Al-Nahrain University College of Medicine Department of Surgery

Research title

Magnetic resonance imaging findings in cerebral venous thrombosis patients

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Dedication

To my beloved parents, who were there for me
with their support and encouragement
i dedicate this work to all their loving tears and beautiful smiles.

To all my respectful teachers, who enlightened me with their knowledge and understanding

To all my fellow students, friends, and colleagues for their unconditional Support and love.

To all patients out there, hoping this little work will do something to help them more in their sufferings.

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List of Abbreviations

CVT	**Cerebral Venous thrombosis**
CVST	**Cerebral venous sinus thrombosis**
MRI	**Magnetic resonant imaging**
MRV	**Magnetic resonance venography**
CT	**Computer tomography**
T1W-FFE	**T1 weighted - fast field echo**
T2W-TSE	**T2 weighted – turbo spin echo**
FLAIR	**Fluid attenuated inversion recovery**
DWI	**Diffusion weighted imaging**
CSF	**Cerebrospinal fluid**
ICP	**Intracranial pressure**
TR	**Repetition time**
TE	**Echo Time**

Abstract

Background: Cerebral vein and dural sinus thrombosis (CVT) is a rare cerebrovascular disease in clinics with highly variable, nonspecific, clinical presentation. The neuroimaging features of CVT can include focal areas of edema or venous infarction, hemorrhagic venous infarction and diffuse brain edema. MRI with MR venography is the most sensitive imaging method for demonstrating the thrombus and the occluded dural sinus or vein with available surgical and medical treatments for this condition.

Aim of the study: We aim in this cross-sectional study to review the commonest MRI abnormalities in patients presenting with cerebral venous thrombosis.

Patients and methods: A cross-sectional analytic study involving 18 patients with suspicious of cerebral venous thrombosis, to the MRI department at Al-imamain Al-Kadhimain medical city, Baghdad/Iraq. With registered and defined significant abnormalities on MRI.

Results: 16 patients had cerebral venous thrombosis confirmed by their MRI examinations, they were 5 males (31%) and 11 females (68%) with male: female ratio of 1:2.5. The most common site of thrombosis encountered was the transverse sinus, it was seen in 15 (93%) patients, the remaining were superior sagittal sinus thrombosis. Regrading secondary cerebral parenchymal changes due to venous thrombosis, they were encountered in 9 (56%) patients and identifiable risk factors found in 11 (68.8%) patients.

Conclusion: Magnetic resonance imaging is the modality of choice to diagnose the cerebral sinus thrombosis and detect its complications. Superficial sinuses involvement was the most common type encountered. The transverse sinus was most commonly involved and infarctions with hemorrhage was the most common abnormal secondary cerebral parenchymal changes found on MRI due to venous thrombosis.

Key words: Cerebral Sinus thrombosis, MRI

Chapter One

Introduction

1. Introduction

Cerebral vein and dural sinus thrombosis (CVT) is a rare cerebrovascular disease in clinics, and less common than most other types of stroke but can be more challenging to diagnose. [1] It results from thrombosis of the intracranial veins and dural venous sinuses that drain blood from the brain. [2] Because of its myriad causes and presentations, CVT is a disease that may be encountered not only by neurologists & neurosurgeons, but also by emergency clinicians, internists, oncologists, hematologist, obstetricians, pediatricians, & family practitioners. [3, 4]

1.1 Epidemiology

The available data suggest that CVT is uncommon, the annual incidence ranges from 0.22 to 1.57 per 100,000, accounts for 0.5-2.0% of strokes and is more common in women than men, with a female to male ratio of 3:1. [5, 6] The imbalance may be due to the increased risk of CVT associated with pregnancy and puerperium and with oral contraceptives. [7] It is easily missed or misdiagnosed by clinicians, its mortality rate is 30-50% according to early reports, so, it is considered rare high-risk cerebrovascular disease. With the development of imaging technology, the early diagnosis and treatment of CVST is gradually becoming possible. [8] According to previous studies, its mortality rate has dropped to 9.4%. [9,10]

