Association between Transverse Sinus Hypoplasia and Cerebral Venous Thrombosis: A Case-Control Study

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Background: Hypoplasia of the transverse sinus (TS) is a common anatomical variation. However, the relationship between TS hypoplasia and venous thrombosis has not been studied. We analyzed the hypothesis that TS hypoplasia is a predisposing factor for ipsilateral thrombosis. *Materials and methods:* We retrospectively evaluated 20 confirmed cases with isolated TS thrombosis and 43 age- and sex-matched controls. TS thrombosis and hypoplasia were diagnosed using both computed tomography and magnetic resonance venography. Hypoplasia was defined as a TS diameter less than 50% of the cross-sectional diameter of the lumen of the distal superior sagittal sinus and by a bony groove ratio less than 1.02. Univariate analysis was performed to evaluate the association between TS hypoplasia and thrombosis. *Results:* There were a total of 45 hypoplastic TS: 31 (49%) left hypoplastic TS (12 (60%) cases vs 19 (44%) controls ($P = .24$), and 14 (22%) right hypoplastic TS (9 (45%) cases vs 5 (12%) controls ($P = .003$). TS hypoplasia was more frequently found in cases (n = 18, 90.0%) than in controls (n = 22, 51.2%; relative risk 1.7, confidence interval [CI] 95% 1.3-2.4, $P = .003$).Hypoplastic TS and ipsilateral TS thrombosis showed a significant association ($P = .002$ for right and $P = .008$ for left TS hypoplasia) with relative risk of 3.8 (95% CI 1.3-10) for right and 7.5 (95% CI 1.1-48) for left hypoplasia. No significant association was found between hypoplastic TS and functional outcome at 30- or 90-day follow-up. *Conclusion:* TS hypoplasia might be a predisposing factor for ipsilateral TS thrombosis, but not for functional outcome. **Key Words:** Cerebral venous thrombosis—transverse sinus hypoplasia—lateral sinus thrombosis—stroke.
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Introduction

Cerebral venous thrombosis (CVT) is a rare but serious neurologic disorder that is potentially reversible with prompt diagnosis and appropriate medical treatment. It is associated with a wide spectrum of etiologic factors and clinical presentations.[1,2]

In general, CVT should be considered a multifactorial disease that may be triggered by a variety of genetic and environmental factors. In the ISCVT study,[1] nearly 44% of patients had more than 1 cause or predisposing factor. The proportion of unknown causes is approximately 15%, and there are still many unsolved issues in the pathophysiology, diagnosis, and management of CVT.

The anatomical sites of sinus thrombosis are usually at the superior sagittal sinus (SSS), in 62%-82% of cases, and transverse sinus (TS), in 38%-86% of cases.[2,3] Isolated TS thrombosis is an uncommon finding among CVT cases, comprising 10%-32% of cases in different series.[3,4]

Previous publications have shown that TS are frequently asymmetric. Hypoplasia or aplasia of TS is a common anatomical variation, and right TS is dominant in 61% of cases.[5] Most individuals with TS hypoplasia lack symptoms of CVT; for this reason, the incidence and clinical significance of this condition have likely been underestimated. Asymmetry of the sigmoid notches on noncontrast brain computed tomography (CT) has been proposed as a measure to differentiate TS thrombosis from an atretic sinus,[6] but the relationship between hypoplastic TS and thrombosis of the TS has not been studied.

We conducted this case-control study to evaluate whether hypoplasia of TS is associated with an increased risk of ipsilateral thrombosis and we also investigated if thrombosis in hypoplastic TS is associated with clinical outcome.

Patients and Methods

Study Design and Patient Selection

This study was a retrospective analysis of data collected prospectively from our institutional CVT registry. Consecutive patients with first-ever confirmed CVT treated at the Instituto Nacional de Neurologia y Neurocirugia in Mexico City were enrolled from January 2001 to January 2015. Inclusion criteria consisted of an acute (less than 30 days) isolated TS thrombosis confirmed by magnetic resonance imaging (MRI) and 3-dimensional (3D) gadolinium-enhanced magnetic resonance venography (MRV), CT, CT venography (CTV), or conventional angiography, and complete follow-up as an outpatient for at least 90 days. All patients underwent a standardized diagnostic follow-up and treatment protocol. Data including demographics, medical history, risk factors, CVT onset time, arrival time at the hospital, complications, treatment, procedures, and ambulatory status at discharge were registered for each patient.

Age- (±3 years) and sex- (2:1 ratio) matched controls were selected among patients who underwent MRI-MRV neuroimaging studies for other neurologic conditions that do not modify the morphology of the venous sinuses, with no history of previous or current CVT. Demographics, risk factors, and clinical and laboratory data were registered for each patient and controls. All CVT cases underwent control MRI-MRV to evaluate recanalization.

Risk factors and predisposing conditions for CVT, such as smoking, anemia, recent use of contraceptives, history of abortions or miscarriage, pregnancy or puerperium, thrombophilia or antiphospholipid syndrome, were investigated.

All patients were treated according to our institutional guidelines (developed based on the clinical experience rather than experimental data) with intravenous heparin followed by oral anticoagulants (OA) for 12 months. Low-molecular weight heparins were used in pregnant patients until delivery and then changed to OA. The intensity of OA was measured monthly up to a level of international normalized ratio of 2.5 and then every 3 months to complete 1 year of treatment.

Prognosis was assessed with the modified Rankin scale (mRS) at 90 days' follow-up. Good functional outcome was defined as an mRS grade of 0-1, and poor functional outcome as an mRS grade between 2 and 5.

Because this investigation was an observational study, based on current practice with no special investigation or procedures, it was exempt from informed consent. However, the Institutional Ethics Committee approved this study.

Diagnosis of TS Thrombosis and TS Hypoplasia

Diagnoses of isolated TS thrombosis was based on clinical signs and confirmed by MRI and MRV. All patients had CTV and MRV. Complete angio-MRI was obtained with a 1.5 T apparatus (Milwaukee, WI, USA) (General Electric Medical Systems Signa Excite HDxt 1.5 T, serial number 2884783 MR) and a 3 T apparatus (Milwaukee, WI, USA) (General Electric Medical Systems Signa HDxt 3.0 T, serial number 5606 MRS3T). The sequence we used was Inhance 3D Velocity, with TE 4.4-5.6 ms, TR 11.2-11.9 ms, 1.20-mm thick serial images and 512 × 512 resolution. TS thrombosis was confirmed with the visualization of the thrombus itself (hyperdensity on CT scan, hypersignal on MRI T1 and T2, hyposignal on MRI T2) together with the nonvisualization of the entire sinus involved using MRI/MRV, CT/CTV, or conventional angiography.

Using previously described methods,[7,8] the diameter of each TS was measured at the distal third, and the diameter of the SSS was measured at the midpoint of an imaginary vertical line drawn from the vertex to the torcula.[8] With these measurements, TS hypoplasia was defined as a TS diameter less than 50% of the cross-sectional diameter of the lumen of the distal SSS. We used

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