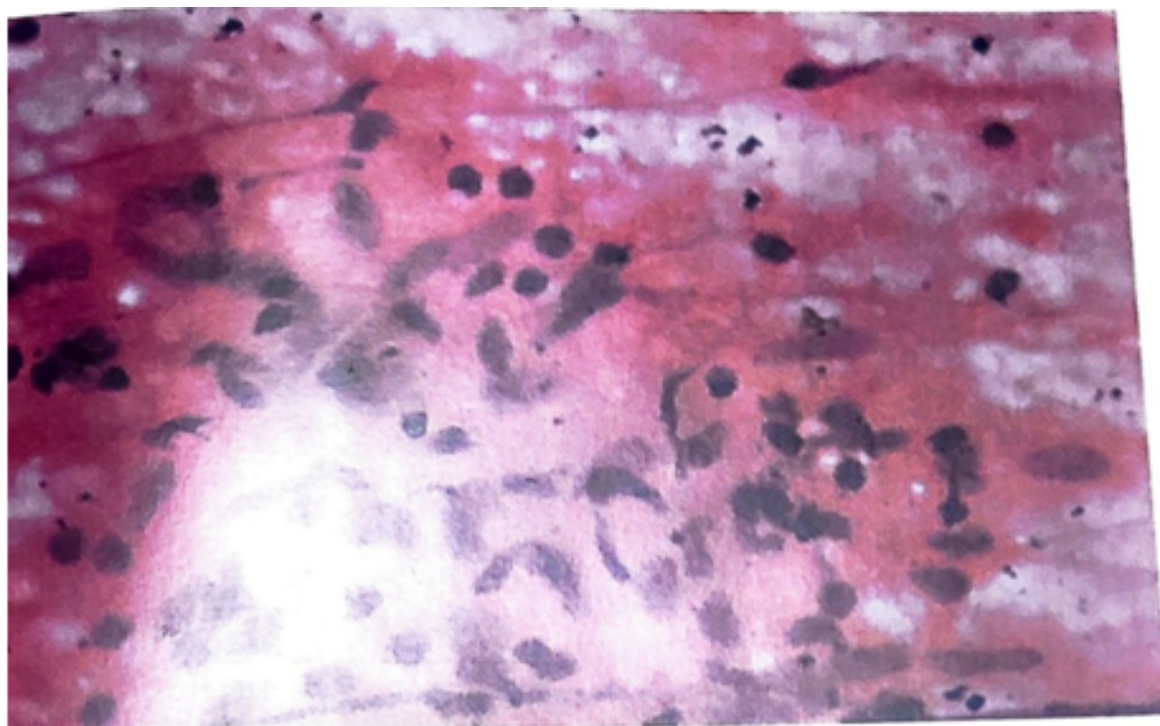


Figure-3: Photo micrograph showing epithelioid granuloma;40x(H&E)

