RELATIONSHIP BETWEEN THE DISTRIBUTION OF GASTROESOPHAGEAL VARICES AND PORTAL AND SPLENIC VEIN SIZE

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ABSTRACT

This work was done on 200 patients with gastric and oesophageal varices to study the relation between the distribution of varices (seen by upper endoscopy) and the portal and splenic vein size (determined by abdominal ultrasonography). It was found that patients with pure oesophageal varices had greater portal vein size than those with pure gastric varices. The difference was statistically highly significant ($P < 0.001$). It was also found that patients with isolated gastric varices had greater splenic vein size than those with pure oesophageal varices. The difference was also highly significant ($P < 0.001$). Consequently, portal / splenic vein ratio ($Pv / Sv$ ratio) was found to be larger in patients with oesophageal varices than those patients with isolated gastric varices ($P < 0.001$). We found that $Pv / Sv$ ratio has a very high specificity (94%) in diagnosing isolated gastric varices (whether alone or accompanied by oesophageal varices but disconnected at the cardia) when it is $\leq 1$. However, the same ratio ($Pv / Sv \leq 1$) is much less sensitive (sensitivity = 38%) in diagnosing isolated gastric varices.

INTRODUCTION

Actually, varices developing at the region of the cardia vary considerably regarding their distribution. Varices may be restricted to the oesophagus (isolated oesophageal varices), traverse the region of the cardia extending from the oesophagus to the gastric fundus or lesser curvature (oesophageal with gastric extension), may be present in both the oesophagus and the stomach but appear disconnected at the cardia (oesophageal and gastric varices), or it may be restricted to the gastric region (isolated gastric varices not accompanied by oesophageal varices).

Why patients with portal hypertension differ from each other regarding distribution of their varices? Does the sonographic picture of the portal venous system, mainly portal and splenic vein diameters, differ from patient to patient according to the distribution of their varices? Can we predict the distribution of varices of patients with portal hypertension from their sonograph picture?

The aim of the present work is to study the detailed endoscopic and sonographic findings of patients with different types of varices in a trial to answer these questions.
SUBJECTS AND METHODS

This study was done on 200 patients who were admitted to department of Internal Medicine, Kasr El-Aini Hospitals, Cairo University. Eighty two patients were admitted to Hospital on emergency basis with active variceal bleeding as proved endoscopy, while 118 patients were electively admitted with chronic liver disease, liver cirrhosis, ascites, hepatic encephalopathy, or just splenomegaly for further investigations. They were 153 males and 47 females. The mean age of the patients was 48.5 ± 7.4 years.

All patients were subjected to

- Full history and clinical examination.
- Liver function tests including:
  * Serum bilirubin: total and direct.
  * Alanine aminotransferase (ALT or SGPT).
  * Aspartate aminotransferase (AST or SGOT).
  * Alkaline phosphatase (ALP).
  * Total plasma proteins, albumin and globulins.
  * Prothrombin time and concentration.
- Upper endoscopy:
  All cases were subjected to upper endoscopy using a flexible fiberoptic endoscope (Olympus GIF, type Q 20). In elective cases (not actively bleeding at time of endoscopy), the patient were fasting for 10-12 hours and sedated by intravenous injection of 10 mg diazepam (Valium). The pharynx was anaesthetized with a xylocaine spray 10% solution. The endoscope was done while the patient was lying on his left side. For patients admitted on emergency basis by hematemesis or melena, primary measures as gastric lavage and urgent blood transfusion were done and immediate endoscopy was performed once hemodynamic stability was achieved. No other measures for control of bleeding as balloon tamponade or vasoconstrictive agents as vasopressin were used.

According to the endoscopic findings, patients with varices were classified into four group according to the distribution of the varices.

Group I:

Patients with pure oesophageal varices, not extending below the cardia, not included within a hiatus hernia and not accompanied by gastric varices.

These varices are further classified according to its grade (Waldram et al., 1977) into:

Grade I: Small straight cords confined to the lower 1/3 of the oesophagus.

Grade II: Moderate sized varices with well defined areas of normal mucosa inbetween, forming several; distinct vertical cords and confined to the lower 1/2 of the oesophagus.

Grade III: Gross varices extending on the proximal 1/2 of the oesophagus, they are so large and tortuous that normal mucosa may not be visible among them.

