Case Report

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Case of Cerebral Venous Thrombosis with Unusual Venous Infarcts

Radiology Section

RAMAKRISHNA NARRA[1], SUSHEEL KUMAR KAMARAJU[2], BHIMESWARAO PASUPALETI[3], NAGANARASIMHARAJU JULURI[4]

ABSTRACT

Cerebral venous thrombosis is a relatively rare condition when compared with vascular accidents of arterial origin representing 0.5-1% of all strokes. Unlike arterial infarcts parenchymal changes are seldom present and when present most of the times are reversible. We present a case report of 28-year-old female with thrombosis of internal cerebral veins and straight sinus and hemorrhagic infarcts in bilateral basal ganglia and bilateral thalami .The findings of bilateral symmetrical hyper intensities in basal ganglia and thalami on MRI may be due to various causes of diverse etiology and cerebral venous thrombosis remains an important cause. Early recognition and prompt anticoagulation therapy helps to reduce the mortality to a great extent. The MRI imaging features of straight sinus thrombosis and other imaging differentials are discussed.

Keywords: Hemorrhagic infarcts, MR Venogram, Obstructive hydrocephalus

CASE REPORT

A 26-year-old female was referred from the Department of Neurology for an MRI presented with drowsiness, altered sensorium, apathy, confusion and later loss of consciousness. She gave birth to a child five days back. Other postpartum history was insignificant. On physical examination tachycardia was present and her GCS was 7.

On MRI, hemorrhagic infarcts were seen in bilateral thalami and basal ganglia causing mass effect on frontal horns of lateral ventricle and third ventricle with obstructive hydrocephalus due to third ventricular outlet obstruction. Periventricular seepage was noted around the frontal and occipital horns of lateral ventricles [Table/Fig-1].

On gradient images petechial hemorrhages and thrombus in the straight sinus which appears as a linear hypointense signal could be seen [Table/Fig-2]. On diffusion imaging areas of restrictions were noted in the basal ganglia and thalami [Table/Fig-3a&b].

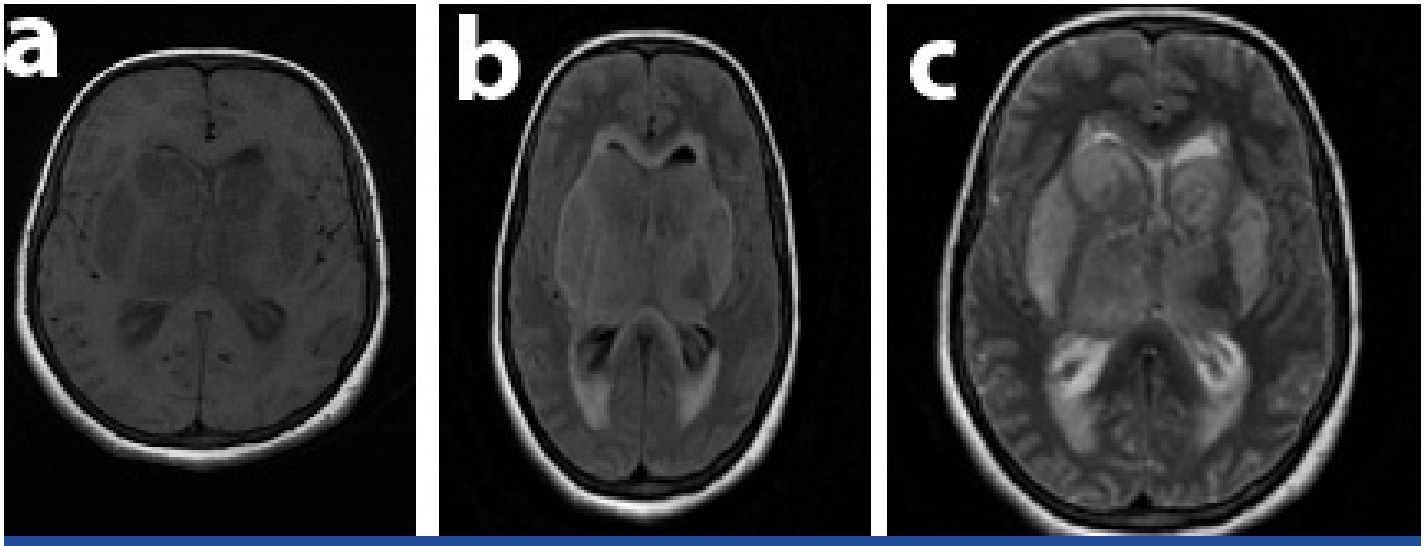
3D TOF MR venogram showed complete occlusion of straight sinus, internal cerebral veins confirming the presence of occlusion due to the thrombus [Table/Fig-4].

Based on patient's history, clinical features and imaging findings a diagnosis of cerebral venous thrombosis was considered. The patient was managed initially with intravenous fluids, anticoagulation (injection heparin) was started and with, antioedema measures (mannitol). Unfortunately three days later the patient died.