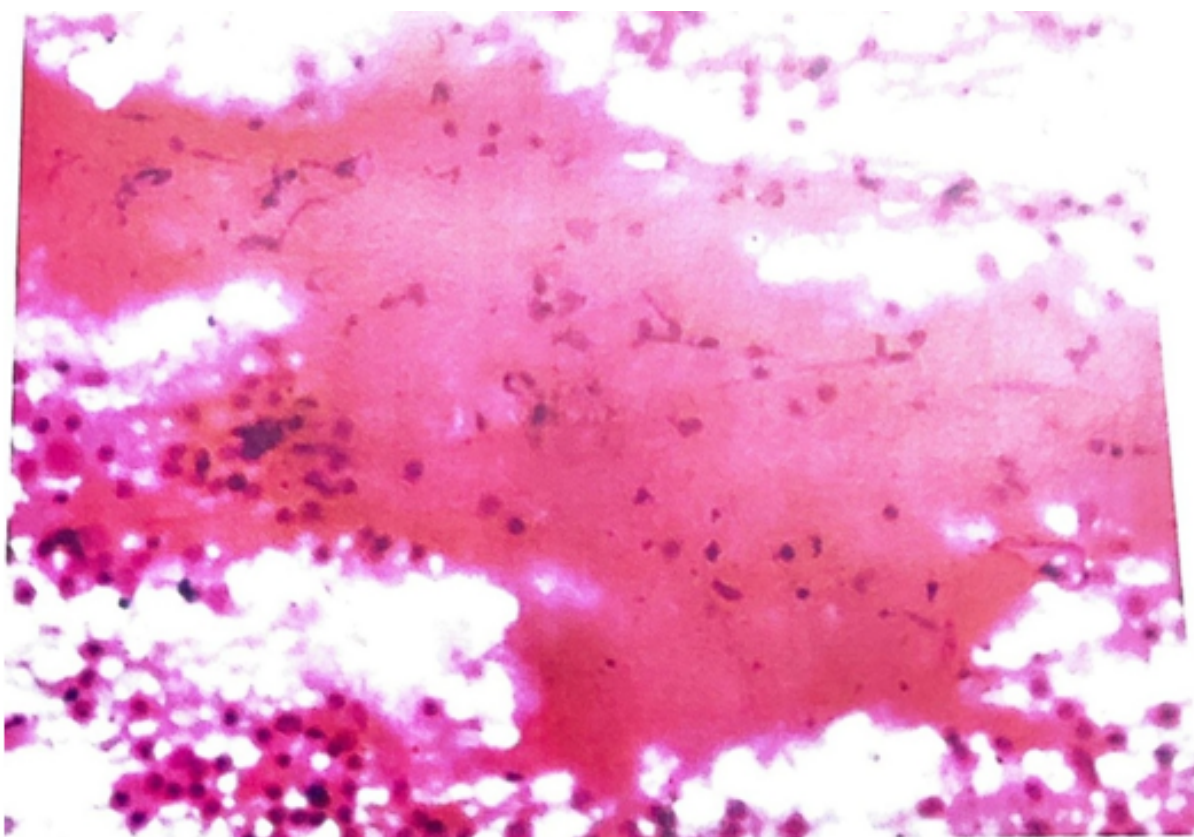


Figure-4:Photo micrograph showing caseous necrosis; 20x(H&E)

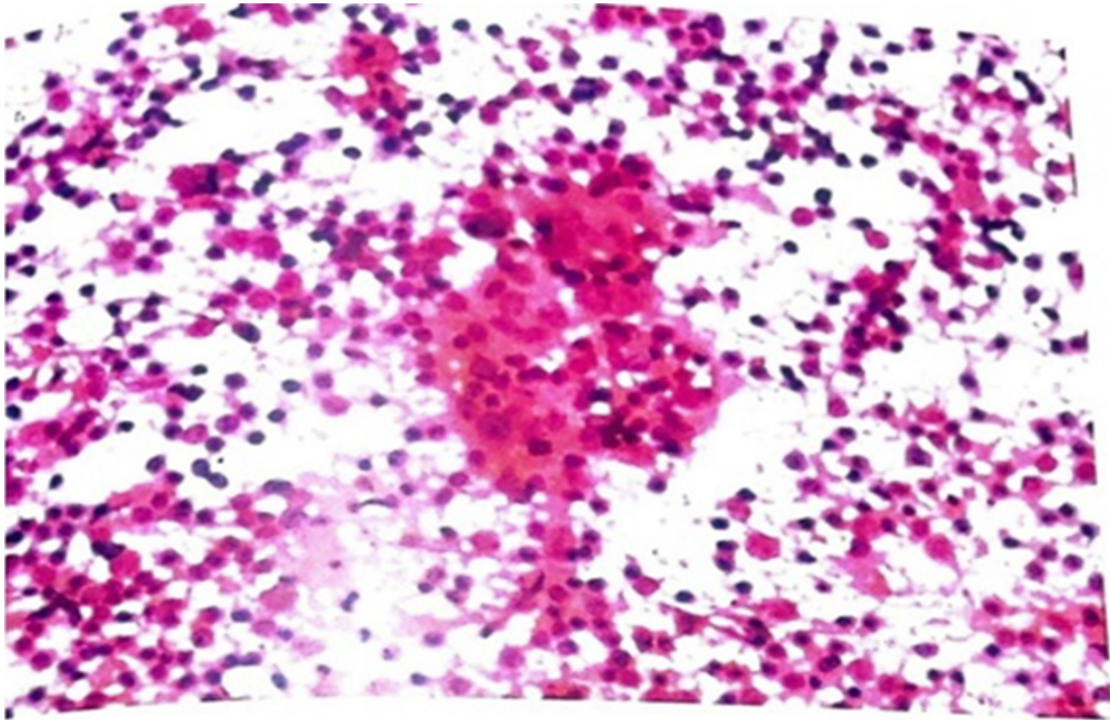


Figure-5: Photo micrograph showing Histiocytic aggregate with back ground 40x (H&E)

