Serum-effusion Albumin Gradient in Separation of Transudative and Exudative Pleural Effusions

To the Editor:

We read with interest the article by Roth et al, which appeared in the September 1990 issue of Chest.

The serum-effusion albumin gradient of 1.2 g/dl is proposed as a diagnostic tool in differentiating exude and transude in patients with pleural effusions, especially following diuretic therapy. The same parameter, but at a level of 1.1 g/dl, has been found useful in the differential diagnosis of ascites in cirrhosis.

We prospectively studied 54 consecutive patients (36 men, 18 women, mean age 57 ± 11 years old) who were undergoing diagnostic or therapeutic thoracocentesis to compare the use of transudate albumin gradient with that using the Light et al traditional criteria.

Using the Light et al criteria, 42 patients were defined as having exudates (16 malignancy, 13 tuberculosis, 9 parapneumonic, 1 systemic lupus erythematosus (SLE), 1 rheumatoid arthritis, 1 radiation-induced, 1 Christian-Weber syndrome), and 12 as transudates (1 cirrhotic ascites, 11 congestive heart failure).

The effusion protein and lactate dehydrogenase (LDH) levels, the effusion/serum protein and LDH ratios, and the serum-effusion albumin gradient of 1.2 g/dl in each group were compared using Student’s unpaired t test and were all significantly different (Table 1).

Using albumin cutoff value of 1.2 g/dl to indicate a transude, 22 patients were classified as having transudates. Eleven of these patients had clinical congestive heart failure and one had cirrhotic ascites. However, ten patients (four parapneumonic, one lymphoma, one radiation-induced, two tuberculosis, one malignancy, one SLE) were misclassified as transudates. The mean albumin gradient in these misclassified patients was 1.84 ± 0.41 (range: 1.3-2.6 g/dl). In the same group, one patient with tuberculosis and one patient with parapneumonic pleural effusion also had chronic renal failure.

Diuretic treatment of patients with congestive heart failure caused significant elevation of the protein content; in some cases, a transude might be converted into a pseudoexudate-high protein transude. We performed thoracentesis immediately on identification of a patient with congestive heart failure before any diuretic therapy was given to rule out the possibility of pseudoexudates. Therefore, we did not observe any patient with high protein transude.

In the present study, the albumin gradient was 76 percent sensitive and 100 percent specific to indicate exudates. Light’s criteria were 100 percent sensitive and 100 percent specific at identifying exudates. The difference between the sensitivities is clearly not significant using the proportion test (p < 0.05). McNemar’s exact test showed a statistically significant difference between these two methods (p < 0.05).

In a group of 26 patients with malignant effusions Roth et al were able to identify only two patients with an albumin gradient in the transudative range, and proposed the use of this parameter, partially in cases of congestive heart failure.

We conclude that the serum-effusion albumin gradient is a reliable criterion for differentiating exudative from transudative effusion. We found this gradient, however, compared with Light’s criteria, has a tendency to overdiagnose a transude, and we believe its use should be limited to patients suffering from heart failure.

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REFERENCES

To the Editor:

The results reported by Dr. Ceyhan differ from ours in two very important aspects. First, he specifically excluded patients with congestive heart failure who are on chronic diuretic therapy. These are the very patients where the albumin gradient appears to be helpful and where it appeared that the albumin gradient was more specific than the Light et al criteria (Chest 1990; 98:546-49).

Second, he reports a much lower sensitivity with 10 of 42 patients having exudative effusions misclassified as transudates using the albumin gradient. This is very concerning and calls into question the utility of the albumin gradient since we propose it as a method to confirm that a "pseudoexudate" by Light’s criteria is actually a transude. By combining multiple tests, Light’s criteria provide a heightened sensitivity for exudates.

However, Dr. Ceyhan states that he used Light’s criteria to define exudates and transudates. If a test is used in the definition of disease, then it will make that test appear 100 percent sensitive and specific. Also, it would be important to review the clinical data from the misclassified patients to closely look for secondary transudative causes of pleural effusion such as coexistent congestive heart failure or renal failure with fluid overload. As in studies of the albumin gradient applied to ascites, our data suggest that when an exudative cause for pleural fluid is combined with a transudative cause, the albumin gradient reflects the pressure gradient and appears transudative.

Although Dr. Ceyhan’s data is relevant, we do not think it changes our suggestion that the albumin gradient can be helpful as an adjunct to the Light et al criteria for exudates.

Table 1 — Pleural Fluid Characteristics

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<thead>
<tr>
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<th>Exudative*</th>
<th>Transudative</th>
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<tbody>
<tr>
<td>Pl eff LDH level (U/ml)</td>
<td>606 ± 140</td>
<td>76 ± 20</td>
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<tr>
<td>Pl eff protein level (g/dl)</td>
<td>4 ± 1.2</td>
<td>1.9 ± 0.9</td>
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<tr>
<td>Pl eff/ser LDH ratio</td>
<td>4.8 ± 1.7</td>
<td>0.48 ± 0.4</td>
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<tr>
<td>Pl eff/ser protein ratio</td>
<td>0.65 ± 0.16</td>
<td>0.31 ± 0.13</td>
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X ± SD
*p < 0.05.