Brain Lesions in Cerebral Venous Sinus Thrombosis

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Background and Purpose—Analyze the relationship between the location and extent of sinus thrombosis and presence and severity of brain lesions.

Methods—Retrospective chart and neuroimaging review of patients with documented CVST. A CVST score was devised to quantify the extent of cerebral venous sinus thrombosis. 

Results—Nineteen of 56 (34%) patients had brain lesions. The extent of sinus thrombus was associated with increased risk of brain lesions (CVST score 1.9 among patients without brain lesions versus 3.1 in those with lesions; \( P=0.006 \)). Age, sex, and acquired or hereditary thrombophilias were not associated with the risk of parenchymal lesions. Functional outcomes were favorable even in patients with extensive CVST and parenchymal lesions at presentation.

Conclusions—The extent of the sinus involvement correlates with the risk of brain lesions in patients with CVST, but additional factors might also contribute to their occurrence. \((\text{Stroke. 2009;40:00-00.})\)

Key Words: cerebral sinus thrombosis ▪ hemorrhage ▪ stroke ▪ neuroimaging
both groups (62% in group 1 and 74% in group 2). All patients in group 1 presented with gradual onset of headaches without focal signs. In group 2, only 2 patients presented with focal signs despite the presence of established parenchymal lesions on brain imaging; the rest of the patients presented with headaches only.

A defined thrombophilia was present in 11 (30%) patients in group 1 and 6 (32%) in group 2. The combination of factor V Leiden mutation and activated protein C resistance was the most frequent abnormality (n=8), followed by antiphospholipid and anticardiolipin antibodies (n=3), protein S deficiency (n=2), prothrombin G20210A mutation (n=2), and

Figure 1. Distribution of parenchymal lesions in relationship to occluded venous sinuses. Each illustration is accompanied by a list of occluded sinuses. SSS indicates superior sagittal sinus; StS, straight sinus; TS, transverse sinus; SS, sigmoid sinus; vG, vein of Galen; ICV, isolated cortical vein; ICVs, internal cerebral veins.
Table. Distribution of Venous Sinus Occlusion*

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=37)</th>
<th>Group 2 (n=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Straight sinus</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Transverse sinus</td>
<td>32</td>
<td>15</td>
</tr>
<tr>
<td>Sigmoid sinus</td>
<td>16</td>
<td>9</td>
</tr>
<tr>
<td>Internal cerebral veins</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Vein of galen</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Isolated cortical vein</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Superior sagittal sinus</td>
<td>9</td>
<td>11</td>
</tr>
</tbody>
</table>

*Most patients had multiple sites of occlusion.

Group 1: patient with sinus thrombosis, but without parenchymal lesions detected by neuroimaging; Group 2: patient with parenchymal lesions, related to sinus thrombosis.

MTHFR mutation (n=1). One patient had a myeloproliferative disorder most consistent with essential thrombocythemia.

The parenchymal abnormalities found in group 2 patients are illustrated in Figure 1, and the association of these abnormalities with specific location of sinus occlusion is summarized in the Table. As expected, deep cerebral vein thrombosis caused bilateral basal ganglia lesions. Occlusion of transverse or sigmoid sinuses caused mostly unilateral posterior temporal lobe lesions. Superior sagittal sinus thrombosis resulted in lesions that were quite variable in size, ranging from minimal low attenuation changes (patient 6 and 10) to large hemorrhagic infarctions (patient 12). Bilateral lesions were infrequent in patients with superior sagittal sinus thrombosis (only present in 1/11 patients) but uniformly present when the vein of Galen and internal cerebral veins were occluded.

The mean CVST score was 1.9 in group 1 and 3.1 in group 2 (P=0.0019). CVST scores ≥4 were also more common in group 2, occurring in 42% of patients in this group versus only 14% patients in group 1 (P=0.02, Figure 2). Although the extent of CVST mostly correlated with the presence of parenchymal lesions, there were notable exceptions. Among the patients in group 1, there was one with a CVST score of 6, and two with CVST score of 5. There was also one patient in group 2 with massive venous sinus occlusion but only very limited parenchymal abnormality (patient 6).

The mean follow-up period was 28.5 months for group 1 and 24.3 months for group 2. Clinical outcome was very favorable in all group 1 patients (all had minimal or no symptoms at the end of the follow up), whereas the mean mRS in group 2 patients was 1.9 (P=0.0003). The number of patients in group 2 was too small to analyze the correlation between CVST score and functional outcome. None of the patients in either group had recurrent CVST during follow-up.

Discussion

We found a correlation between the extent of CSVT and the risk of parenchymal damage. Age, sex, and acquired or congenital thrombophilia did not influence the likelihood of presenting with parenchymal lesions. In agreement with previous studies, the outcome was generally very favorable in our patients but worsened by the presence of intraparenchymal lesions.

To our knowledge, this is the first study to provide a CVT score in relation to the distribution of parenchymal lesions. Our smaller study attempted to correlate the site of the CVT to the distribution of parenchymal lesions in 26 patients, but no significant correlation between the extent and site of CVST and the extent and location of brain lesions was found.

In our study, although the extent of CSVT correlated with the presence of parenchymal abnormalities, there were notable exceptions arguing that there were other factors contributing to the production of parenchymal lesions. Overestimation of the degree of thrombosis by MR venography in areas of very slow venous blood flow could explain some of the discrepancy.

Because of the retrospective nature of our analysis, there was no uniformity in the timing of imaging in relation to the onset of symptoms. This study lacked sufficient power to assess whether specific sinus involvement influences the risk of parenchymal injury.

Disclosures

None.

References

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