Role of pleural fluid adenosine deaminase in aetiological diagnosis of pleural effusion

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Citation

Abstract

Study Objective: To find out the diagnostic value of adenosine deaminase in aetiological diagnosis of pleural effusion.

Design: A prospective study. The diagnostic value of pleural fluid adenosine deaminase was studied in 50 patients of pleural effusion.

Setting: Out and In patients service of department of Tuberculosis & Chest Diseases, MLN Medical college Allahabad.

Patients: 50 patients who were above the age of 12 years were studied. Total no. of male patients was 34 and female were 16.

Results: Total no. of tuberculosis patients were 41 and in all pleural fluid adenosine deaminase were more than 36 IU/L (36 to 229.7 IU/L). In case of malignancy no. of patients was 08 and pleural fluid adenosine deaminase was more than 18.5 IU/L (18.5 to 87.6 IU/L). While in one case of hypoproteinemia pleural fluid adenosine deaminase was 8.21 IU/L. If 36 IU/L is taken as cut of limit the sensitivity and specificity of ADA for tuberculosis is 100 % and 22.3 % and for malignancy 87.5 % and 20.1 %

Conclusion: Pleural fluid adenosine deaminase level of more than 100 IU/L is 100% specific for tubercular effusion.

INTRODUCTION

Pleural effusion is a common chest problem, yet it is difficult to establish the aetiological diagnosis in as many as 20% cases. In spite of good history, thorough clinical and radiological examination of patients and full examination of aspirated fluid and pleural biopsy, so there is a need of simple, rapid and reliable diagnostic test to establish the aetiology of pleural effusion. Considering this a prospective study was designed to find out how much is pleural fluid adenosine deaminase level helpful in establishing the diagnosis of pleural effusion.

MATERIAL AND METHOD

The study comprised of 50 patients, both male and female above the age of 12 years who attended the Swaroop Rani Nehru Hospital of Motilal Nehru Medical College, Allahabad, U.P. India between September 95 to August 96. Patients in whom history of typhoid fever, acute viral hepatitis and active cirrhosis were present, were excluded from history. Detailed history was taken and thorough clinical examination was done in each and every patients and they were then subjected to a batteries of investigation which included routine haemogram, urine examination, skiagram chest PA and lateral view, sputum smear examination for AFB and sputum culture for mycobacterium tuberculosis, pleural fluid for protein, Glucose, cell count etc. malignant cells, Gram's stain, pleural fluid examination for AFB and pleural fluid culture for mycobacterium tuberculosis, and other relevant investigation as per need of cases. ADA was measured in pleural fluid by colorimetric method of Guisti and Galanti.

RESULTS

50 patients above the age of 12 years were studied. Male were 34 and female were 16.

Pleural Fluid ADA level was more than 36 IU/L in cases of Tubercular pleural effusion while in case of malignancy it was more than 18.5 IU/L. In one case of hypoproteinemia it was 9.21 IU/L (Table 1).
When we take 36 IU/L as cut off point sensitivity and specificity of ADA for TB and malignancy is shown in Table 2. Out of 50 patients tuberculosis was diagnosed in 41 cases (by history + sputum results + pleural fluid results + response to ATT). Similarly 8 cases of malignancy were diagnosed (4 by direct histology of pleural tissue and 4 by tissue biopsy from lung parenchyma mass or lymph node).

DISCUSSION

Present study confirms that ADA level in tubercular pleural effusion is increased and in non tubercular pleural effusion ADA level seldom exceeded the cut of limit for tubercular pleural effusion. Tuberculosis is a common cause of Pleural effusion. Especially in countries like India. More over incidence of tuberculosis is increasing world wide. Although tubercular pleural effusion can resolve spontaneously but up to 65 % untreated tubercular pleural effusion can develop active tuberculosis. So rapid and accurate diagnosis and current treatment is necessary for tubercular pleural effusion. Whenever a patient of pleural effusion presents we usually investigate online of gross, microscopic and biochemical parameters (excluding ADA level). Although lymphocytic predominant fluid is usually seen in tubercular pleural effusion but all lymphocytic predominant fluid can't be tubercular, it could be malignant but again all malignant are not lymphocytic predominant. So there is a need to differentiate among various causes of pleural effusion. Definitive diagnosis of tubercular is often difficult as in more than 50 % of patients, pleura is the only site of infection. Tuberculin test is non specific and finding can be negative. Because bacterial load is less so pleural fluid culture for mycobacterium tuberculosis is also low (< 20). Pleural fluid ADA estimation is quick and relatively inexpensive.

In present study ADA level in tuberculosis cases was more than 36 IU/L in agreement with Niwa et al. (1985),>38IU/L; and Rodziguez (1962), >37 U/L. In case of malignant pleural effusion our findings co-relate with most of the authors. ADA level in malignancy was up to 87.6 IU/L. ADA level more than 100 IU/L observed only in cases of tubercular pleural effusion so form the study we concluded that if ADA level of more than 100 IU/L is taken as cut off point it is exclusively seen in cases of tubercular pleural effusion. So we can say that estimation of ADA level in pleural fluid is extremely helpful in establishing the aetiology of tubercular pleural effusion and to rule out other diagnosis especially of other diseases in which lymphocyte predominance of pleural effusion is seen such as malignancy and collagen vascular diseases (i.e. rheumatoid arthritis and systemic erythematosus).

LIMITATION OF STUDY

No. of patients studied are small. Definitive criteria can't established on this no. so a large no. of patients are required to confirm our findings further and establish the definitive criteria.

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References

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