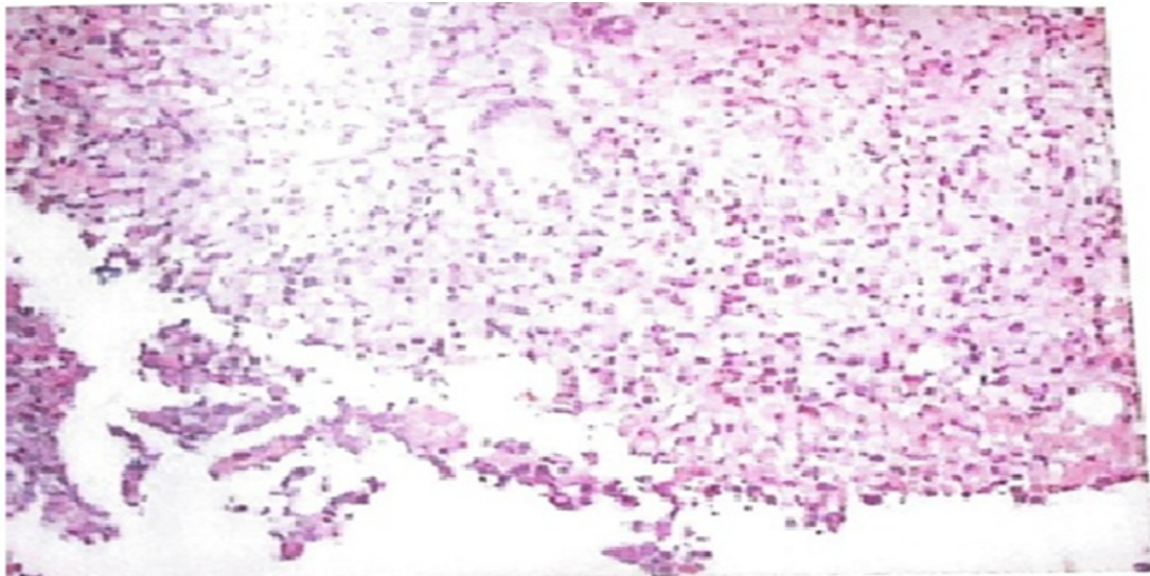


Figure-6: Histopathology of Koch's lymphadenitis showing epithelioid granuloma and Langhan's giant cell 10x (H&E)

Observation and Results

Table 1: Distribution of FNAC under type of reactive group of studied patients studied – FNAC study out of ten patients 6 (60%) reactive lymphadenitis, 2 (20%) abscess, 1 (20%) suppurative lymphadenitis.

Table 2: Distribution of FNAC under type of tubercular pattern involvement

FNAC study: Out 60 patients 48 (80%) had granulomatous lymphadenitis, 6 (10%) cold

abscess, 4 (6.7%) caseous necrosis, 2 (3.3%) early granulomatous lesion.

Table 3: Distribution of granulomas / 20 HPE patients

Number of granulomas/20 (5 sq mm) HPE: Nil – 10 (100%) in reactive lymphadenitis, 12 (20%) tubercular lymphadenitis, total 22 (31.4%)

1-2: 30 (50%), Total 30 (42.9%)

3-4: 14 (23.3%) tubercular lymphadenitis, total 14 (20%)

> 4: 4 (6.7%) tubercular lymphadenitis, total 60 (85.7%)

Table 4: Distribution of number of giant cells / 20 HPE of patients

Number of giant cell / 20 HPE: 10 (100%) reactive lymphadenitis, 50 (83.3%) tubercular lymphadenitis, 2:10 (16.7%) tubercular lymphadenitis

Table 5: Distribution of adenosine deaminase levels U/L of patients

- Adenosine level: <30: 2 (20%) reactive lymphadenitis, 2 (3.3%) tubercular lymphadenitis
- 31-40: 6 (60%) reactive lymphadenitis, 40 (66.6%) tubercular lymphadenitis
- 41-60: 2 (20%) reactive lymphadenitis, 12 (20%) tubercular lymphadenitis
- 60: 2 (20%) reactive lymphadenitis, 6 (10%) tubercular lymphadenitis
- Total 10 (14.2%) reactive lymphadenitis, and 60 (85.7%) tubercular lymphadenitis