1.2 Anatomical considerations

The intracranial venous system consisting of the dural venous sinuses and cerebral veins are traditionally categorized into the superficial and deep cerebral venous systems (Figure 1.1). In the superficial venous system, cortical veins drain into the superior sagittal sinus, which drains into the torcular and then predominantly into the dominant right transverse (lateral) sinus, and right internal jugular vein. The superficial dural sinuses includes The Superior Sagittal Sinus (SSS), the cavernous sinuses, the straight sinus, and the lateral sinuses. The deep system includes the inferior sagittal sinus, the basal vein of Rosenthal, and two paired internal cerebral veins that empty into the vein of Galen and straight sinus and then into the torcular and the smaller-caliber left transverse (lateral) sinus and left internal jugular vein. [11] The flow in the dural venous system occurs typically in rostro-caudal direction. The internal jugular veins appear to drain blood primarily in the supine position whereas the vertebral venous plexus drainage occurs mainly in the upright position in humans. [13]

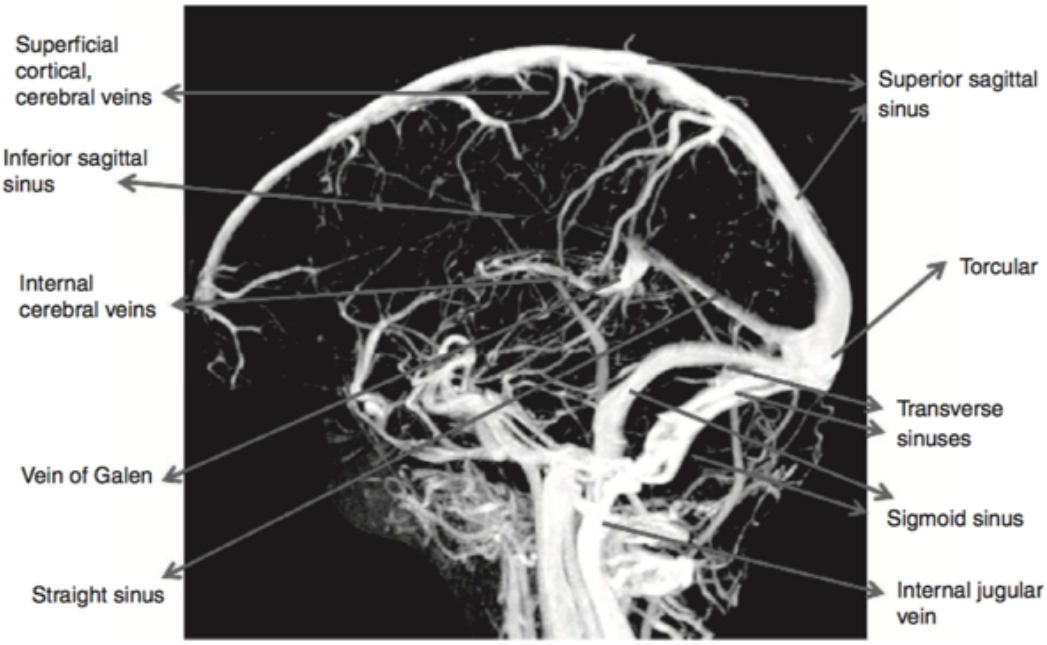

Figure 1.1: MR venography detecting the anatomical components of the intracranial venous system [50]

1.3 Pathogenesis

The pathogenesis of CVT remains incompletely understood because of the high variability in the anatomy of the venous system.

However, there are at least two different mechanisms that may contribute to the clinical features of CVT [14]:

- Thrombosis of cerebral veins or dural sinus obstructs blood drainage from brain tissue, leading to increased venous and capillary pressure. [15]
- Occlusion of dural sinus resulting in decreased cerebrospinal fluid (CSF) absorption and elevated intracranial pressure. [16] Obstruction of the venous structures results in increased venous pressure, leads to blood-brain barrier disruption, causing vasogenic edema, with leakage of blood plasma into the interstitial space. As intravenous pressure continues to increase, mild parenchymal changes, severe cerebral edema, and venous hemorrhage may occur due to venous or capillary rupture. [17]

  The increased intravenous pressure may lead to an increase in intravascular pressure and a lowering of cerebral perfusion pressure, resulting in decreased cerebral blood flow (CBF) and failure of energy metabolism.