Grade IV: The lumen of the oesophagus is completely obstructed, and a lot of telangectasias and sometimes erosions can be detected.

Group II:

Patients with oesophageal varices with extension to the fundus of the stomach or
the lesser curvature or extending into a hiatus hernia.

**Group III:**

Patients with combined oesophageal and gastric varices that were disconnected at the cardia.

**Group IV:** Patients with isolated gastric varices that were disconnected at the cardia.

- **Abdominal ultrasonography:**

All patients were subjected to detailed abdominal sonographic examination and the diameter of portal and splenic veins were accurately estimated. Ultrasonography was done using a linear and sector transducers of Aloka 630 machine. Patients were examined while fasting for at least 8 hours to avoid the variations of portal vein diameter in relation to meals. Bellamy et al., (1984) reported 47 - 50% increase in the portal vein diameter 30 minutes to one hour following a meal, returning to baseline values after 3 hours. Examination was done in the supine and left lateral positions during resting inspiration to avoid the variation of splenic vein caliber during deep inspiration (Weill, 1982). Bolondi et al., (1982) reported 50 to 100% increase in splenic vein caliber during deep inspiration.

Eighty two out of the 200 patients were in active upper gastrointestinal bleeding at the time of admission to hospital. Urgent upper endoscopy with injection sclerotherapy was done to all of them, then abdominal sonography was done electively the next morning after control of bleeding. In 1990, Bourbenet et al., studied the effect of variceal injection sclerotherapy using polydocalon on hepatic venous pressure gradient and azygos flow. This hemodynamic study was done before and about one week after complete variceal obliteration. It was found that endoscopic variceal sclerotherapy did not change hepatic venous pressure gradient and azygos blood flow. Consequently, endoscopic injection sclerotherapy is unlikely to affect portal and splenic vein diameters, especially if sonography was done, as in our work, less than 24 hours after sclerotherapy.

**RESULTS**

Comparing the size of the portal vein in the four studied groups, it was found that patients of group I and II had greater portal vein size than those of group III and IV. The mean portal vein size in patients of group I and II was $15.5 \pm 2.4$ and $16.1 \pm 3.0$ mm respectively, while that of group III and IV was $13.7 \pm 1.9$ and $12.5 \pm 2.2$ mm respectively. The difference is highly significant with $p$ value $< 0.001$.

The difference between group I & II and between group III & IV was not significant (Table 2 and Figure 1).

Comparing the size of the splenic vein in the four studied groups, it was found that patients of group IV had greater splenic vein size than those of group I. The mean splenic vein size in patients of group IV was $12.6 \pm 1.7$ mm while that of group I was $10.0 \pm 3.1$ mm. The difference is highly significant with $P$ value $< 0.001$.

The mean splenic vein size in patients of group II and III was $11.8 \pm 3.2$ and $11.4 \pm 2.8$ mm respectively. It was found that the patients of these two groups (II and III) were neither significantly different from
each other nor from those of group I and IV regarding their splenic vein size (Table 1).

Regarding the portal / splenic vein ratio (Pv/Sv) in the studied groups, it was found that patients of group I had the highest mean value being 1.7 ± 0.6 and patients of group IV had the lowest mean value being 1.0 ± 0.2, while those of group II and III had intermediate values, being 1.5 ± 0.5 and 1.3 ± 0.4 respectively.

The difference of the portal / splenic vein ratio between patients of group I and IV was found to be highly significant with P value < 0.001. There was no significant difference between the portal / splenic vein ratio in patients of each of group II & III and the other groups (Table 1).

Out of the 150 patients of group I and II (patients with only oesophageal varices and patients with oesophageal varices and gastric extension, respectively), only 9 patients had portal / splenic vein diameter ratio (Pv/Sv) equal or less than one i.e. portal vein diameter was equal or less than the splenic vein diameter. The remaining 141 patients had Pv/Sv > 1. We also found that 19 patients out of the 50 patients of group III and IV had Pv/Sv ≤ 1, while the remaining 31 patients had Pv/Sv > 1 (table 2). We found that Pv/Sv has a very high specificity (94%) in diagnosing isolated gastric varices (whether alone (group 4) or accompanied by oesophageal varices but disconnected at the cardia (group III) when it is ≤ 1. However, the same ratio (Pv/Sv ≤ 1) is much less sensitive (Sensitivity = 38%) in diagnosing isolated gastric varices.

