E-009 HYPERCOAGULABLE AND GENETICALLY THROMBOPHILIC PATIENTS WITH CEREBRAL VENOUS THROMBOSIS

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Introduction Cerebral venous thrombosis (CVT) may occur due to a number of common etiologies such as thromboembolism, atherosclerotic disease, or small vessel disease. When these are ruled out or considered unlikely, a hypercoagulability workup is performed. We describe a series of 30 patients with CVT and medical and/or genetic basis for the underlying hypercoagulable state and thrombophilia.

Methods A retrospective review of all CVT cases treated with venous thrombectomy between June 2016 and August 2021 was performed within our institutional, neuroendovascular database.

Results Of the 30 patients identified, 18 were associated with a hypercoagulable state and/or thrombophilia. Underlying illness was present in seven (23.3%) patients due to polycythemia vera, systemic lupus erythematosus, a combination of nephrotic syndrome and morbid obesity, a combination of rheumatoid arthritis and diabetes, chronic rejection of a small bowel transplant further complicated by acute renal failure and ARDS, a combination of diabetes, DVT, and a dyslipidemic state, and Covid-19. Hypercoagulable states were identified in seven (23.3%) patients due to elevated Factor VIII (1/7), antiphospholipid syndrome (3/7), and Protein S deficiency (3/7). Genetic thrombophilia was identified in four (13.3%) patients in the form of a heterozygous Factor V mutation in RS06Q (2/4), a heterozygous Prothrombin Factor II mutation in G20210A (1/4), and a homozygous 4G/4G promoter Plasminogen Activator inhibitor I deletion mutation (1/4). Overall, no subset of hypercoagulability (i.e., mutation, disease, transient state) nor hypercoagulability overall was predictive of outcome as measured by recanalization, discharge disposition, or recrudescence likelihood.

Conclusion The most common cause of hypercoagulability was an underlying disease or transient antiphospholipid syndrome/ elevated pro-coagulation factor. While we are unable to report hypercoagulability as a predictive variable of outcome in our cohort, we outline the presence of various coagulopathies within this medically refractory, CVT cohort. While CVT may occur due to many common pathologies, in cases where the cause is unknown a hypercoagulability workup my shed light on mitigating factors underlying the thrombosis.

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E-010 EXPLORING DIFFERENCES IN CORTICAL VOLUME IN ADULTS WITH PULSATILE TINNITUS: A CROSS-SECTIONAL STUDY

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Introduction Pulsatile tinnitus is a debilitating disease in which patient’s hear a whooshing sound that correlates with heartbeat, leading to significantly impaired quality of life. Many etiologies can result in pulsatile tinnitus including, idiopathic intracranial hypertension, dural arteriovenous fistulas and other intracranial vascular anomalies. While prior studies have revealed cortical anatomical differences in patients with chronic tinnitus, predominantly in locations such as the cingulate cortex, these studies are limited by small sample sizes, large age ranges, and excluded or at least failed to differentiate patients with pulsatile tinnitus.

Purpose To evaluate for differences in cortical and subcortical volumes in patients with pulsatile tinnitus

Materials/Methods High-resolution T1 images were successfully obtained from 74 adults consisting of 37 participants with clinical diagnosis of either unilateral or bilateral pulsatile tinnitus recruited from our institution’s pulsatile tinnitus clinic and 37 cognitively normal age matched controls from the local community. Subjects recruited were between the ages of 60 to 70 years old and data was acquired using MPRAGE or BRAVO acquisition protocols. Cortical thickness, cortical volume, and subcortical volume was calculated and analyzed using FreeSurfer for 34 brain regions in each hemisphere. Differences in these metrics for each neuroanatomical location were compared between groups using T-tests assuming unequal distributions and unequal variance. Significant anatomical difference was defined as $p < 0.05$ corrected for multiple comparisons using the Bonferroni corrections.

Results Participants in each group were matched for race, ethnicity, age, and confirmed to be neurocognitively intact without evidence of dementia or mild cognitive impairment. Tinnitus laterality was defined for each patient as well as concurrent idiopathic intracranial hypertension. Significant decreases in cortical volume was observed in the left rostral middle frontal gyrus and lateral orbitofrontal cortex.

Discussion While many studies explore differences in neuroanatomy in chronic tinnitus, very little data exists looking at structural anatomical differences in patients with pulsatile tinnitus. One prior study revealed that patients in a younger cohort from ages 24–53 had decreased cortical volume in the left orbitofrontal cortex as well as decreased volume in the bilateral putamen and right middle occipital gyrus. Our results expand on these prior findings and reveal that patients ages 60–70 with pulsatile tinnitus exhibited statistically significant differences in their cortical anatomy. A larger study is needed to see if similar or different regions are affected in a wider patient age range.


REFERENCES