Indian Journal of
Applied Radiology

Open Science Publications

Volume 7, Issue 1 - 2021

www.opensciencepublications.com

Imaging in Cerebral Sinovenous Thrombosis

Review Article

Shashank Raj[1], Vikas Bhatia[1]*, Pratibha Bhatia[1], Manisha Joshi[2], Arushi Gahlot Saini[3] and Paramjeet Singh[1]

[1]*Department of Radio-diagnosis and Imaging, PGIMER, Chandigarh, India*

[2]*Consultant Neurointervention Radiologist, Rajagiri Hospital, Aluva, Erunakulam, Kerala*

[3]*Department of Pediatrics, PGIMER, Chandigarh, India*

***Corresponding author:** Dr. Vikas Bhatia, Neuroradiology section, Department of Radio-Diagnosis, PGIMER, Chandigarh, India 160012 Email: drvikasbhatia@gmail.com

Article Information: **Submission: 30/10/2020; Accepted: 06/01/2021; Published: 11/01/2021**

Abstract

Background: Cerebral Sinovenous Thrombosis (CSVT) is a frequent pathology associated with a wide range of causative factors and non-specific clinical symptoms which need a timely diagnosis and adequate therapy.

Objective: To describe the imaging features of CSVT.

Material and methods: We review the role of various diagnostic modalities in the management of CSVT. Besides, various mimics and pitfalls in imaging of these cases are described.

Results and conclusion: Imaging plays a key role in the early diagnosis and management of CSVT cases.

Keywords: CSVT; MRI; CTV; Imaging

Introduction

CSVT is a frequent pathology associated with a wide range of causative factors and non-specific clinical symptoms. The projected annual incidence is 2 to 7 cases per million population [1]. We briefly review the underlying pathophysiology, causes, and clinical presentation in CSVT, followed by a radiologic approach to the investigations and management.

Etiopathogenesis

The Superior Sagittal Sinus (SSS) is most frequently involved (66.7%), followed by the lateral and sigmoid sinuses [2,3]. The causative factors in CSVT are numerous and can be broadly divided into local or systemic factors. *Local factors* are related to the inherent or mechanical conditions of the intracranial veins or venous sinuses, which alter the venous flow and may predispose to the development of thrombosis. Common examples include neoplastic infiltration, regional infection such as mastoiditis, or injury caused by trauma. *Systemic factors* may affect the blood flow and dynamics or pertain to an underlying systemic illness. Examples include oral contraceptive use and pregnancy/puerperium, which alter the hormonal homeostasis and lead to a hypercoagulable state. Various systemic illnesses such as dehydration, sepsis, connective tissue disorders can lead to hypercoagulable states. Hematological conditions such as protein S and C deficiencies, factor V Leiden mutation, antiphospholipid syndrome, and vasculitis can contribute to a thrombogenic state. Nevertheless, in nearly 25% of cases, no cause may be identified and are termed as *idiopathic* [2].

Parenchymal changes can occur secondary to the increased venous pressure in thrombosed vessels, especially if the collateral pathway is insufficient. However, if suitable venous collaterals are in existence, parenchymal changes may partially or entirely resolve [4-6].

Clinical presentation

The clinical presentation can range from asymptomatic to severe encephalopathy, coma, or death. The more common neurological features at presentation are acute-onset headache, focal neurological deficits, seizures, raised intracranial pressure, irritability, and altered consciousness. Parenchymal involvement is more commonly associated with focal neurological deficits and seizures. Intracranial hypertension is seen in up to 22-40% of patients with CSVT. Hence, CSVT must be excluded in patients with unexplained intracranial hypertension [4].

ISSN: 2581-3919

Imaging techniques

Non-contrast computed tomography (NCCT): NCCT is the screening imaging technique of choice in these patients as they commonly have a non-specific clinical presentation. The most crucial radiological clue is the 'dense clot sign' (Figure 1), which is visualized as a hyperattenuating thrombus in the occluded sinus [7]. However, it may be seen in only 25% of the cases. Altered attenuation of venous sinuses may be seen in dehydration, elevated hematocrit, or adjacent subarachnoid or subdural hemorrhage. The physiological increase in sinus attenuation can be differentiated from CSVT by comparing it with the arterial attenuation value. The 'cord sign' symbolizes the thrombus of a cortical vein/sinus and is visualized as a linear hyperdensity along the cortical vein (Figure 2). Ancilliary signs include venous infarction involving the subcortical region with sparing of the cortex [8]. It does not correspond to any specific arterial territory. Hemorrhage may appear hyperdense within the infarcted parenchyma (Figure 3). The location of the infarct can give a fair idea about the venous structure involved. For example, superior sagittal sinus often leads to parenchymal changes in the parasagittal position (Figure 4). Vein of Labbe thrombosis involves the temporal lobe (Figure 4), and deep venous system involvement leads to the infarction of thalami, and basal ganglia, including the internal capsule (Figure 4).