  In turn, this allows intracellular entry of water from failure of the Na+/K+ ATPase pump, and consequent cytotoxic edema. [18]

1.4 Risk factors and causes

The most frequent risk factors for CVT are prothrombotic conditions, either genetic or acquired, oral contraceptives, pregnancy and the puerperium, malignancy, infection, head injury and mechanical precipitants. [15] In more than 85 percent of adult patients, at least one risk factor for CVT can be identified, most often a prothrombotic condition. In the Canadian pediatric ischemic stroke registry, a risk factor was identified in 98 percent of the children. [19] A prothrombotic state was found in 41 percent. In infants older than four weeks of age and in children, head and neck disorders, mostly infections and chronic systemic diseases (e.g., connective tissue disease, hematologic disorder, and cancer) were common. The most common risk factors in those ≥65 years old are genetic or acquired thrombophilia, malignancy, and hematologic disorders such as polycythemia. [20, 21] As with venous thrombosis in other parts of the body, multiple risk factors may be found in about half of adult patients with CVT. No underlying etiology or risk factor for CVT is found in approximately 13 percent of adult patients. In older adult CVT patients, the proportion of cases without identified risk factors is higher (37 percent) than it is in adults under age 65. [20]

1.5 Clinical aspects

Cerebral vein and dural sinus thrombosis has a highly variable, nonspecific, clinical presentation, thus, making difficulty in definite diagnosis. [22] The onset can be acute, subacute, with symptoms evolving over days to weeks, and chronic. [23] In general, symptoms and signs of CVT can be grouped in three major syndromes: Isolated intracranial hypertension syndrome, in most patients with deep venous sinus thrombosis. [24] as headache, usually the first symptom, with or without vomiting, papilledema, and visual problems. more frequent in patients with a chronic presentation than in those who present acutely. [25]

Second major syndrome is Focal syndromes as focal deficits, seizures, or both and the third major syndrome is encephalopathy (multifocal signs, mental status changes, stupor, or coma. [3, 15] Less common presentations include cavernous sinus syndrome, subarachnoid hemorrhage, and multiple cranial nerve palsies. A case of CVT mimicking a transient ischemic attack has also been reported. [26]

The clinical symptoms and signs in CVT depend upon several factors, including patient age and sex, the site and number of occluded sinuses and veins, the presence of parenchymal brain lesions, and the interval from CVT onset to presentation. In children, signs of diffuse brain injury, coma, and seizures are the main clinical manifestations, especially in neonates. [19] In older children, the manifestations of

CVT resemble those in adults, with headache and hemiparesis. [27] Women are more likely than men to have a headache on presentation, and less likely to have a chronic onset of symptoms. [11] Older adults may also have a distinctive presentation; mental problems are common while headaches and isolated intracranial hypertension are less frequent than in younger patients. [20]

1.6 Neuroimaging

The neuroimaging features of CVT can include focal areas of edema or venous infarction, hemorrhagic venous infarction, diffuse brain edema, or (rarely) isolated subarachnoid hemorrhage. [15] In patients with CVT, the proportion who present with intracerebral hemorrhage is 30 to 40 percent. [28, 29] Brain MRI in combination with magnetic resonance (MR) venography is the most informative technique for demonstrating the flow, presence of dural thrombus, cortical vein thrombosis, extent of brain injury, edema, infarction, and concurrent abnormalities. [30-33] Head CT is often normal in patients with CVT, and MRI techniques for confirming the diagnosis are not readily available in some hospitals and geographic locations. In this situation, CT venography is a useful alternative to MR venography or intra-arterial angiography for the diagnosis of CVT, demonstrating filling defects, sinus wall enhancement, and increased collateral venous drainage. [38, 39] When combined with head CT, it adds considerable information in suspected cases of CVT. [34] The overall accuracy of head CT combined with CT venography is 90 to 100 percent, depending on the occlusion site. [41]

MRI — MRI using gradient echo T2-susceptibility-weighted sequences in combination with MR venography is the most sensitive imaging method for demonstrating the thrombus and the occluded dural sinus or vein. [32, 36] The characteristics of the MRI signal depend on the age of thrombus: [37, 38]

- In the first five days, the thrombosed sinuses appear isointense on T1-weighted images and hypointense on T2-weighted images
- Beyond five days, venous thrombus becomes more apparent because signal is increased on both T1- and T2-weighted images
- After the first month, thrombosed sinuses exhibit a variable pattern of signal, which may appear isointense

On gradient echo T2-weighted MRI sequences, the clot can be directly visualized as an area of hyper-intensity in the affected cortical vein and/or sinus. [29-31] However, a chronically thrombosed sinus may still demonstrate low signal on these sequences.

