Case Report

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Acute Ischemic Stroke from Sinovenous Occlusive Disease. tpa or no tpa?

Muhammad Ubaid Hafeez[1*], Syeda Qasim[2], Komal Hafeez[3], Ravi Shastri[4]

[1]Department of Neurology, Cambridge Street, United States

[2]Resident physician, Dept of Pathology RWJBH: RWJBarnabas Health, NJ

[3]Resident physician, Dept of Neurology Baylor College of Medicine, Houston TX

[4]Department of Interventional NeuroradiologyBaylor College of Medicine, Houston TX

[*]**Corresponding author:** Muhammad Ubaid Hafeez, Department of Neuro science, Department of Neurology, Cambridge Street, United States, Tel: 3134661305; E-mail: ubaid_hafeez@yahoo.com

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Abstract

Cerebral venous thrombosis (CVT) is a rare cause of acute ischemic stroke (AIS). There is no existing literature on the use of recombinant tissue plasminogen activator (rtPA) for venous strokes if they present within the window for thrombolysis. In addition, establishing venous thrombosis as a cause of acute stroke when deciding to administer rtPA can be challenging. We present a case of acute venous stroke who received rtPA when she presented within 4.5-hour window. The report discusses early imaging findings and role of thrombolysis in acute phase of CVT.

Introduction

Cerebral venous thrombosis (CVT) is a rare cause of acute ischemic stroke (AIS).[1-3] The progression of venous symptoms is often subacute or chronic and frequently outside of the current therapeutic windows for thrombolytic therapy.[4] We present the case of an acute ischemic stroke in a young woman secondary to CVT with discussion about imaging findings in acute phase as well as role of thrombolysis.

CASE

42-year-old woman with a history of uncontrolled diabetes presented with acute global aphasia and right visual field deficit. Her initial NIHSS was 13. The initial CT Head showed loss of grey white differentiation in the left posterior temporal lobe (Figure 1-A), which was felt to be consistent with early ischemic changes. She received a CT angiogram (CTA) of the head and neck and CT perfusion (CTP) while awaiting more information to determine the exact time of symptoms onset. The CTA did not show any large vessel occlusion or stenosis, although an increased area of vascularity was observed around the area concerning for early ischemia (Figure 1-B). CTP showed increased focal mean transit time (MTT), increased cerebral blood volume (CBV), and a mild focal reduction in cerebral blood flow (CBF) suggestive of an ischemic penumbra (Figure 1-C).

Her last known normal was determined to be 170 minutes prior to her presentation. She was given intravenous tPA according to the acute stroke protocol. Unfortunately, the tPA did not immediately reverse the deficits, and the following day she had a hemorrhagic transformation without an additional clinical decline (Figure 2e).

Her echocardiogram and telemetry monitoring were unremarkable. An MRI and CT venogram demonstrated an occlusive thrombus in the left transverse sinus, sigmoid sinus and proximal left jugular vein (Figure 1-D and 1e). An evaluation of hypercoagulable disorders, infectious etiologies, and malignancies was unrevealing. She was started on therapeutic anticoagulation for 6 months. At a 90-day follow up, she had full comprehension and improving expressive aphasia.

Discussion

Cerebral venous thrombosis (CVT) may be under-recognized as a cause of acute stroke but is more established as a cause of all strokes at 0.5 to 1%.[2] The role of intravenous thrombolysis in the management of CVT is unclear, and the emergency room decisions will be particularly difficult in the acute setting without an established mechanism. To our knowledge, there is no published data on the use of thrombolysis for acute venous strokes presenting within the traditional thrombolysis window for AIS. Anticoagulation is recommended for periods usually of 6 months as long as the underlying case does not become a life-long issue.[5] Understandably, there are no prospective trials evaluating this therapeutic dilemma and only a few case reports or series with descriptive analysis.[6, 7]

Radiological features suggestive of venous thrombosis are hyperdense sinus on non-contrast CT, bilateral involvement or a stroke not limited to an isolated arterial territory[1-3]. A CTA may demonstrate a filling defect in a sinus, or 'empty delta sign' if able to capture the venous phase[1, 4]. In our case, initially the stroke was thought to be arterial in the territory of distal middle cerebral artery branches. CTP showed mildly reduced CBF with increased CBV and MTT which is similar to findings of an arterial stroke with a penumbra. However, upon reviewing the initial neuroimaging, an area of subtle hyperdensity was identified

along the vein of Labbe on initial CT which likely represented the presence of thrombus on presentation. The area of increased vascularity on CTA and increased CBV on perfusion imaging were likely secondary to increased venous congestion due to venous thrombosis.