It was found that there is good correlation between the size of the spleen and the size of the splenic vein in all patients (correlation coefficient "r" = 0.65).

It was also found that there is no significant difference of the size of the spleen between the studied groups (the mean values of splenic long axis in the four studied groups were 16.1 ± 2.7, 17.4 ± 3.7, 16.4 ± 2.5 and 15.4 ± 1.7 cm respectively) (Table 1).

Regarding the size of the portal vein in relation to the grade of varices in studied groups, it was found that group II patients with grade III or IV varices have a significantly larger portal vein than patients with grade I or II varices (p value = 0.015). No significant difference was found between the size of the portal vein and the grade of oesophageal varices in patients of each of group I and III (Table 3).

However, it was found that there is no significant difference between the size of the splenic vein and the grade of varices in the studied groups (Table 4).

It was found that 41.4% of patients of group I who presented by variceal bleeding had positive red colour sign, while only 12.8% of group I patients without variceal bleeding had positive red colour sign. This difference was found to be significant with p = 0.002. We also found that 44.4% of patients of group II who presented by variceal bleeding had positive red colour sign, while only 18.8% of group II patients without variceal bleeding had positive red colour sign. However, this difference was not found to be significant. It was also found that 26.3% of patients of group III who presented with variceal bleeding had positive red colour sign, while 28.6 % of group II patients...
Relationship between the ....

without variceal bleeding had positive red colour sign. This difference was not found to be significant. None of group IV patients was found to have red colour sign. Regarding the total number of patients 29 out of the 82 patients with variceal bleeding had positive red colour sign (16.1%). This difference is significant with p value of 0.002 (Table 5).

It was found that of the 82 patients with grade III & IV oesophageal varices, 42 had history of bleeding, while out of the 108 patients with grade I and II oesophageal varices, only 33 had history of bleeding. This difference was found to be significant with p value of 0.004 (Table 6).

DISCUSSION

After using the advanced generations of endoscopy combined with angiography, it has been found that gastric varices occur in portal hypertension just as frequent as oesophageal varices (Mathur et al., 1990). In 1988, Watanabe and colleagues reported that gastric varices were present in 57% of the patients with varices occurred nearly as frequent as oesophageal varices but large gastric varices are rather infrequent (10% of patients with varices) (Watanabe et al., 1988).

It is of the utmost importance to search carefully for, and document the presence of gastric varices for several reasons; first, a proportion of cases eventually bleed (60% in the experience of Hosking and Johnson, 1988). Bleeding from gastric varices has been reported occur in 3 to 30% of cases of variceal bleeding in general (Buset et al., 1987 and Mathur et al., 1990). It has also been reported that gastric varices were the source of bleeding in 3 to 15% of cases of rebleeding after endoscopic eradication of oesophageal varices (Paquet and Oberhammer, 1978 and Bretagne et al., 1987). Second, until about 10 years ago, differentiation between gastric and oesophageal varices as the source of bleeding was not important, as both would usually have been treated by a portosystemic shunt. Now that this has become less popular with the widespread use of injection sclerotherapy, differentiation became not only important but even mandatory (Hosking and Johnson, 1988).

In this study, portal vein diameter was found to be larger in patients of group I and II than in patients of group III and IV. This difference was found to be highly significant with p < 0.001. On the other hand, the largest diameter of the splenic vein was found in group IV patients while the smallest diameter was found in group I patients. The difference was also found to be highly significant (P < 0.001). Consequently, the portal / splenic vein ratio was found to be high in group I patient while it was low in group IV patients. This difference was found to be highly significant with P < 0.001.
Table (1): Sonographic findings in studied groups.