Table 1: Distribution of FNAC under Type of reactive patients (No. of patient: 10)

FNAC	Number of patients	Percentage (%)
Reactive lymphadenitis	6	60.0
Abscess	2	20.0
Suppurative lymphadenitis	2	20.0
Total	10	100.0

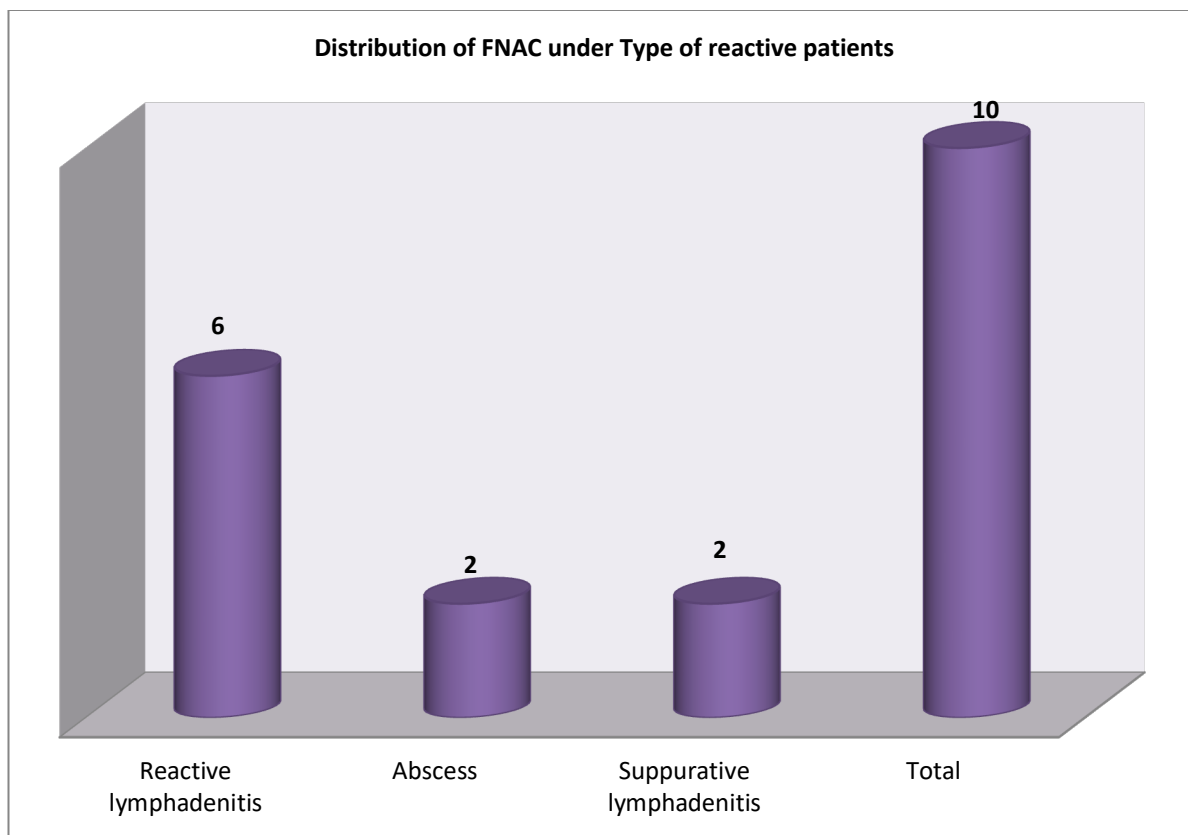


Figure 7: Distribution of FNAC under Type of reactive patients

Table 2: Distribution of FNAC under Type of tubercular pattern involvement in two groups of patients studied

FNAC	Number of patients	Percentage (%)
Granulomatous lymphadenitis	48	80.0
Cold abscess	6	10.0
Caseous necrosis	4	6.7
Early Granulomatous lesions	2	3.3
Total	60	100.0