Limited data from a series of 28 patients with CVT suggest that the presence of hyperintensities in the veins or sinuses on diffusion-weighted MRI sequences predicts a low recanalization rate. [39]

Parenchymal brain lesions secondary to venous occlusion, including brain swelling, edema, or venous infarction, appear as hypointense or isointense on T1-weighted MRI, and hyperintense on T2-weighted MRI. Hemorrhagic venous infarcts appear as hyperintense lesions on both sequences. [39]

1.6.1 MR venography — MR venography, usually performed using the time-of-flight (TOF) technique, is useful for demonstrating absence of flow in cerebral venous sinuses (figure 1.2), though interpretation can be confounded by normal anatomic variants such as sinus hypoplasia and asymmetric flow, which can be differentiated from CVT as narrowing in the caliber of the sinus, with no abnormal signal intensity on T2 or Flair, within the territory of the sinuses, and attenuated opacification of the subsequent venous circulation due to low blood flow. [45]

Chronically thrombosed hypoplastic sinus will show absence of flow on two-dimensional TOF MR venography and enhancement on contrast-enhanced MRI and MR venography. [15] MR of inferior sinus is not visualized normally [13]

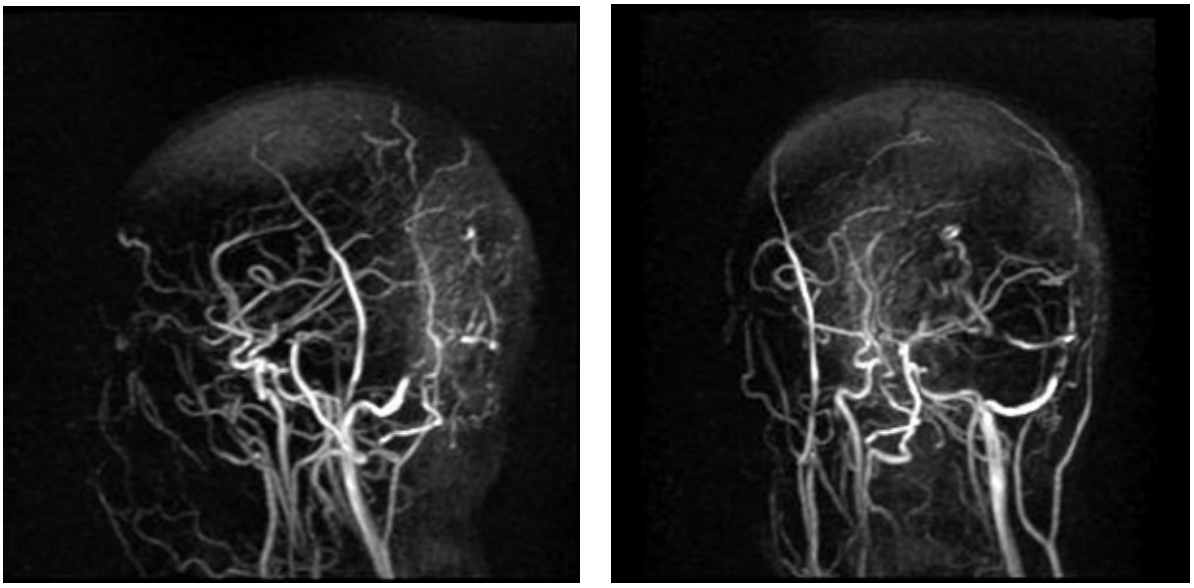

Figure 1.2: MRV- extensive venous sinus thrombosis [51]

1.6.2 CT — Head CT is normal in up to 30 percent of CVT cases, and most of the findings are nonspecific. However, CT is often the first investigation to be performed in clinical practice, and it is useful to rule out other acute or subacute cerebral disorders. [3]

1.6.3 CT venography — CT venography gives a good visualization of the major dural sinuses, is readily available, and is quicker than MRI [40, 41]. It can be used for patients who have contraindications to MRI (e.g., pacemaker). [15] CT venography is often particularly helpful in subacute or chronic CVT because it can demonstrate heterogeneous density in thrombosed venous sinuses. However, its use may be limited because of low resolution of the deep venous system and cortical veins, inexperienced operator, the risk of contrast reactions, and radiation exposure, but still used in suspicious cases. [35, 37, 42].