CT venography

CT venography is a notable imaging procedure for detecting CSVT [9]. It is considered even superior to conventional TOF MR venography. The thrombus is seen as a filling defect in the dural venous sinus along with enhancement along the periphery which likely results from the development of collaterals and is described as an "empty delta sign" (Figure 1). Additionally, 20% of cases show enhancement of the tentorium as well as falx which is probably secondary to venous stasis and hyperemia [9]. However, a normal NCCT or CECT does not exclude the diagnosis of CVT (false negative in 10-30%) and MR venography needs to be carried out in case of strong clinical suspicion.

MRI

MRI is considered to be a more sensitive modality for the detection of CVTas compared to NCCT. Non-visualization of flow void along with altered SI in the venous sinus is the principal finding of CVT (Figure 5). The SI of the thrombus on T1WI and T2WI is dependent on the age of the thrombus which in turn is dictated by the paramagnetic effect of hemoglobin breakdown products [10,11].

An acute venous thrombus may simulate the signal of a normal flow void (Figure 6). Therefore, contrast-enhanced MRV or CTV is usually important to attain the diagnosis at this stage. The thrombus in the subacute stage is seen in ~ 55 % of the patients at presentation and is the easiest state at which MR can detect a thrombus as SI of the sinus is most distinct as compared to that in a normal flow state (Figure 6) [12]. MR black blood thrombus imaging technique is a 3D variable flip angle TSE T1W technique that quells the signal in the normal vessel and demonstrates the thrombus as a hyperintense signal. Table 1 shows the relative signal of the thrombus at various stages on conventional sequences.

As many as 15% of patients with CVT may have chronic thrombus with incomplete recanalization and may possess a diagnostic challenge at MR images (Figure 7) [11]. Marked enhancement may be observed in the thrombus that can resemble the typical enhancement of a normal sinus [13]. It presumably results from intrinsic vascularization of the organized thrombus having intrinsic vascularisation. Therefore, enhancing the sinus does not unavoidably signify patency and it should be correlated with the MR venography. SWI ((Susceptibility weighted)/Gradient Recalled Echo (GRE) sequences are very sensitive in detecting blood breakdown

Table 1: Venous thrombus appearance on routine MRI.

	RBC product stage	T1	T2
Acute (0-5 days)	Deoxy-Hemoglobin	Isointense	Hypointense
Subacute (6-15 days)	Meth-Hemoglobin	Hyperintense	Hyperintense
Chronic (>15 days)	Hemosiderin	Isointense	Iso/Hyperintense

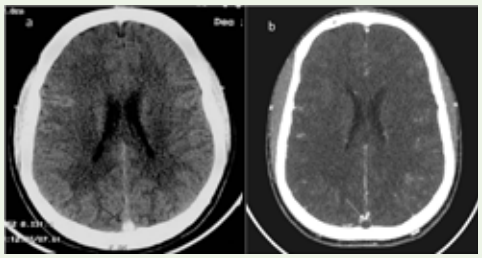

Figure 1: (a) Axial NCCT image shows hyperdense thrombus in SSS (black arrow) known as "dense clot" or "delta sign". (b) Axial CTV in the same patient shows filling defect (white arrow) in the SSS outlined by enhancing dura called "empty delta sign".

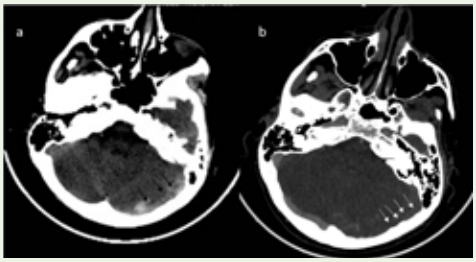

Figure 2: Axial NCCT head (a) showing linear hyperdensity along the left transverse sinus (black arrows) called "cord sign" b. Axial CTV image (b) in the same case shows filling defect (white arrows) in the left Transverse sinus s/o thrombosis.