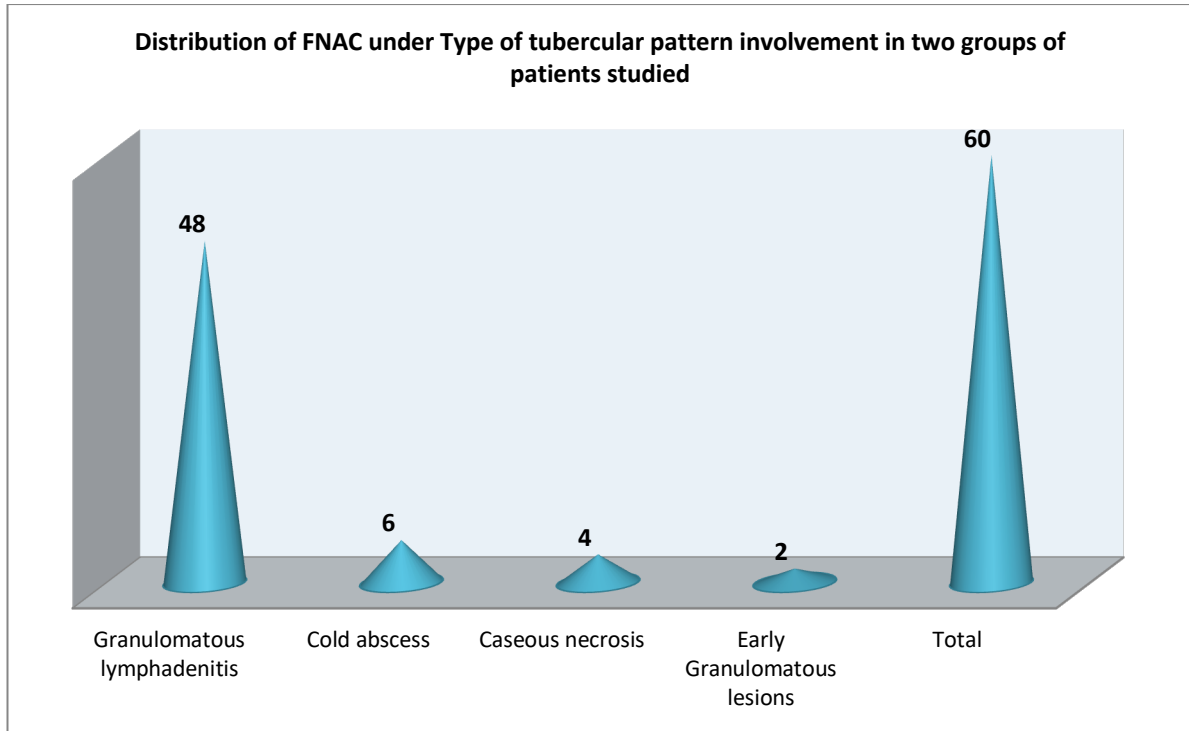


Figure 8: Distribution of FNAC under Type of tubercular pattern involvement in two groups of patients studied

Table 3: Distribution of number of granuloma / 20 HPF of patients studied (Total number of patients: 70)

granulomas / 20 (5 sq mm) HPF	Reactive lymphadenitis	Tubercular lymphadenitis	Total
Nil	10 (100%)	12 (20.0%)	22 (31.4%)
1-2	0 (0%)	30 (50.0%)	30 (42.9%)
3-4	0 (0%)	14 (23.3%)	14 (20.0%)
> 4	0 (0%)	4 (6.7%)	4 (5.7%)
Total	10 (100%)	60 (85.7%)	70 (100%)

