Study On Serum Electrophoretic Pattern In Congestive Cardiac Failure

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INTRODUCTION:

Congestive cardiac failure is not an uncommon manifestation of the heart diseases. This state is due to the increased venous pressure leading to congestion and failure of the functions of various systems specially the liver. There is hepatic dysfunction due to oligemia in the liver resulting from a rise in hepatic venous pressure. It has been well observed by many workers, that due to the involvement of liver there is disturbed protein metabolism in congestive cardiac failure. In the present work total serum protein and its various fractions has been undertaken to study by paper-electrophoresis in congestive cardiac failure.

METHODS AND MATERIAL:

The clinical material included 50 cases of rheumatic heart diseases with polyvalvular lesion and congestive cardiac failure (C. C. F.) from Medical Ward of Darbhanga Medical College Hospital, Laheriasarai. 25 Normal individuals were also studied for Control group. Total serum protein estimation was done by Biuret method (King and Wotton, 1964) and paper-electrophoresis was done by a Systronic Horizontal Electrophoretic Tank. Staining and washing of electrophoretogram was done by Friglen technique (Varley, 1969).

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OBSERVATION:

**TABLE-1**

Showing mean value of Total serum protein and its component in Gm.% in Control group.

<table>
<thead>
<tr>
<th>No. of Cases</th>
<th>Total serum protein</th>
<th>Electrophoresis</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Serum albumin</td>
<td>alpha-1</td>
<td>alpha-2</td>
<td>Beta</td>
<td>Gamma</td>
</tr>
<tr>
<td>25</td>
<td>6.9 ± 0.05</td>
<td>4.13 ± 0.13</td>
<td>0.29 ± 0.05</td>
<td>0.43 ± 0.03</td>
<td>0.56 ± 0.07</td>
<td>1.27 ± 0.07</td>
</tr>
</tbody>
</table>

**TABLE-2**

Showing mean value of Total serum protein and its component in Gm.% in Congestive cardiac failure (C. C. F.)

<table>
<thead>
<tr>
<th>No of cases</th>
<th>Total Serum protein</th>
<th>Electrophoresis</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Serum albumin</td>
<td>alpha-1</td>
<td>alpha-2</td>
<td>Beta</td>
<td>Gamma</td>
</tr>
<tr>
<td>50</td>
<td>4.8 ± 0.09</td>
<td>1.9 ± 0.07</td>
<td>0.20 ± 0.03</td>
<td>0.39 ± 0.05</td>
<td>0.59 ± 0.04</td>
<td>1.87 ± 0.09</td>
</tr>
</tbody>
</table>

DISCUSSION.

In the present study Total serum protein and Serum albumin were found to be less than the Control group. William et al, Waltz and Ikuichi Ueyama have also reported a low total serum protein and serum albumin in C.C. F. Payne et al, suggested that the fall in serum albumin and thus in total serum protein may be due to venous congestion of the gastro-intestinal tract, which in turn causes anorexia and vomiting. But Jeejee Bhow suggested that low serum albumin is mostly due to enteric loss of albumin i.e. from blood vessels to extravascular space. On the other hand Ikuichi Ueyama suggested that low serum albumin is due to decreased hepatic synthesis of the albumin. He found a definite correlation between hepatomegaly and a decreased albumin synthesis. Thus the fall of serum albumin...
Total serum protein is of multiple etiology. Besides reduced synthesis of albumin by the liver, anorexia, vomiting, decreased absorption of protein due to gastro-intestinal congestion, and enteric loss of albumin also work as a contributory factor.

The level of alpha-1, alpha-2, and beta globulin fractions remained nearly the same as in the control group. Saunderman and Jeejee Bhoy have also reported a normal value of alpha-1, alpha-2, and beta globulin in their study in C. C. F.

Value of gamma-globulin in the present study is higher than in the control group. Other workers like Ogryzlo et al. and Ikuichi Ueyama have also reported high level of Gamma-globulin in C. C. F. Ikuichi Ueyama suggested that increase of Gamma-globulin seen in compensated stage is due to some degree of organic change of hepatic interstitium resulting from oligemia and venous stagnation of blood in hepatic vein. In our opinion, the increase of Gamma-globulin in C. C. F. due to rheumatic valvular lesion may partly be explained by the fact that the valvular lesion are produced after streptococcal infection and partly because recurrent infection like sore-throat and chest infection. As Ikuichi Ueyama has suggested hepatic damage due to oligemia may also work as a contributory factor.

SUMMARY:

Electrophoresis of serum protein was done in 50 cases of Rheumatic heart disease with polyvalvular lesion and congestive cardiac failure. 25 cases were also studied as Control group. Total serum protein and serum albumin were less than the control group while no change was seen in alpha-1, alpha-2 and beta globulin. Considerable rise in the level of Gamma-globulin was noted.

REFERENCES:


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