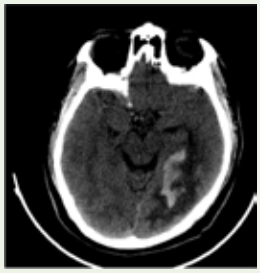

Figure 3: Axial NCCT head shows haemorrhagic venous infarct in left temporoparietal region.

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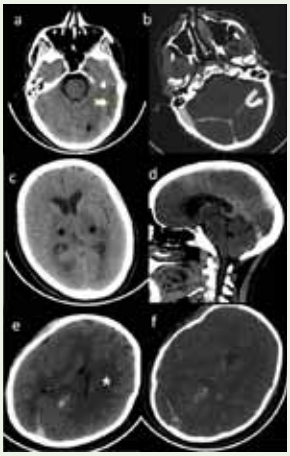

Figure 4: a. Axial NCCT scan showing left temporal lobe infarct (thick white arrow) suggestive of vein of labbe thrombosis b. Axial CTV in the same patient shows filling defect in left TS, Vein of labbe and left sigmoid sinus (curved arrow) s/o thrombosis. c. Axial NCCT head showing b/l basal ganglia and thalamic edema (black star) d. Saggital CTV in the same case shows filling defect in the straight sinus and ICV (black arrows)s/o thrombosis. e. Axial NCCT head shows hyperdense thrombus (white arrow) in the SSS and SS with subtle edema (white star) in left frontoparietal white matter f. Axial CTV in the same patient shows filling defect involving the SSS (white arrow).

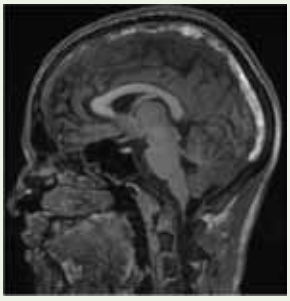

Figure 5: Sagittal T1WI shows loss of normal flow void in the SSS s/o sinus thrombosis.

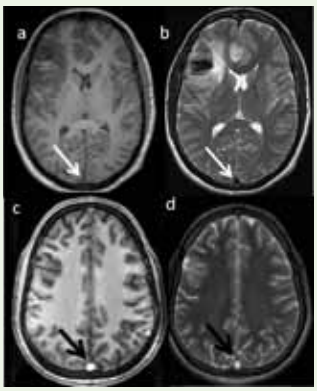

Figure 6: Axial T1WI (a) shows isodense clot in the SSS (white arrow)which in the same patient appears hypointense (white arrow) on T2WI (b) and mimics a normal flow void s/o acute thrombus. Note the haemorrhage with surrounding vasogenic edema in the right frontal lobe (c) Axial T1WI shows hyperintense clot (black arrow)in the SSS s/o subacute thrombus d. Axial T2WI in the same patient shows the hyperintense (black arrow) sub acute thrombus in the SSS.

products and play an important role in diagnosing CVT especially cortical vein thrombosis [11]. Presence of paramagnetic products like deoxyhemoglobin and meth-Hb in the thrombus yield blooming artifact in the venous segments that are thrombosed. SWI/GRE is more important in acute thrombosis where the SI may falsely be normal as discussed earlier. Well delineated tubular hypointensity on SWI also suggests cortical vein thrombosis which can be persistent for weeks and can be associated with underlying cortical/subcortical WM petechial hemorrhages along with sulcal SAH (Figure 8).

Few studies have also evaluated the role of DWI in CVT [14]. Diffusion restriction has been demonstrated in 41% of patients with CVT. It has also been seen that in patients with diffusion restriction complete recanalization was less frequent and the duration of clinical symptoms way longer.

Kalita et al evaluated the role of CVT score, which was computed giving 1 point for each thrombosed sinus and 3 points for SSS. They demonstrated that the CVT score did not correlate with clinical severity and risk factors. Additionally, there was no relation of CVT score with death and 6 months outcome [15].