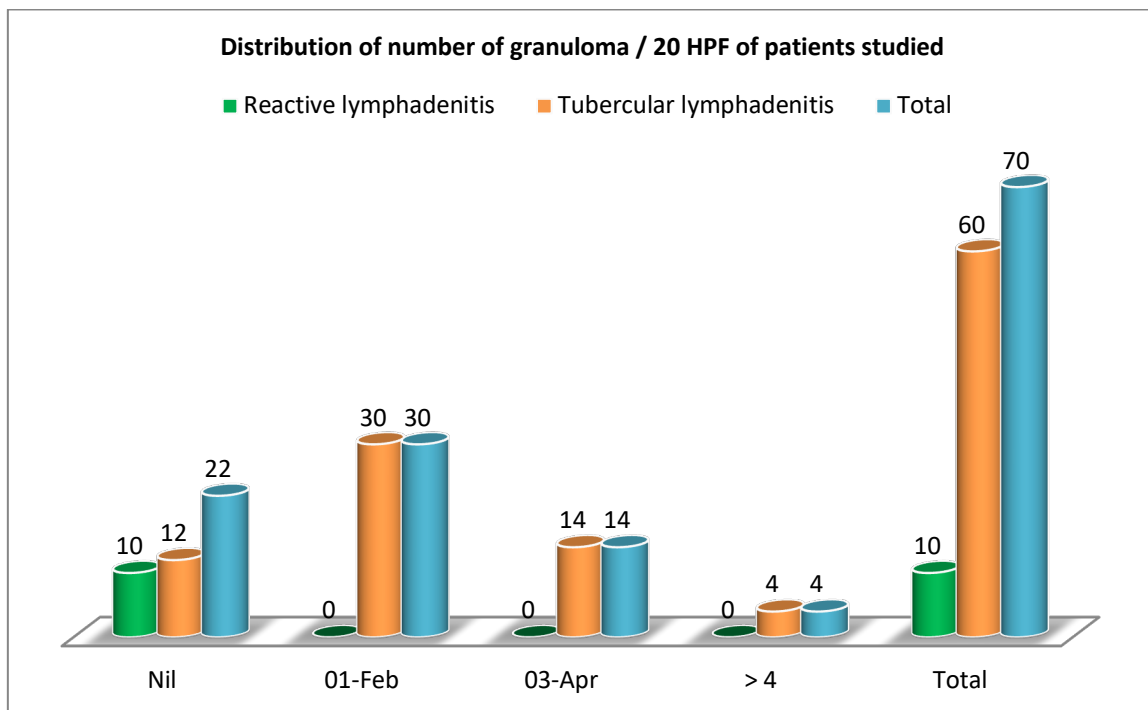


Figure 9: Distribution of number of granuloma / 20 HPF of patients studied

Table 4: Distribution of number of giant cells/20 HPF of patients studied

Number of giant cells/20 HPF	Reactive lymphadenitis	Tubercular lymphadenitis	Total
Nil	10 (100%)	50 (83.3%)	60 (85.7%)
1	0 (0%)	10 (16.7%)	10 (14.3%)
Total	10 (100%)	60 (100%)	70 (100%)

