



## EVALUATION OF ASPARTATE TRANSAMINASE IN PLEURAL FLUID TO DIFFERENTIATE EXUDATE FROM TRANSUDATE

### Biochemistry

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### ABSTRACT

**Objectives:** To evaluate the value of pleural fluid aspartate transaminase and pleural fluid/serum aspartate transaminase ratio for the purpose of differentiating exudate from transudate pleural effusion.

**Materials and Methods:** A total of 100 indoor patients, admitted to our hospital, having pleural effusion and suffering from varying etiologies, were included in this study. According to the final diagnosis, these 100 patients were divided into two groups: exudate (60) and transudate (40) pleural effusion.

**Results:** The mean serum AST level in exudates and transudates are  $34.25 \pm 2.55$  IU/L and  $43.92 \pm 4.86$  IU/L respectively and they are statistically insignificant ( $p = 0.083$ ). The mean pleural AST level in exudates and transudates are  $28.98 \pm 2.37$  IU/L and  $28.80 \pm 1.93$  IU/L respectively and they are statistically insignificant ( $p = 0.952$ ).

**Conclusion:** From this study it is concluded that aspartate transaminase activity having no significant value for differentiation of exudate from transudate pleural effusion.

### KEYWORDS

Aspartate Transaminase, pleural effusion, exudate, transudate

### INTRODUCTION

Pleural effusion is an excessive accumulation of fluid in the space lies between the lung and chest wall i.e. pleural space.<sup>1</sup> In normal condition, pleural space contains 0.1–0.3 ml/kg body weight of fluid (near about of 10 ml of fluid on each side) between the parietal and visceral pleura.<sup>2-4</sup> Fluid filtered from systemic capillaries down a small pressure gradient. Fluid drains into the systemic circulation via a delicate network of lymphatics and eventually enters the mediastinal lymph nodes. This pleural fluid acts as a lubricant and allows the visceral pleura to slide along the parietal pleura during respiratory movements.<sup>5</sup>

### MATERIAL AND METHOD

Present study was carried out at the People's College of Medical Sciences and research center Bhopal during period of February 2012 to September 2013. A total of 100 (male and female) patients with pleural effusions of diverse etiologies, attending various departments (pulmonary, cardiology, surgery and gynecology) of People's College of Medical Sciences and research center Bhopal. Patients with malignant pleural effusion from Jawahar Lal Nehru cancer Hospital, Bhopal and patients with tubercular pleural effusion from TB hospital Bhopal were included in this study.

The protocol for this study was approved by the Institutional ethical Committee, and informed consent was obtained by all participants before percutaneous thoracentesis and blood sampling. All estimation was performed in the Department of Biochemistry in People's college of Medical Sciences and research center Bhopal.

In all cases, a standard clinical protocol was followed and routine laboratory tests of pleural fluid were carried out. Pleural fluid samples were cultured and pleural biopsy (Transthoracic needle biopsy as well as tru cut biopsy) was also done to obtain a definitive diagnosis.

The patients were divided in two groups: *Group A - exudates*, and *Group B - transudates* on the basis of extensive clinical, radiological and biochemical evaluation, achieved by standard methods.

- I. Group – A (exudates) – This group comprise of 60 patients –
  - I. A total of 30 Patients of tubercular pleural effusion in which diagnosis was confirmed by clinical and radiological evaluation.
  - II. A total of 13 patients with parapneumonic pleural effusion or empyema in which diagnosis was confirmed by clinical presentation, positive microbial culture and a radiographic pulmonary infiltrate that disappeared after antibiotic treatment.
  - III. A total of 7 patients with malignant pleural effusion in which diagnosis were confirmed by medical history (about family members and sibling and addiction) and histopathology and cytology examination of the pleural fluid, sputum and fiber optic

brochoscopy guided transthoracic biopsy tissue for malignant cells.