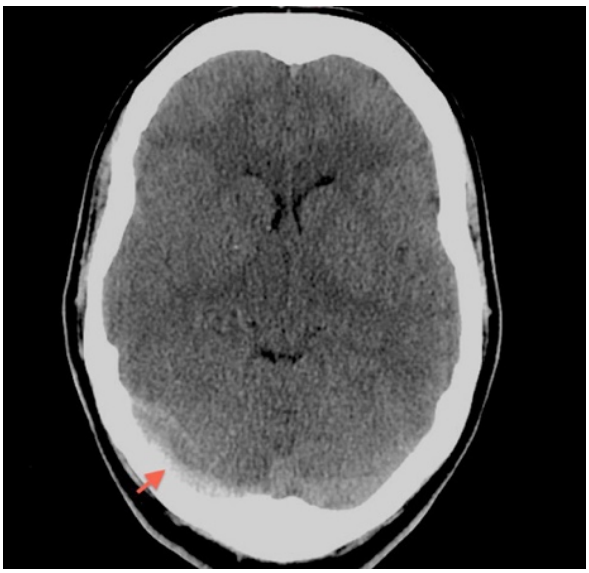

Figure 1.3: Cord sign - transverse sinus [52]

Filling defect, empty delta sign, dense clot, cord sign, are all related to CVT in CT venography. The **cord sign** (figure 1.3) refers to cordlike hyper-attenuation within a dural venous sinus on non-contrast enhanced CT of the brain due to dural venous sinus thrombosis. It is important to appreciate that normal blood within the dural sinuses is usually of slightly increased density relative to brain parenchyma and that true hyperdensity is the key to recognizing thrombosis. [40]

1.7 Treatment

Highly effective medical and surgical treatments are available for decreasing CSF production and for diverting CSF [43], and in some patients intra-sinus stenting has been effective in reducing ICP and treating symptoms [44] Prompt diagnosis is crucial because early intervention, including anticoagulation and systemic or catheter-directed thrombolysis, is associated with favorable clinical outcomes, whereas hemorrhagic complications from dural venous sinus thrombosis can result in devastating neurologic outcomes [45]

Aim of the study

We aim in this cross-sectional study to review the commonest MRI abnormalities in patients presenting with cerebral venous thrombosis.

Chapter Two

Patients and method

2. Patients and methods:

A cross-sectional analytic study involving 18 patients referred from the neurology outpatient clinic with clinical suspicious of cerebral venous thrombosis, to the MRI department at Al-imamain Al-Kadhimain medical city, Baghdad/Iraq

2.1 Patients:

The data were collected from October, 2018 to March, 2019. Their ages range from 7 to 60 with mean age 37 years.
2 patients were excluded from this study because of MRV shows a bad resolution and couldn't be interpreted even by asking another radiologist.

2.2 Inclusion criteria:

- Patients suspected to have CVT with abnormal MRV findings

2.3 Exclusion criteria:

- Poor quality MRI images.

2.4 Imaging technique:

The MRI study was carried out using a 3 Tesla MRI device Philips medical system / Netherland. The following sequences were performed. Each patient was examined while lying in supine position using a head coil, which provides excellent anatomical details and enhances the quality of the MRI images. Slice thickness 5 mm thickness sections and the sequences used were:

- T1 weighted fast field echo images (T1W-FFE) in coronal and axial planes, with the following parameters: Echo time (TE): 3.8, Repetition time (TR): 245.9
- T2 weighted turbo spin echo images (T2W-TSE), in axial plane with the following parameters: Echo time (TE): 80, Repetition time (TR): 3000
- MRV Images in axial and coronal planes with the following parameters: Echo time (TE): 5.63, Repetition time (TR): 15.69
- FLAIR images in coronal plane with the following parameters: Repetition time (TR) = 11000, Echo time (TE) = 120.
- Diffusion weighted images (DWI) in axial plane with the following parameters: Repetition time (TR) = 3340.6, Echo time (TE) = 98.1.