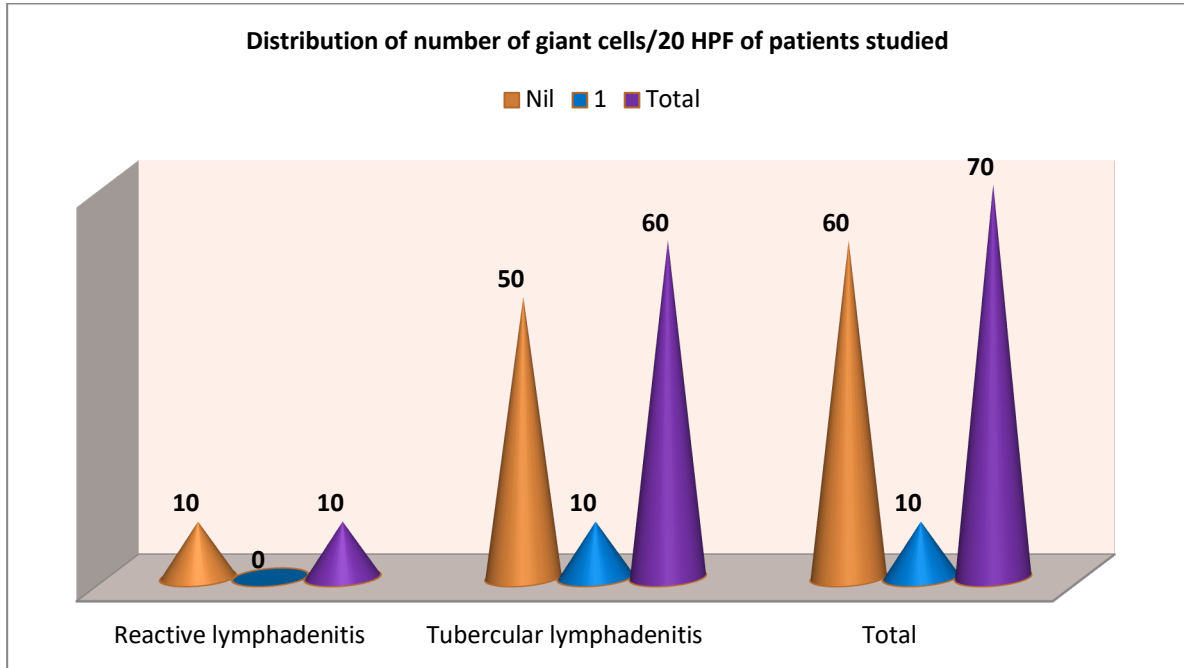


Figure 10: Distribution of number of giant cells/20 HPF of patients studied

Table 5: Distribution of adenosine deaminase levels U/L of patients studied

Adenosine deaminase levels U/L	Reactive lymphadenitis	Tubercular lymphadenitis	Total
<30	2 (20.0%)	2 (3.3%)	2 (2.9%)
31-40	6 (60.0%)	40 (66.7%)	46 (65.7%)
41-60	2 (20.0%)	12 (20.0%)	14 (20.0%)
>60	2 (20.0%)	6 (10.0%)	8 (11.4%)
Total	10 (14.2%)	60 (85.7%)	70 (100%)

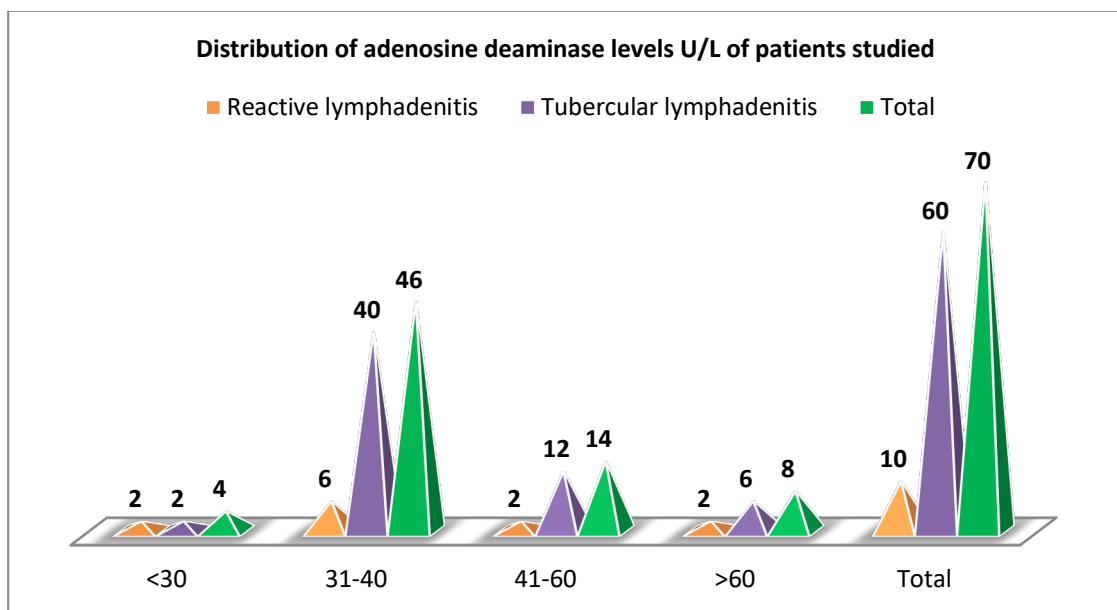


Figure 11: Distribution of adenosine deaminase levels U/L of patients studied

Discussion

The present study of serum adenosine deaminase levels in FNAC-confirmed cases of tuberculous lymphadenitis in the South Karnataka population. Out of 70 patients, 10 (14.2%) had 6 (60%) reactive lymphadenitis, 2 (20%) had an abscess, and 2 (20%) had suppurative lymphadenitis (Table 1). The remaining 60 (85.7%) had 48 (80%) granulomatous lymphadenitis, 6 (10%) cold abscesses, 4 (6.7%) caseous necrosis, and 2 (3.3%) early granulomatous lesion (Table 2). The distribution of adenosine deaminase levels (U/L) was less than 60 in 2 (2.9%) patient, 31-40 ADA level was in 46 (65.7%) patients, 41-60 ADA levels were in 14 (20%) patients, and >60 ADA level was in 8 (11.4%) patients (Table 5) (Figure 1, 2, 3, 4, 5 and 6). These findings are more or less in agreement with previous studies [5,6,7].