<table>
<thead>
<tr>
<th></th>
<th>Group I (n = 107)</th>
<th>Group II (n = 43)</th>
<th>Group III (n = 40)</th>
<th>Group IV (n = 10)</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Size of spleen (cm)</td>
<td>16.1 ± 2.7</td>
<td>17.4 ± 3.7</td>
<td>16.4 ± 2.5</td>
<td>15.4 ± 1.7</td>
<td>N.S.</td>
</tr>
<tr>
<td>P.V. diameter (mm)</td>
<td>15.5 ± 2.4a</td>
<td>16.1 ± 3.0a</td>
<td>13.7 ± 1.9b</td>
<td>12.5 ± 2.2b</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>S.V. diameter (mm)</td>
<td>10.0 ± 3.1b</td>
<td>11.8 ± 3.2 ab</td>
<td>11.4 ± 2.8ab</td>
<td>12.6 ± 1.7a</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Portal / splenic vein ratio</td>
<td>1.7 ± 0.6a</td>
<td>1.5 ± 0.5 ab</td>
<td>1.3 ± 0.4 bc</td>
<td>1.0 ± 0.2 c</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

- Values are means ± standard deviation.
- For each significant F-test, group means sharing same subscript are not significantly different from each other.
- N.S. = Not significant.
- Group I: Isolated oesophageal varices.
- Group II: Oesophageal varices with gastric extension.
- Group III: Both oesophageal and gastric varices which are disconnected at the cardia.
- Group IV: Isolated gastric varices.

Table (2): Portal / splenic vein ratio (Pv / Sv) in studied groups.

<table>
<thead>
<tr>
<th>Studied Groups</th>
<th>Number of patients with portal / splenic vein ratio &gt; 1</th>
<th>Number of patients with portal / splenic vein ratio ≤ 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I and II</td>
<td>141 / 150 (94%)</td>
<td>9 / 150 (6%)</td>
</tr>
<tr>
<td>Group III and IV</td>
<td>31 / 50 (62%)</td>
<td>19 / 50 (38%)</td>
</tr>
</tbody>
</table>

Specificity = 94 %
Sensitivity = 38 %
Table (3): Size of portal vein in relation to grade in studied groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Grade</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I &amp; II</td>
<td>III &amp; IV</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>Mean ± SD</td>
<td>n</td>
</tr>
<tr>
<td>Group I</td>
<td>71</td>
<td>15.3 ± 2.0</td>
<td>36</td>
</tr>
<tr>
<td>Group II</td>
<td>8</td>
<td>13.5 ± 2.2</td>
<td>35</td>
</tr>
<tr>
<td>Group III</td>
<td>29</td>
<td>13.7 ± 1.7</td>
<td>11</td>
</tr>
</tbody>
</table>

ANOVA Results:

<table>
<thead>
<tr>
<th>Effect</th>
<th>P value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade</td>
<td>0.015</td>
<td>Yes</td>
</tr>
</tbody>
</table>

ANOVA Results: Analysis of variance results, P-value ≤ 0.05 is considered significant.
Grade: Patients in group II with grade III or IV oesophageal varices have a larger portal vein diameter.

Table (4): Size of splenic vein in relation to grade in studied groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Grade</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I &amp; II</td>
<td>III &amp; IV</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>Mean ± SD</td>
<td>n</td>
</tr>
<tr>
<td>Group I</td>
<td>71</td>
<td>10.0 ± 3.3</td>
<td>36</td>
</tr>
<tr>
<td>Group II</td>
<td>8</td>
<td>10.0 ± 2.6</td>
<td>35</td>
</tr>
<tr>
<td>Group III</td>
<td>29</td>
<td>11.3 ± 2.7</td>
<td>11</td>
</tr>
</tbody>
</table>

ANOVA Results:

<table>
<thead>
<tr>
<th>Effect</th>
<th>P value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade</td>
<td>0.383</td>
<td>No</td>
</tr>
</tbody>
</table>

ANOVA Results: Analysis of variance results, P-value ≤ 0.05 is considered significant.
Grade: No significant difference.
### Table (5): Frequency of red colour in relation to bleeding in studied groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>No</th>
<th>Yes</th>
<th>P - value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>Yes</td>
<td>P - value</td>
</tr>
<tr>
<td>Group I</td>
<td>10 (12.8%)</td>
<td>12 (41.4%)</td>
<td>0.002</td>
</tr>
<tr>
<td>Number of cases</td>
<td>78</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Red colour sign</td>
<td>12</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Group II</td>
<td>3 (18.8%)</td>
<td>12 (44.4%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Number of cases</td>
<td>16</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>Red colour sign</td>
<td>12</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Group III</td>
<td>3 (18.8%)</td>
<td>12 (44.4%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Number of cases</td>
<td>21</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>Red colour sign</td>
<td>12</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Group IV</td>
<td>0 (0.0%)</td>
<td>5 (26.3%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Number of cases</td>
<td>3</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Red colour sign</td>
<td>12</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>All groups</td>
<td>19 (16.1%)</td>
<td>29 (35.4%)</td>
<td>0.002</td>
</tr>
<tr>
<td>Number of cases</td>
<td>118</td>
<td>82</td>
<td></td>
</tr>
<tr>
<td>Red colour sign</td>
<td>13</td>
<td>20</td>
<td></td>
</tr>
</tbody>
</table>