2.4 Image analysis

A significant abnormality was defined as MRI abnormal findings that were registered and these include: complete non-visualization or filling defect as abnormal Signal intensity with the dural venous sinuses at their sites on T1, T2 and MRV, which were correlated and confirmed with feedback from department of neurology. For each patient, the Images was loaded on a CD and reviewed on a personal computer using the software Horos 3.3.

2.5 Statistical analysis

These findings were analyzed and tabulated using Microsoft Excel software (2016 version) and SPSS software version 23 (SPSS Inc., USA). Represented in the form of tables and charts.

Case sheet questionnaire:

Patient ID: _____

Name: _____

Age: _____

Date of examination: _____

Imaging modality: _____

Computer No. _____

Clinical Data: _____

Referral notes:

Consent:

Signature:

Chapter Three

Results

3. Results

During the study period, 16 patients had cerebral venous thrombosis confirmed by their MRI examinations, they were 5 males (31%) and 11 females (68%) with male: female ratio of 1:2.5 as shown in fig.1. Their ages range 7-60 years with mean age of 37 years.

Table 3.1: Gender distribution

Frequency	Percent	
Male	5	31.3
Female	11	68.8
Total	16	100.0

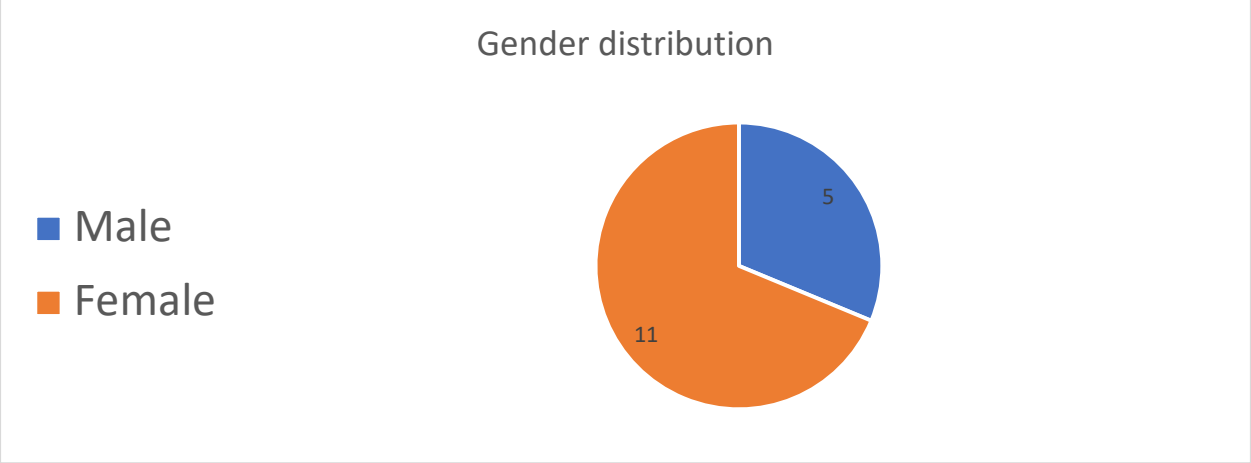

Figure 3.1: Gender distribution

The most common site of thrombosis encountered was the transverse sinus, it was seen in 15 patients (93%), of those, 9 patients (56%) were left transverse sinus thrombosis and 6 patients (37.5%) were right transverse sinus thrombosis.
One patient (7%) had superior sagittal sinus thrombosis

Table 3.2 The distribution of CVT according to anatomical sinus drainage

Sites	Frequency	Percent
left transverse sinus	9	56.3
right transverse sinus	6	37.5
Superior sagittal sinus	1	6.3
Total	16	100.0

No sigmoid or straight sinus thrombosis, were encountered in this study. These findings were shown in fig.2.