A pathological or abnormal lymph node is commonly quoted to be > 1 cm in size; however, in the pediatric population, > 2 cm is considered abnormal. Acute lymphadenopathy is 2 weeks in duration, subacute is 2-6 weeks in duration, and chronic is considered any lymphadenopathy that does not resolve by 6 weeks [8]. Adenosine deaminase (ADA) is an enzyme that catalyzes the deamination of adenosine, forming inosine in the process. ADA activity increases in cell-mediated immune responses during T-cell differentiation and proliferation [9]. The estimation of ADA activity in body fluid serves as a good and reliable tool in the diagnosis of TB pleural effusion and tubercular meningitis when other clinical laboratory tests are negative within sensitive limits [10].

Adenosine deaminase (ADA) is an enzyme of the purine salvage pathway secreted by activated T lymphocytes and macrophages. ADA levels are more significant in adults than children [11]. Apart from TB, in enteric fevers.

There are many methods to confirm lymphadenitis tuberculous, such as the radioactive bromide partition test and antibodies to the mycobacterial antigen. The direct tests are usually measuring a product of infecting organisms such as 3-(2-ketohexyl) indoline, detecting tuberculostearic acid, mycobacterial antigen, or fragments of mycobacterial DNA by polymerase chain reaction, but these methods are expensive and financial burden to middle socioeconomic patients.

Elevated levels of ADA are also reported in typhoid fever, infectious mononucleosis, and brucellosis, but ADA activities in cerebrospinal, pleural, and peritoneal fluids play a vital role in diagnosing tubercular meningitis, pulmonary disease, and ascites.

Summary and Conclusion

The present study of serum ADA levels in FNAC-confirmed cases is a confirmatory diagnosis for tuberculous. The method of ADA estimation is cost-effective; a simple calorimeter is enough to find out the level of ADA within 2 hours. Serum adenosine deaminase levels significantly increase in lymphocyte pleural effusion of tuberculous origin only as compared to peripheral enlargement of lymphadenitis. Hence, the present study demands further cytopathological, genetic, and biochemical studies to rule out the discrimination of ADA levels.

Limitation of study: Owing to remote location of research centre, small number of patients lack of latest techniques we have limited findings and results.

This research work was approved by the ethical committee of Shridevi Institute of Medical Sciences and Research Hospital, Tumkur, Karnataka-572106.

References

1. Verma M, Narang S: Study of adenosine deaminase activity in pulmonary tuberculosis and other respiratory diseases. *Indian J of Clinical Biochemistry* 2004, 19; 129-31.
2. Sharma SK, Suresh V: A prospective study of sensitivity and specificity of adenosine deaminase estimation in the diagnosis of tuberculosis pleural effusion. *Indian J. Chest Dis. Allied Sci.* 2001, 43; 149-55.
3. Mathur PC, Tiwary KK: Diagnostic value of adenosine deaminase activity in tubercular serositis. *Indian J. Tuberc* 2006, 53; 92-95.
4. Surendar, M., Tuli: Historical aspects of Pott's disease (spinal tuberculosis) management. *Eur. Spine J.* 2012, 14: 320-24.
5. Lumsal M, Goutham N: Diagnostic utility of adenosine deaminase activity in pleural fluid and serum of tuberculous and non-tuberculous respiratory disease. *Southeast Asian J. Trop. Med. Public Health* 2007, 38; 363-69.
6. Narayan Gautam, Madhukar Arya I: Comparative study of cerebrospinal fluid Adenosine deaminase activity in patients with meningitis. *Indian journal of path* 2007; 18: 362-64.
7. PC Mathur, KK Tiwari: Diagnostic value of adenosine deaminase (ADA) activity in tubercular serositis. *Indian J. Tuberc.* 2006, 53; 92-95.
8. Rubin BP, Blanke CD: Protocol for the examination of specimens from patients with gastrointestinal stromal tumors. *Arch Pathol Lab Med.* 2010; 134: 165-70.

9. S.K. Verma, A.L. Dubey: Adenosine Deaminase (ADA) Level in Tubercular Pleural Effusion Lung, India, 2008, Jul-Sep; 109-10.
10. Shibagaki T: Adenosine deaminase isoenzymes in tuberculous pleural effusion J. Lab. Clin. Med. 2015, 127; 348-352.
11. Verma M, Narang S: Study of adenosine deaminase activity in pulmonary tuberculosis and other respiratory diseases. Indian J. of Clinical Biochemistry 2004; 19; 129-31.