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 CTVS. A CTVS 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 (CTVS 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 CTVS and parenchymal lesions at presentation.

Conclusions—The extent of the sinus involvement correlates with the risk of brain lesions in patients with CTVS, but additional factors might also contribute to their occurrence. (Stroke. 2009;40:1509-1511.)

Key Words: cerebral sinus thrombosis ▪ hemorrhage ▪ stroke ▪ neuroimaging

The spectrum of clinical presentation in patients with cerebral venous sinus thrombosis (CVST) varies considerably.

While some patients with CVST have relatively mild symptoms, others develop devastating complications, including hemorrhagic venous infarctions and severe intracranial hypertension. In the largest study of CVST patients reported to date, the indicators of poor prognosis were older age, male sex, depressed level of consciousness, central nervous system infection, cancer, thrombosis of the deep cerebral venous system, and hemorrhage on admission brain scan.1 Among patients with early intracerebral hemorrhage, older men with deep venous system or right lateral sinus thrombosis or motor deficits were more likely to have an unfavorable outcome.2 Thus, it is relevant to investigate what factors influence the occurrence and evolution of these lesions.

This study was undertaken to analyze the relationship between the location and extent of sinus thrombosis and the presence and severity of parenchymal lesions.

Methods

The records of all consecutive patients with CVST treated at Mayo Clinic, Rochester, Minn between November 1996 and July 2005 were reviewed. Inclusion criteria for this study included radiologically documented CVST with clinical and neuroimaging data available for our review. Patients with underlying structural brain lesions and those in whom we could not personally review the images were excluded from the analysis.

Clinical information included age, sex, comorbid conditions, clinical presentation, thrombophilia profile, functional outcome, and CVST recurrence. Radiological data collected included location and extension of the CVST on venography (typically 2D time of flight MR venography), presence, size, and location of the venous infarct, and subsequent enlargement of the hematoma and associated mass effect as seen on CT scan of the head. The reviewer of the radiological images (A.Y.Z.) was blinded to the clinical information. Multiple brain scans were reviewed when available for each patient. CT scans were chosen because most of the patients had only one MRI scan and to ensure that our lesion measurements were consistent across scans. The volume of the parenchymal lesions was measured manually using the previously described ABC/2 method, that is validated for CT scans but not for MRIs.3

A CTVS scoring system was designed to quantify the overall distribution of the sinus clot, assigning 1 point for each sinus that was involved with thrombosis; the superior sagittal sinus was divided into thirds, and 1 point was assigned to each third. Occlusion of each internal cerebral vein and the vein of Galen was assigned 1 point per vein.

Descriptive statistics are reported as mean and range. Outcomes analyzed included short-term mortality and functional outcome as assessed by the modified Rankin score (mRS). One-way ANOVA or Wilcoxon Rank Sums test when appropriate were used to assess relationships between variables. Probability values <0.05 were considered statistically significant. The analysis was performed using JMP statistical software (Version 6, SAS Institute Inc).

Results

Fifty-six patients were included in the study. Patients were subdivided into two groups depending on the presence of parenchymal abnormalities on neuroimaging. There were 37 patients without evidence of parenchymal lesions (group 1) and 19 patients with parenchymal lesions (group 2). There was no significant difference in age between the groups (mean age 42 years [range, 3 to 84 years] in group 1 versus 39 years [9 to 79 years] in group 2). Women predominated in
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
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 sinus 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 CVST score in relation to the distribution of parenchymal lesions. One 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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