MR venography

The most commonly used technique is TOF MR venography which is based on the occurrence of flow-related enhancement of spins inflowing into an imaging slice. The 2D TOF technique is preferred

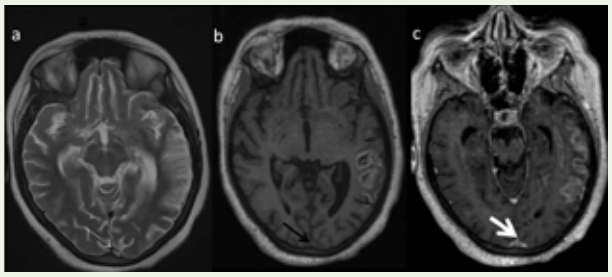

Figure 7: a. Axial T2WI shows hyperintense signal (thin white arrow) within the SSS. b. Axial T1WI in the same case shows the isointense thrombus 9black arrow) in SSS which on T1PC. c. shows partial recanalization (thick white arrow). Findings represent partially recanalized chronic SSS Thombosis. Note the cortical laminar necrosis in the left temporoparietal region.

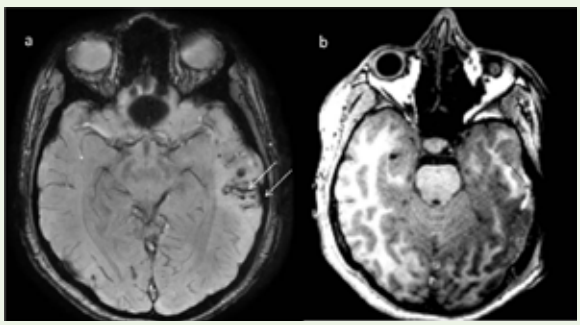

Figure 8: Axial SW image (a) showing tubular hypointensities s/o cortical vein thrombosis along the left temporal sulci (white arrows) with associated subcortical haemorrhages.The thrombosed cortical vein is hyperintense on T1w (b) image (black arrow).

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over the 3D TOF technique. It has reduced sensitivity to signal loss from saturation effect experienced in 3D technique in which a volume of the image is obtained concurrently by phase encoding in the slice select direction. Another technique i.e., phase contrast MRV is also uncommonly used due to its reliance on operator-dependent velocity encoding parameters.

These 2D techniques show the most sensitivity to blood flowing perpendicular to the plane of acquisition. Conversely, blood flowing within the plane of the acquisition can lead to saturation resulting in loss of signal, a known pitfall of TOF MRA. To negate this effect, acquisition in an oblique plane is advantageous. Contrast-enhanced MRV utilizes the T1 shortening effect of gadolinium and helps in improved visualization of smaller vessels as well as venous sinuses as there is a reduction in the artefactual absence of flow due to turbulence, in-plane, or slow flow [16].

According to ACR appropriateness criteria MR venography +/- intravenous contrast is the investigation of choice in a patient suspected of having CVT with a rating score of 9 followed by CT venography with a score of 8 [17].

Parenchymal changes in CVT

In addition to the delineation of vessels on MRI, parenchymal lesions related to CVT are also depicted better on MR imaging as compared to CT. Focal parenchymal edema is visible on CT in ~8% versus 25% cases on MRI [13,18,19]. DWI plays an important role in differentiating vasogenic edema (which shows increased ADC values) from the cytotoxic type of edema (shows reduced ADC values). It has been shown that patients with decreased ADC values more commonly demonstrate parenchymal sequalae whereas in subjects with increased ADC values there may be a complete or near-complete resolution as these are mere because of venous hypertension [20]. Hemorrhage may or may not be associated with both types of edema. Parenchymal enhancement is also seen in 1-29 % of cases which is classically gyral and may also involve the adjacent WM. It likely occurs secondary to damage to the blood-brain barrier (Figure 3) [2].

The role of MR perfusion has also been studied in cases of CVT although the data is scarce. The most common finding in a study was increased MTT with normal rCBV in the areas drained by thrombosed vein [21]. The MTT also showed resolution with treatment on follow up.

A study by Khandelwal et al. [3], in which they compared CTV and MRV, concluded that there is a significant correlation between these techniques. Considering MRVas a gold standard, CT had good sensitivity and specificity of around 75-100%. In another study by Issar et al. MRI was able to detect sinus and parenchymal abnormalities in 100 and 52% as compared to NCCT which was able to diagnose these abnormalities in only 36 and 42% respectively [22].