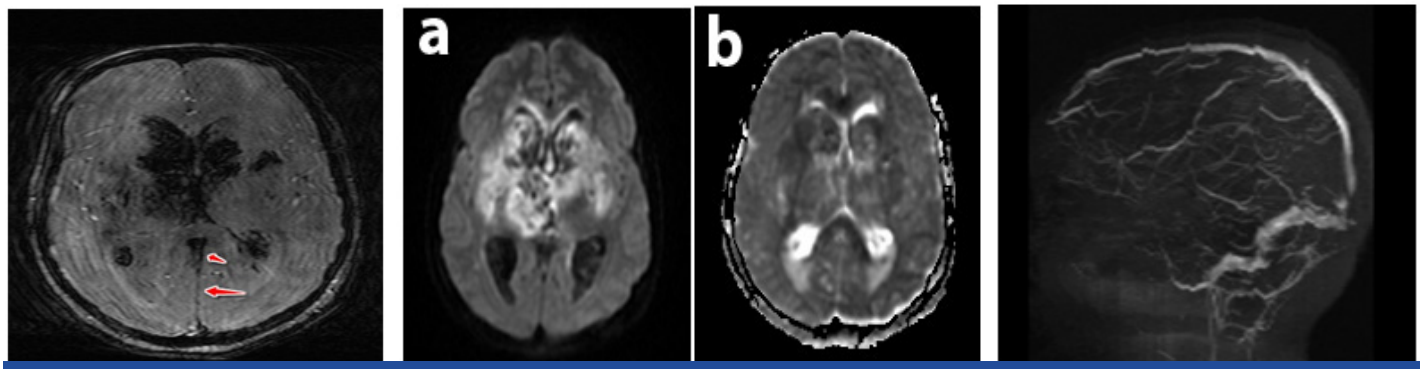
DISCUSSION

Cerebral venous thrombosis is relatively uncommon condition which occurs commonly in the young and early adults. Approximately representing 0.5%-1% of all strokes it is associated with multiple risk factors or predisposing conditions which include pregnancy ,dehydration, infections, medical conditions such as thrombophilias, inflammatory bowel disease, medications such as oral contraceptives, and head injury [1,2].

In the absence of hemorrhage or infarction the imaging findings of CVT on an unenhanced CT are very subtle. A linear hyperdensity of the thrombus may be visualized within a venous sinus or a cortical vein referred to as "Dense sinus sign" and "Cord sign" respectively. On contrast enhanced scan or a CT Venogram a filling defect may be seen referred to as "Empty delta sign". Compared to DSA which is considered as a gold standard CT Venogram has a reported sensitivity of 95% in the diagnosis of CVT [1,2]. The potential pitfalls of CT Venogram include the filling defects of arachanoid granulations or Pacchionian bodies which may be confused with a thrombus. Compared to CT, MRI has an advantage of visualising both the thrombus within the vessel and parenchymal changes of hemorrhagic infarctions more accurately. In acute stage the thrombus is isointense on T1W and hypointense on T2W images. Hence, on T2W images in acute stage thrombus may mimic a flow



[Table/Fig-1]: Axial TIW (a) FLAIR (b) T2W (c) MRI showing heterogenous signal in bilateral basal ganglia and thalami representing hemorrhagic infarcts. Note compression on bilateral frontal horns and periventricular seepage



[Table/Fig-2]: Axial gradient images showing extensive blooming in bilateral thalami and basal ganglia representing hemmorhages.Note the presence of thrombus as a liner hypointensity in the traight sinus (arrows) **[Table/Fig-3a,b]:** Axial diffusion,adcmri images showing areas of restriction due to reduced adc in the bilateral basal ganglia and thalami **[Table/Fig-4]:** 3D time of flight mr venogram showing complete occlusion of straight sinus and internal cerebral veins

void. The thrombus in subacute stage is hyperintense on T1W images. On gradient imaging or suceptability imaging "blooming" of the clot may be seen which appears as hypointense signal [3]. Subtle parenchymal changes of cerebral oedema can be identified on MRI even before the hemorrhagicinfaction or neurological dysfunction ensues [4]. 2D Time of Flight (TOF) MR Venogram may demonstrate absence of flow signal within the thrombosed sinuses. Compared to CT Venogram it is usually performed without contrast and is dependent on the signal of flowing blood within the vessel/sinus [4]. Potential pitfalls of MR Venogram include the presence of physiological flow gaps which may mimic an occluded sinus, in case of hypoplastic sinuses and recanalization of thrombus. This may be overcome by performing an contrast enhanced MR venogram [5]. Although digital subtraction angiography has historically been the gold standard, with the advent of recent advanced non invasive CT and MR venogram techniques and the invasive nature of the DSA has led to a dramatic decline as it's use as a primary mode of diagnosing cerebral venous thrombosis.