### Table (6): Grade of oesophageal varices in relation to bleeding in studied groups.

<table>
<thead>
<tr>
<th>Grade</th>
<th>No number (%)</th>
<th>Yes number (%)</th>
<th>P - value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I &amp; II</td>
<td>75 (69.4%)</td>
<td>33 (30.6%)</td>
<td></td>
</tr>
<tr>
<td>Grade III &amp; IV</td>
<td>40 (48.8%)</td>
<td>42 (51.2%)</td>
<td></td>
</tr>
</tbody>
</table>
Table (7): Size of portal vein in relation to ascites in studied groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>No</th>
<th></th>
<th>Yes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Mean ± SD</td>
<td>n</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Group I</td>
<td>56</td>
<td>15.1 ± 1.8</td>
<td>51</td>
<td>15.9 ± 2.8</td>
</tr>
<tr>
<td>Group II</td>
<td>22</td>
<td>16.3 ± 3.0</td>
<td>21</td>
<td>15.9 ± 3.1</td>
</tr>
<tr>
<td>Group III</td>
<td>21</td>
<td>13.7 ± 2.0</td>
<td>19</td>
<td>13.6 ± 1.9</td>
</tr>
<tr>
<td>Group IV</td>
<td>4</td>
<td>13.0 ± 3.3</td>
<td>6</td>
<td>12.0 ± 1.6</td>
</tr>
</tbody>
</table>

ANOVA Results:

<table>
<thead>
<tr>
<th>Effect</th>
<th>P value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bleeding (yes / no)</td>
<td>0.471</td>
<td>No</td>
</tr>
</tbody>
</table>

ANOVA Results: Analysis of variance results, P-value ≤ 0.05 is considered significant. Bleeding effect: No significant difference.
Table (8): Size of splenic vein in relation to ascites in studied groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Ascites</th>
<th></th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>Mean ± SD</td>
<td>Yes</td>
<td>Mean ± SD</td>
<td></td>
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<tr>
<td>n</td>
<td></td>
<td></td>
<td>n</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group I</td>
<td>56</td>
<td>9.9 ± 2.8</td>
<td>51</td>
<td>10.1 ± 3.4</td>
<td></td>
</tr>
<tr>
<td>Group II</td>
<td>22</td>
<td>12.3 ± 2.8</td>
<td>21</td>
<td>11.3 ± 3.6</td>
<td></td>
</tr>
<tr>
<td>Group III</td>
<td>21</td>
<td>11.5 ± 2.4</td>
<td>19</td>
<td>11.3 ± 3.2</td>
<td></td>
</tr>
<tr>
<td>Group IV</td>
<td>4</td>
<td>12.0 ± 1.6</td>
<td>6</td>
<td>13.0 ± 1.8</td>
<td></td>
</tr>
</tbody>
</table>

ANOVA Results:

<table>
<thead>
<tr>
<th>Effect</th>
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<tbody>
<tr>
<td>Bleeding (yes / no)</td>
<td>0.471</td>
<td>No</td>
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</tbody>
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ANOVA Results: Analysis of variance results, P-value ≤ 0.05 is considered significant. Bleeding effect: No significant difference.
Out of the 150 patients of group I and II (patients with only oesophageal varices and patients with oesophageal varices and gastric extension, respectively), only 9 patients had portal / splenic vein diameter ratio (Pv/Sv) equal or less than one i.e. portal vein diameter was equal or less than the splenic vein diameter. The remaining 141 patients had Pv/Sv > 1. We also found that 19 patients out of the 50 patients of group III and IV had Pv/Sv ≤ 1, while the remaining 31 patients had Pv/Sv > 1. In other words, patients with oesophageal varices alone or with gastric extensions (group I and II) rarely had Pv/Sv ≤ 1 (6%) while patients with isolated gastric varices whether accompanied by oesophageal varices (group III) or gastric varices alone (group IV), frequently had Pv/Sv > 1 (62%).