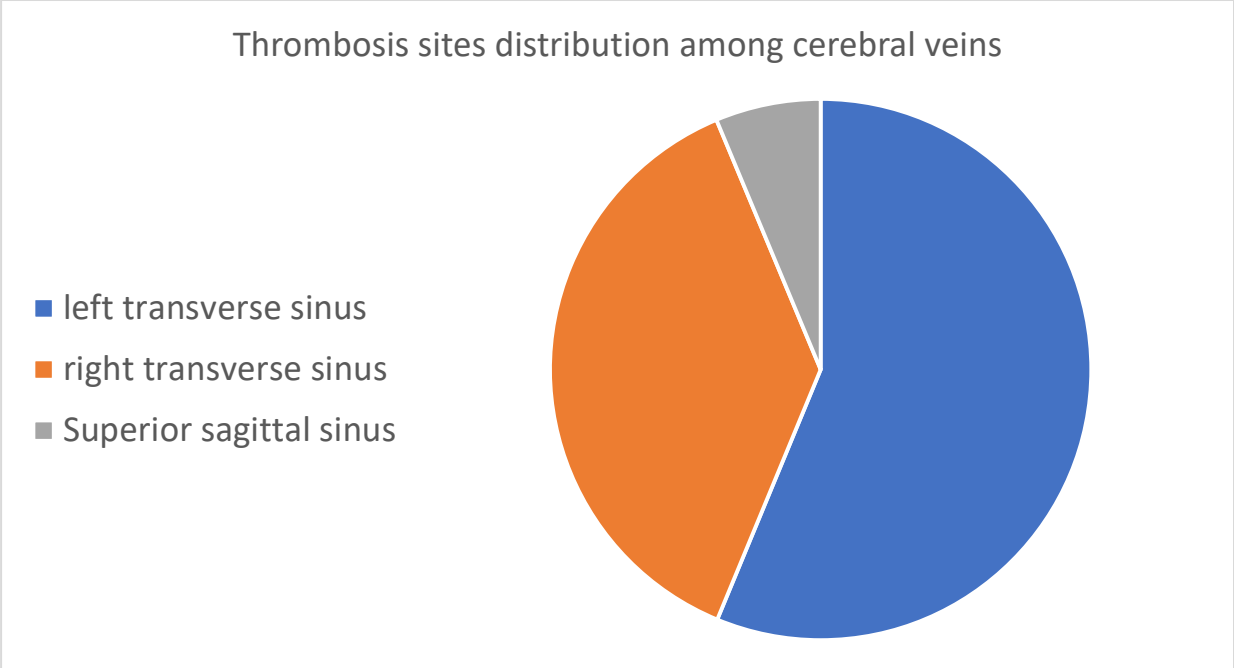

Figure 3.2: Thrombosis sites distribution among cerebral veins

Regrading secondary cerebral parenchymal changes due to venous thrombosis, they were encountered in 9 patients (56%) as follow: venous infarctions in (25%), venous hemorrhage in (6.3%) and hemorrhagic infarctions in (25%).
No cerebral parenchymal changes were found in (43.8%).

These changes were demonstrated in fig. 3

Table 3.3: Cerebral parenchymal changes among sinus thrombosis patients

Cerebral parenchymal changes	Frequency	Percent
only infarct	4	25.0
only hemorrhage	1	6.3
hemorrhagic infarct	4	25.0
No parenchymal changes	7	43.8
Total	16	100.0