The case highlights the importance of considering CVT as a rare cause of AIS. Knowledge of acute presentation and proper interpretation of radiographical findings can guide initial management and prevent potentially harmful interventions. The role of thrombolysis in acute venous strokes remains to be explored in larger studies.

Figure 1: Non-contrast CT Head on presentation: Black arrow pointing towards subtle hypodensity in left posterior temporal region. Red arrow pointing towards hyperdensity in left transverse venous sinus. B) CT Perfusion showing mildly reduced cerebral blood flow, increased cerebral blood volume and increased mean transit time.

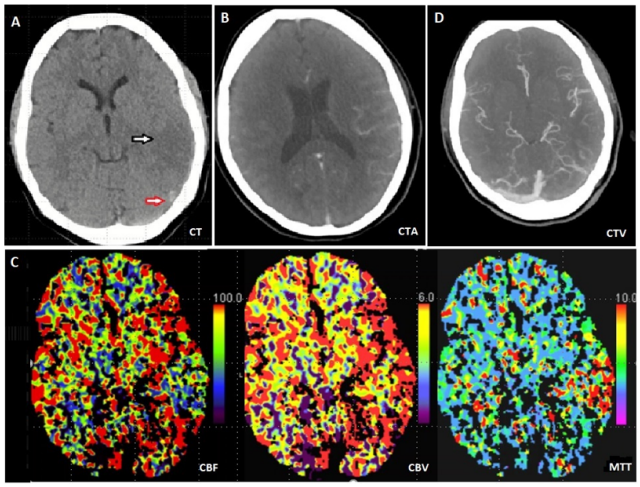


Figure 1e: A) Axial MRI Flair and T2 (right) suggesting filling defect in left transverse sinus and B) Axial CT Venogram (left) later confirmed venous sinus thrombosis.

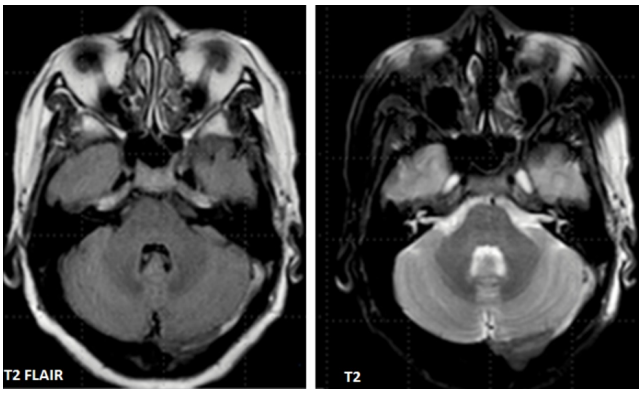
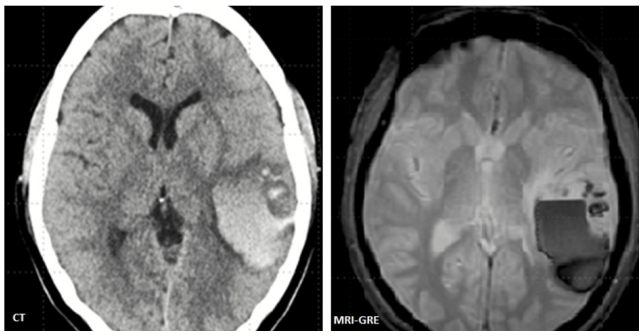


Figure 2: Post-tpa axial unenhanced CT scan on left and MRI GRE sequence on right showing hemorrhagic conversion.



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