We found that Pv/Sv has a very high specificity (94%) in diagnosing isolated gastric varices [whether alone (group IV) or accompanied by oesophageal varices but disconnected at the cardia (group III)] when it is ≤ 1. However, the same ratio (Pv/Sv ≤ 1) is much less sensitive (sensitivity = 38%) in diagnosing isolated gastric varices. So we can conclude that portal/splenic vein diameter ratio equal or less than 1 is very specific but not sensitive in detecting patients with isolated gastric varices.

Thus when the sonographic picture of a patient with portal hypertension reveals Pv/Sv ≤ 1 (i.e. portal vein diameter is equal to or smaller than the splenic vein diameter), we should anticipate the presence of isolated gastric varices, which should be carefully searched for during upper endoscopy especially that gastric varices are sometimes difficult to diagnose endoscopically being frequently misdiagnosed for fundal masses as leiomyomas and lymphoma and even exaggerated gastric rugae (Rice et al., 1977). In contrast, gastric extension of oesophageal varices are more easy to diagnose if properly looked for during endoscopy, by following the oesophageal varices down to the cardia and properly examining the cardia from below by retroflexing the endoscope in the stomach. As bleeding gastric varices is more serious and more difficult to control than oesophageal varices, prophylactic sclerotherapy by tissue adhesives is highly universally justified especially after the use of tissue adhesives (Soehendra et al., 1986).

It was found that gastric varices are frequently supplied by short and posterior gastric veins which end directly in the splenic vein. In these patients with gastric varices, it was found that most of the superior mesenteric venous blood is diverted to the splenic vein to flow in a retrograde manner, then it is delivered to the gastric varices through the short and posterior gastric vein and finally it joined the systemic circulation through the azygos venous system. In contrast, oesophageal varices are supplied mainly from the left and right (coronary) gastric which end directly into the portal vein. In these patients with oesophageal varices, it was found that portal venous blood was delivered by the superior mesentric vein to the portal vein, then to the oesophageal varices through the left and right gastric vein and finally to the systemic circulation through the azygos venous system. In patients with both oesophageal and gastric varices, the previously mentioned two pathways contributed equally to portosystemic circulatory shunt.
These patterns of venous flow in patients with different types of various studied by portal venography was confirmed by measurement of venous pressure inside the portal vein through sonography guided percutaneous transhepatic catheterization of portal vein (Watanabe et al., 1988). It was found that the mean portal venous pressure (PVP) of patients with isolated oesophageal varices (EV) was $326 \pm 66$ mmHg while PVP in patients with purgastric varices (GV) was $240 \pm 37$ mmH2O. The difference between the EV and GV groups was found to be significant ($P < 0.01$). The relatively low venous pressure in the portal vein in patients with large gastric varices was due to diversion of most of the superior mesenteric venous blood away from the portal vein (hepatofugal) to flow retrogradely in the splenic vein. On the other hand, with the presence of large oesophageal varices, the pressure inside the portal vein was relatively higher as most of the superior mesenteric and splenic venous blood flows to the portal vein (hepatopetal) (Watanabe et al., 1988).

It was found that there is a good correlation between the portal venous pressure estimated by direct operative measurement and the diameter of the portal and splenic veins measured by abdominal ultrasonography, the higher the portal venous pressure, the wider the diameter of portal and splenic veins (Esmat, 1984). This can explain why the portal vein diameter was larger while the splenic vein diameter was smaller in patients with oesophageal varices compared with patients having gastric varices.

The question remained to be answered is, what determines the distribution of varices, whether gastric, oesophageal or both in response to the increased portal venous pressure? The venous vascular make up of each patient may be the determining factor. In other word, patients whose venous vascular supply is mainly through short and posterior gastric veins will develop gastric varices when they develop portal hypertension while those patients whose venous vascular collateral supply is mainly through right and left gastric veins will develop mainly oesophageal varices in response to increased portal venous pressure (Watanabe et al., 1988).