Deep venous occlusion

It is not uncommon and can be observed in ~ 16 to 20 % of CVT cases. Clinically these patients present with rapidly progressive deterioration of the sensorium and signs of raised ICT and can mimic encephalitis [23,24]. Most striking imaging finding is thalamic vasogenic edema which can be seen in 76% of CT and 86% of MR images (Figure 4) [23]. This edema may extend to involve caudate as well as deep WM. The thrombus may also be visualized on MR images in ICV, VOG, or straight sinus. 19% of cases may also show thalamic hemorrhage and mortality rates being ~ 22 to 37% [23].

Causes of CVT Mimics and Chameleons

CVT Chameleons (Misleading Signs Concealing CVT)

NCCT

- Lack of increased attenuation of a thrombosed sinus. It can occur if the imaging is delayed [25].

- Hyperdense sinus might not be seen due to volume averaging and is most commonly encountered in transverse sinus.

- Hypoplastic dural sinus can be difficult to discern in the background of a hyperdense skull even if thrombosed. It is a frequent finding in which there can be a partial or complete absence of one of the TS. More commonly right TS is bulkier as compared to the left. In about 59% of cases, left TS is atretic (20%) or hypoplastic (39%) [26]. CT may be supportive in these asymmetric sinuses as congenitally hypoplastic TS/SS would have smaller jugular foramina or sigmoid sinus grooves. "Gibraltar sign" was proposed by Pettersson et al to aid in the diagnosis of a dominant TS with a very high PPV. The junction of SSS with the calvarium on axial images shows a resemblance to the Rock of Gibraltar. The direction of the slope of the groove points towards the dominant TS and the opposite side may be hypoplastic [27].

CT venography

The high density of the thrombus sometimes makes it invisible against the background contrast enhancement.

Partially recanalized chronic thrombus

Thrombus with vascularity, may enhance and become isodense to sinus.

MRI

An acute thrombus may mimic flow void as discussed earlier.

Enhancement of the chronic sinus thrombus due to vascularity.

Signal shine through of the thrombus. It occurs due to the T1 shortening effect of the thrombus. The thrombosed segment of the sinus shows intermediate SI on TOF MRV falsely appearing as that of normal sinus flow. However, it shows a lesser intense signal than the other patent veins. Correlation with source images as well as T1 and T2 sequences is of paramount importance.

CVT Mimics

NCCT: Sinuses are normally slightly hyperdense in infants and young children (Figure 9). It is due to higher hematocrit values than in adults as well as lower brain attenuation.

High hematocrit can cause hyperdense sinus. Arteries in these patients also show raised attenuation which is an important clue.

CECT: Fenestrations and arachnoid granulations. Arachnoid granulation may mimic thrombus and are most frequent in the lateral part of TS where the vein of Labbe enters the TS (Figure 10) [28]. However, their round shape &small extent along the dural sinus differentiates them from thrombus. On MRI they show CSF signal intensity.

High or asymmetric bifurcation of the superior sagittal sinus.

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In about 20% of patients with a high bifurcation of SSS can give a "pseudo delta sign" which mimics sinus thrombosis. A similar sign can be seen in head trauma on NCCT images (Figure 11).

MRI: SI of a venous sinus may imitate thrombus on SE MR sequences. The prime causes are decreased flow velocity, in-plane flow& entry slice phenomenon which results in a lack of expected flow void [29]. It is particularly a common finding in children <2 years of age. Slow flow phenomenon more commonly involves left TS, SS, and jugular bulb likely secondary to physiological compression by left brachiocephalic vein during the respiratory cycle [30].

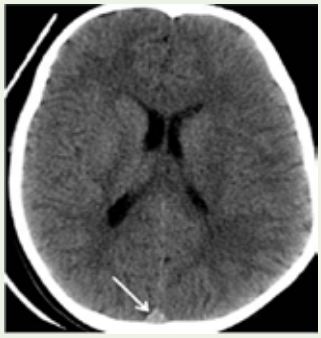

Figure 9: Axial NCCT Head shows hyperdensity (arrow) in the SSS in a young child which occurs due to higher hematocrit and lower brain attenuation.

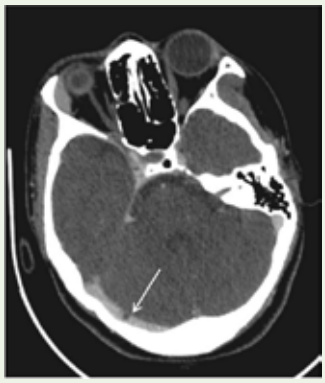

Figure 10: CTV showing round filling defect in the right TS s/o arachnoid granulation.