- IV. Ten patients of exudates other than the above causes, out of which three patient are of pleural effusion due to cardiac origin out of one having history of anterior pericardiectomy for constrictive pericarditis and other two develop pleural effusion after few weeks of coronary artery bypass graft previous all three were diagnosed by clinical presentation and colour dopplar echocardiography. Remaining 7 patients of peritonitis and pancreatitis, diagnosis settled by clinical history, clinical examination and finally by ultrasonography (USG) and computed tomography (CT) scan.
2. Group – B (transudates) – This group comprise of 40 patients, out of which five were of nephrotic syndrome diagnosed if the patient had proteinuria, edema and hypoalbuminemia; two patients of pulmonary embolism diagnosed if having out of proportion of shortness of breath, chest discomfort, pleuritic chest pain, deranged coagulation profile and confirmed by CT pulmonary angiography showing thrombus in lumen; three patients of cirrhosis of liver diagnosed by liver biopsy in the presence of ascitis; eighteen patients of congestive cardiac failure diagnosed due to cardiomegaly, radiological evidence of congested lungs, peripheral edema and response to treatment of congestive cardiac failure and remaining 14 patients of chronic renal failure who were receiving peritoneal dialysis showing increase urea, creatinine level in blood.

Exudates were separated from transudates by Light's Criteria:<sup>[6]</sup>

1. Pleural fluid to serum LDH greater than 0.9, &/or Pleural fluid LDH more than 280 IU/L or pleural fluid LDH more than two-third normal upper limit for serum.
2. Pleural fluid / serum protein ratio greater than 0.5.

The investigation of a pleural effusion is, in general, a very straight forward process with the combination of clinical history, clinical examination, radiology and pleural fluid analysis leading to diagnosis in most cases. While most fluid samples are sent for routine analysis including protein, LDH, Glucose, cytology and microbiology, there are a number of more unusual fluid analysis available which in some cases directly lead to, and in others are suggestive of the diagnosis. Moreover, other fluid markers are constantly being evaluated as a diagnostic tool.

In all these patients, following biochemical parameter were estimated in serum and pleural fluid

- Aspartate transaminase
- Lactate dehydrogenase
- Protein

**Collection of sample:**

Use a serum separator tube and allow samples to clot for 2 hours at room temperature or overnight at 2-8°C. Centrifuge at approximately 1000 × g (or 3000 rpm) for 15 minutes. Remove serum and assay immediately or aliquot and store samples at -20°C or -80°C

**OBSERVATION**

The collected data was analyzed using SPSS windows (version 20). Normality of data was checked using Shapiro Wilk's test. The parameters those were normally distributed were analyzed by parametric (independent samples T test) and the results were expressed in Mean ± Standard deviation (SD). Parameters which showed non normal distribution were analyzed by non parametric (one way analysis of variance) and the results were expressed in Mean ± Standard error (SE). All the results and observations are presented as Tables, bar diagram (along with error bar). The cut off for significance was taken at *p* value equal to or less than 0.05.

**Table no. 1 – Showing serum and pleural fluid AST (IU/L) in group I and group II**

AST	Group I	Group II (Mean ± SE)	F value (Mean ± SE)	P value
Serum	34.25 ± 2.55	43.92 ± 4.86#	8.32	0.083
Pleural fluid	28.98 ± 2.37	28.80 ± 1.93#	0.03	0.952

\$: Standard error

#: One way ANOVA, *p* > 0.05

AST showed non normal distribution, was analyzed by non parametric (one way analysis of variance) and the results was expressed in Mean ± Standard error (SE). **Table 1** represents the serum and pleural fluid AST value of the study groups in which value is expressed as Mean ± SE and consider F value instead of T value and both the study groups [*p* = 0.083) and (*p* = 0.952) respectively] had no statistically significant difference.

In exudative pleural effusion and transudative pleural effusion conditions it is also found there is no statistically significant difference [Table 2].