Another explanation is the presence of an additional route of venous drainage through a pre-existing anomalous vein, that is the polar vein which was present in 35% of patients studied by Vianna et al., (1987). It is about 2.5 cm in length and extends from the posterior wall of the gastric fundus to the middle section of the splenic vein, 7 cm from the confluence with the portal vein (Vianna et al., 1987).

In our work we found that there was a good correlation between the size of the spleen and the diameter of the splenic vein (correlation coefficient $r = 0.06$) i.e. the larger the size of the spleen, the greater the splenic vein diameter. These results agree with that of Laforetune et al., (1984). As there was no significant difference in the size of the spleen among patients of different groups, this good correlation between the size of the spleen and splenic vein diameter did not contribute to the difference of splenic vein diameter among the four groups of patients.

We observed that the diameter of the portal vein (in groups I, II and III) is significantly larger in those patients with
grade III or IV oesophageal varices than in patients with grade I or II oesophageal varices. In contrast, no significant relationship was found between the grade of oesophageal varices and the splenic vein diameter in these patients. This can also be explained on the basis of portal hemodynamics of patients with portal hypertension demonstrated by Watanabe et al., (1988). They found that the mean portal vein pressure in patients with large oesophageal varices and patients whose oesophageal varices were larger than gastric varices was high (326 ± 66 and 340 ± 65 mm H2O respectively) compared to portal vein pressure of patients whose oesophageal varices were smaller than gastric varices (302 ± 75 mmH2O). That difference of portal venous pressure between patients with oesophageal varices and those with gastric varices is due the difference in collateral venous supply of these two types of varices. These results are also in agreement with that of Esmat's study (1984) who found a significant relationship between the grading of oesophageal varices, the portal vein diameter and the portal venous pressure; the larger the grade of varices, the higher the portal vein pressure and the larger the portal vein diameter (Esmat, 1984).

It was found that 29 out of the 82 patients by variceal bleeding had red colour sign (RCS) seen by endoscopy (35.4%), while only 19 out of 118 patients with non bleeding arices had RCS (16.1%). This difference was significant with P = 0.002. These results agree with previous studies which found that red colour sign is the most predictive sign of impending variceal rupture. The Japanese have the longest experience in evaluating the endoscopic criteria to predict future variceal rupture. A total of 2543 patients from 10 medical centers in Japan were followed for a mean of 2.5 years. Bleeding occurred in only 14% of patients without red colour sign and 55% of patients with positive red colour sign (Endo and Fujita, 1983). A similar conclusion was reported by Beppu et al., (1981). It is to be mentioned that red colour sign which is frequently seen over the surface of oesophageal varices was not observed in gastric varices. This finding is agreement with that of Watanabe et al., (1988). Red colour sign (cherry red spots, red wafe marking and hematocystic spots) are dilated minute vessels and capillaries in the mucosa of the oesophagus and seen overlying the oesophageal varices (sometimes called varices over varices). Whereas the veins in the palisade zone (which when dilate form oesophageal varices) lie in the mucosa and lamina propria, the veins in the gastric zone (which when enlarge from gastric varices) lie in the submucosa (Vianna et al., 1987). This may explain absence of red colour sign in cases of gastric varices (Watanabe et al., 1988).

It was also found that patients with large oesophageal varices (grade III and IV) are more liable to bleed than those patients with grade I and II. These results are in agreement with that of Beppu et al., (1981) and Sandy and Feinman (1988). These findings have been recently confirmed by similar studies involving patients with alcoholic and non alcoholic cirrhotic patients (Kleber et al., 1991).

No correlation was found between the presence of ascites and portal and splenic vein diameters. Also no relationship was
established between the presence of ascites and frequency of variceal bleeding.

We also found that portal and splenic vein diameters had no relation to either Child's class or score in studied groups. Child's score reflects the degree of hepatic cellular dysfunction while the portal and splenic vein represent the degree of portal hypertension. Thus our results show that there is no actual relation between the degree of portal hypertension and liver cell dysfunction.