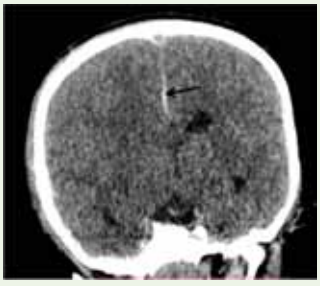

Figure 11: Coronal NCCT Head in a child with subdural hematoma which layers along dura (black arrow) leading to empty delta sign (white arrow) on NCCT (not CECT).

MR venography

Saturation effect due to in-plane flow or decreased flow velocity results in signal loss Hypoplasia/atresia (Figure 12).

Inferior saturation band used to saturate arterial signal can also saturate the cephalad blood flow in the anterior and middle part of the SSS and can lead to signal loss.

Management

Medical management

Systemic anticoagulation and hydration is the cornerstone of treatment. Anticoagulation aims at preventing thrombus propagation rather than dissolving it. A randomized trial consisting of 20 patients revealed significantly improved outcomes in CVT patients who received heparin, even in the coexistence of ICH [31]. Recommendation of the European federation of neurological societies [32]:

Administration of oral anticoagulants for at least 3 months if CVT is due to a transient risk factor.

In the case of mild thrombophilia or "heterozygous" factor V Leiden or prothrombin G20210A mutation, anticoagulants need to be given for 6 to 12 months.

In the case of recurring CVT or a patient with severe thrombophilia like protein C and S mutation, "homozygous" factor V Leiden mutation, antiphospholipid antibody, anticoagulation should be continued for an indefinite period.

Deep venous involvement along with parenchymal hemorrhages if associated with CVT is regarded as poor prognostic factors.

Endovascular management

Mechanical Thrombectomy (MT) and administration of thrombolytics are the endovascular options for CVT (Figure 13). **Indications include: -**

-Patients having neurological deterioration despite anticoagulation and hydration

- Patients in whom anticoagulation is contraindicated

- Patients with ongoing intractable headache [33]

In a study including 52 patients having CVT, MT along with

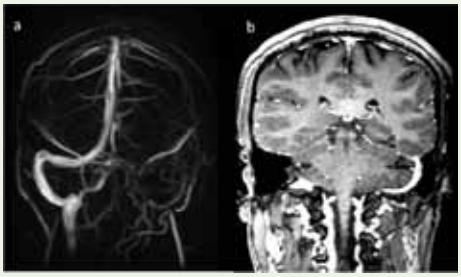

Figure 12: Hypoplastic left TS & Sigmoid sinus which appears thrombosed due to signal loss on 2D TOFMRV. 3D post contrast MPRAGE image shows normal enhancement.

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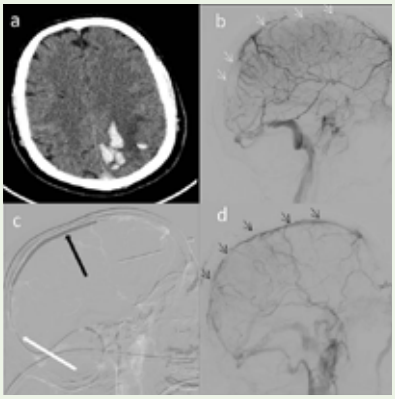

Figure 13: 50 year male with history of seizure and headache. Axial NCCT (a) image shows presence of thrombosed SSS with hemorrhagic venous infarct in left parietal lobe. DSA lateral view venous phase (b) shows non opacified superior saggital sinus (black arrows). DSA lateral view image (c) shows presence of aspiration catheter (thick white arrow) and Angioplasty balloon (thick black arrow). Final DSA lateral view image (d) shows significant recanalization (black arrows) of the SSS post thromoaspiration and angioplas.

urokinase injection lead to complete and partial recanalization of the thrombus in 87% and 6% respectively [34].

Several non-randomized studies to compare endovascular therapy and systemic anticoagulants have shown that the results with endovascular therapy are at par with that of intravenous heparin [35,36].

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Citation: Raj S, Bhatia V, Bhatia P, Joshi M, Saini AG, et al. Imaging in Cerebral Sinovenous Thrombosis. Indian J Appl Radiol. 2021;7(1): 157.

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