**Table no. 2 – Showing AST (IU/L) in pleural fluid and serum in exudative effusion**

AST	Pleural fluid (Mean ± SE)	Serum (Mean ± SE)	F value	P value
Exudates [E]	28.98 ± 2.37	34.25 ± 2.55#	2.281	0.134
Transudates [T]	28.80 ± 1.93	43.92 ± 4.86#	8.32	0.086
Ratio [E/T]	0.83 ± 0.095	0.68 ± 0.02	15.18	0.001*

\$: Standard error

#: One way ANOVA, *p* > 0.05

\*: One way ANOVA, *p* < 0.05

**DISCUSSION**

AST is a pyridoxal phosphate (PLP)-dependent transaminase enzyme [7]. AST catalyzes the reversible transfer of a α-amino group between aspartate and glutamate and, as such, is an important enzyme in amino acid metabolism [8].

Liver being the primary site for amino acid metabolism and second in order is heart. So that AST level goes rise (up to the twice or thrice than normal value) in liver parenchymal diseases and heart diseases. Especially lungs are not known to site of amino acid metabolism so, AST activity significantly not seen in respiratory system.

We observed that AST value more than 50 IU/L in pleural fluid in case of cirrhosis (transudative pleural effusion); the possible reason behind those pleura have close proximity with the liver and communicate with pleura via micro channels. This channel guides movement of fluid from the peritoneal cavity; this transdiaphragmatic unidirectional flow of fluid from peritoneal cavity is due to pressure gradient across the diaphragm as occurs in hepatic hydrothorax that sometimes complicates the ascites of liver cirrhosis. [9]

In present study, the mean serum AST level in exudates and transudates are 34.25 ± 2.55 IU/L and 43.92 ± 4.86 IU/L respectively and they are statistically insignificant (*p* = 0.083) (Table - 1). The mean pleural AST level in exudates and transudates are 28.98 ± 2.37 IU/L and 28.80 ±

1.93 IU/L respectively and they are statistically insignificant (*p* = 0.952) (Table - 1). The mean pleural / serum AST ratio in exudates and transudates are 0.83 ± 0.095 and 0.68 ± 0.02 respectively and they are statistically significant (Table - 2).

**Shashi seth et al (1986)** [10] also observe there is no significant change seen in pleural fluid in both condition.

So we conclude that AST has no significant value to exclude exudative pleural effusion to transudative pleural effusion.

**REFERENCE**

1. Sahn SA. State of the art. The pleura. Am Rev Respir Dis. 1988; 138:184-234.
2. Miserocchi, G. Physiology and pathophysiology of pleural fluid turnover. Eur. Respir. J. 1997;10:219-25.
3. Miserocchi, G., and E. Agostoni. Contents of the pleural space. J. Appl. Physiol. 1971;30:208-13.
4. Sahn, S. A., M. L. Willcox, J. T. Good, D. E. Poots, and G. F. Filley. Characteristics of normal rabbit pleural fluid: physiologic and biochemical implications. Lung 1979;150:63-69.
5. Storey DD, Dines DE, Coles DT. Pleural effusion: a diagnostic dilemma. JAMA 1976; 236: 2138-6.
6. Light RW, MacGregor MI, Luchsinger PC, Ball WC. Pleural effusion: the diagnostic separation of transudates and exudates. Ann Intern Med 1972; 77: 507 - 13.
7. Kirsch JF, Eichele G, Ford G, Vincent MG, Jansonius JN, Gehring H et al. (1984). "Mechanism of action of aspartate aminotransferase proposed on the basis of its spatial structure". J Mol Biol 174 (3): 497-525
8. Berg, JM; Tymoczko, JL; Stryer, L (2006). Biochemistry. W.H. Freeman. pp. 656-660
9. Boggs DS and Kinawatz GT: Review: physiology of pleural space. Am Med Sci 1995; 309: 53-59.
10. Shashi Seth, Harbans Lal, S.C. Seth and A.S. Saini. Serum and pleural fluid changes in patients with effusion. Ind. J. Tub., 1986, 33, 17