There are some characteristic patterns of brain parenchymal changes that distinguish CVT from other entities [6]. Also, to some extent, lesions related to specific sinuses are regionally distributed. Brain parenchymal changes in frontal, parietal, and occipital lobes usually correspond to superior sagittal sinus thrombosis. Temporal lobe parenchymal changes correspond to lateral (transverse) and sigmoid sinus,and vein of labbe thrombosis [6]. Deep parenchymal abnormalities, including thalamic hemorrhage, oedema, or intraventricular hemorrhage, correspond to thrombosis of the vein of Galen or straight sinus [7]. Causes of bilateral basal ganglia and thalamic signal changes on MRI include a wide variety of differentials [Table/Fig-5]. Clinical correlation and other imaging findings help in further differentiation [8]. The present case showed bilateral extensive hemorrhagic infarcts in basal ganglia in addition to thalami possibly due to thrombus extending and involving thalamostriate veins draining the basal ganglia.

Pathology	Etiologic Condition	Structures Involved	Others Areas Involved	Assosiated Clinical Laboratory Features
	Wilsons Disease	Bgandventrolateralthalamus(Decreasedadc)	Mid Brain,Cerebellum Cerebral Hemispheres,Giant Panda Sign Seen In Midbrain	K-F Ring,Cirrhosis Liver,Ceruloplasmin,Copper Levels
	Osmotic Myelinolysis	Basal Ganglia And Thalamus	Pons-Trident Sign	Rapid Hyponatremia Correction
	Wernickes Encephalopathy	Thalami (Medial)	Periaqueductal Gray Matter,Tectal Plate,Mamillary Body	Alcoholic Patients,Low Serum Thiamine Levels
	Hypoxic Ischemic Encephalopathy	Basal Ganglia,Thalami	Cortex,Perirolandic Regions,Reduced Adc	Cardiac Arrest,Near Drowning,Asphyxia
	Leighs Disease	Basal Ganglia,Thalamus	Periaqueductal Region	Muscle Biopsy,Genetic Analysis
	Hyperammonia	Basal Ganglia	Cingulate Gyrus,Insular Cortex	Hyperammonia Levels,Increased Urea Cycle Metabolites
	Liver Disease	Globus Pallidus,Increased T1w Signal	Substantia Nigra	Cirrhosis,Portosystemic Shunt,H/O Tips
	Hypoglycemia	Basal Ganglia	Hippocampus,Corpus Callosum	H/O Diabetes With Treatment Overdose
Toxic	Carbon Monoxide	Globus Pallidus	Delayed Leukoencephalopathy	Elevated Carboxy Hemaglobin
	Methanol	Putamen	White Matter Oedema	Toxicity
	Cyanide	Putamen		Toxicity
Vascular	Deep Venous Thrombosis	Basalganglia,Thalamus, Hemorrhages	White Matter	Risk Factors Present Eg:purperium,Ocp,Dehydration
	Arterial Occlusion (Art Of Percheron)	Thalamus,Red Adc	Pons,Mid Brain Temporal Occipital Cortex	Sudden Onset Of Localising Neurologic Signs
CJD		Bgandthalamus(Decreasedadc,"Pulvinar Sign") May Be Seen	Cortex	Rapidly Progressive Dementia,Myoclonus,
NBIA		GP("Eyeofthetiger"Sign [Hyperintensecenter Withhypointenserim])	Subtantia Nigra Iron Deposition	Genetic Analysis For Pank2 Mutations
Viral Encephalitis		Basalganglia,Thalami	Pons,Cerebellum,Red Nucleus,Substantia Nigra	Serum,Csf Assay, Clinical
Thalamic Gliomas		Thalami		Usually Chidren ,Young Adults

[Table/Fig-5]: Differential diagnosis of various lesions causing bilateral symmetric basal ganglia and thalamic hyperintensities which can mimic straight sinus and internal cerebral veins thrombosis [8]
Abrevations: Nbia-Neurodegeneration With Brain Iron Accumlation,Bg-Basal Ganglia,Gp-Globus Pallidus,Adc –Apparent Diffusion Coefficient

CONCLUSION

Though venous infarcts occur less frequently compared to arterial counterparts they do not strictly confine to a specific territory but a rough lobar and anatomic distribution can provide a clue to the vein/sinus involved. Occlusion of straight sinus, vein of galen can present with hemorrhagic infarcts in bilateral basal ganglia and thalami. Bilateral basal ganglia and thalamic hyperintensites may be present in various conditions and venous thrombosis due to straight sinus occlusion is an important differential especially in the presence of petechial hemorrhages and mass effect.

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PARTICULARS OF CONTRIBUTORS:

1. Assosiate Professor, Department of Radiology, Katuri Medical College, Guntur, Andhra Pradesh, India.
2. Professor, Department of Radiology, Katuri Medical College, Guntur, Andhra Pradesh, India.
3. Professor, Department of Radiology, Katuri Medical College, Guntur, Andhra Pradesh, India.
4. Assistant Professor, Department of Radiology, Katuri Medical College, Guntur, Andhra Pradesh, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Ramakrishna Narra,
Assosiate Professor, Department of Radiology, Katuri Medical College, Guntur, Andhra Pradesh-522019, India.
E-mail : narra.ramki29@gmail.com

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