REFERENCES


Mathur S.K., Dalvi A.N., Someshwar V Supe A.N, Ramukantan R (1990) : En-


العلاقة بين نوع الدوالي وحجم الوريد البابي والطحالبي

في مرضى ارتفاع ضغط الدورة البابية

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عند مقارنة مرضى دوالي المعدة بمرضى دوالي المرء، وجد أن دوالي المعدة المقدمة تدم في أحيان كثيرة بواصلة الوريد العددي القصير وخلقي الذين ينتهي مباشر في الوريد الطحالبي، وذلك يمكن دوالي المرء التي من المعروف أنها تم بواسطة الوريد العددي الأمين والأيسر الذين ينتهي مباشر في الوريد البابي.

وقد أجري هذا البحث على مائتين من المرضى الذين يعانون من دوالي المعدة والمريء لدراسة العلاقة بين نوع الدوالي (وذلك بواسطة المنظر العلوي) وحجم الوريد الطحالبي والبابي (وذلك بواسطة الوجبات فوق الصوتية على البطن).

وقد وجد أن الوريد البابي في مرضى دوالي المرء أكبر من الوريد البابي في مرضى دوالي المعدة، وقد كان هذا الفارق ذا دالة إحصائية عالية. وقد وجد أيضًا أن الوريد الطحالبي في مرضى دوالي المعدة أكبر من الوريد الطحالبي في مرضى دوالي المرء. وقد كان هذا الفارق ذا دالة إحصائية عالية أيضًا. وبالتالي فإن نسبة حجم الوريد البابي إلى الوريد الطحالبي كانت أكبر في مرضى دوالي المرء بالمقارنة بمرضى دوالي المعدة. وقد وجد أن نسبة حجم الوريد البابي إلى الوريد الطحالبي لها خصوصية عالية جدا (0.94%)

وخصائص متفاوتة (7/2) في تشخيص مرضى دوالي المعدة، وذلك عندما تكون تلك النسبة أقل من أو تساوي 1. لذا فإنه عندما تشير الموجات فوق الصوتية على البطن إلى أن نسبة حجم الوريد البابي إلى الوريد الطحالبي أقل من أو تساوي 1، فإنه يجب أن تقترب جيدًا دوالي المعدة وبالتالي أن المريء يكون أكبر نوعًا من دوالي المعدة كثيرة ما تتشابه مع أورام المعدة الحميذة والخبيثة أو حتى ثانية المعدة المتوسطة. ولأن تزيف دوالي المعدة أكثر خطورة وأصعب في السيطرة عليه من نزيف دوالي المرء، فإنه يوصي بالتحقيق الوقائي لدوالي المعدة على نطاق واسع وحصوصا بعد استعمال مصادر الأسمدة. لذا فإن التشخيص الدقيق والبديكور لدوالي المعدة أصعب ليس فقط مما ولكن ضروري.

وقد كان حجم الوريد البابي أكبر في مرضى تردد الدوالي بالمقارنة بمرضى الذين لا يتسيل لهم حديثًا تنزيف الدوالي ولكن هذا الفارق لم يكن له دالة إحصائية. وقد أوضح أن حجم الوريد الطحالبي أكبر في مرضى تردد الدوالي بالمقارنة بالمرضى الذين لا يتسيل لهم حديثًا تنزيف الدوالي وقد كان هذا الفارق له دالة إحصائية.
وقد لوحظ أن حجم الروريد البياري أكبر في الرضي الذين وجد عندهم نوالي مرئي متقدمة (درجة ثالثة أو رابعة) بالمقارنة بمرضي نوالي الدرجة الأولى أو الثانية وقد كان هذا الفارق له دلالة إحصائية، بينما لم يوجد أي علاقة بين حجم الروريد الطحالى وحجم نوالي المرئي.

وقد لوحظ أن حجم الروريد البياري والطحالى أكبر في الرضي الذين وجد عندهم علامات حمراء على النوالي وذلك بالمقارنة بالمرضى الآخرين، وقد كان هذا الفارق له دلالة إحصائية.

وقد لوحظ أيضاً أن الرضي الذين وجد عندهم علامات حمراء على النوالي وكذلك مرئي النوالي المتقدمة (درجة ثالثة أو رابعة) أكثر عرضة لتشويه النوالي وذلك بالمقارنة بالمرضى الآخرين، وقد كان هذا الفارق له دلالة إحصائية.

ولم توجد علاقة بين حجم الروريد البياري أو الطحالى وجود الاستشفاء البروتوني أو تصنيف تشخيص.