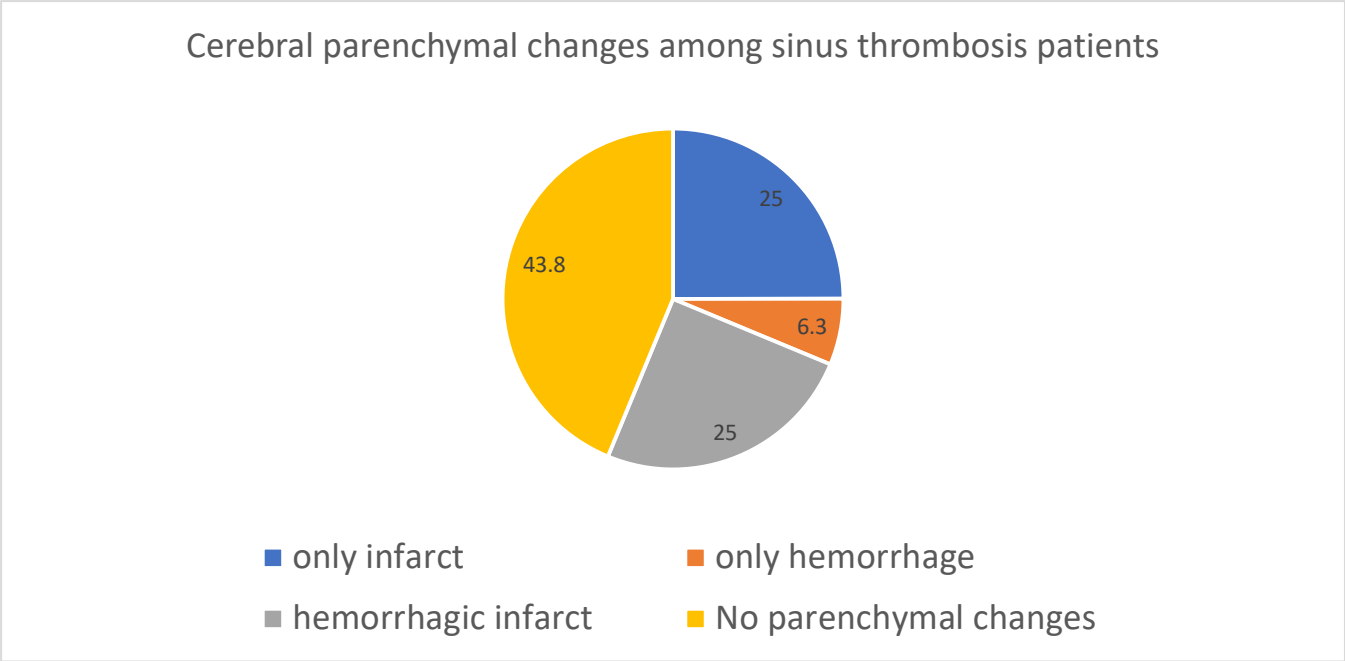

Figure 3.3: Cerebral parenchymal changes among CVT patients

Risk factors assessment in this study showed (68.8%) of patients who have risks for cerebral venous thrombosis, while (31.2%) have no identifiable risk factors.

Table 3.4: Risk factors assessment in cerebral sinus thrombosis patients

Risk factors assessment	NO.	Percent
Identifiable risk factors	11	68.8
No Identifiable risk factors	5	31.2

Figure 3.4: Risk factors assessment

Cases from this study:

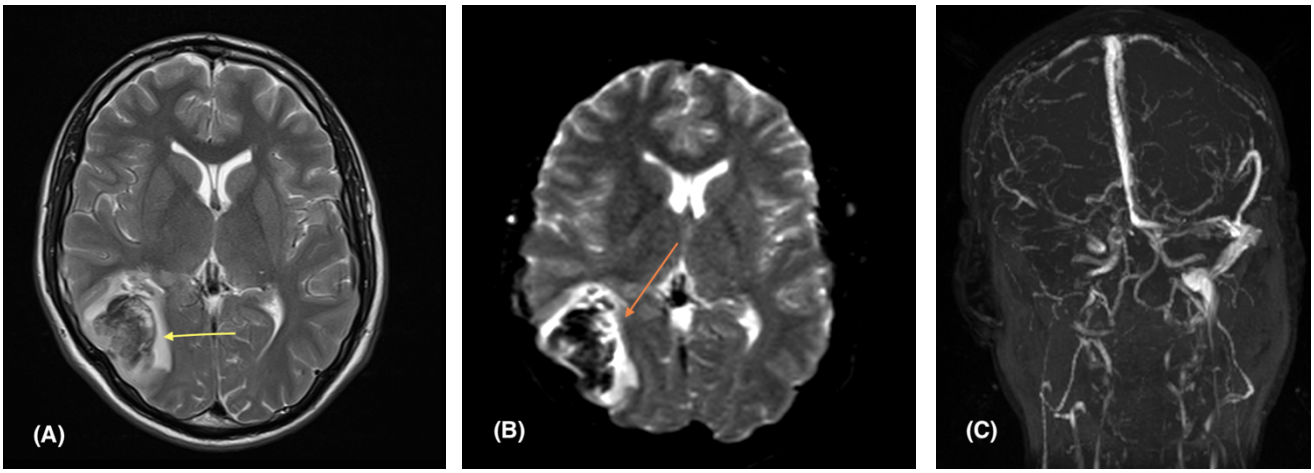

Figure 3.5: 26 years old male presented with headache. Axial T2 (a)axial DWI (b) and MR venography (c) shows right transverse sinus thrombosis with intra-cerebral hemorrhage (yellow arrow) within the right postero-temporal lobe.

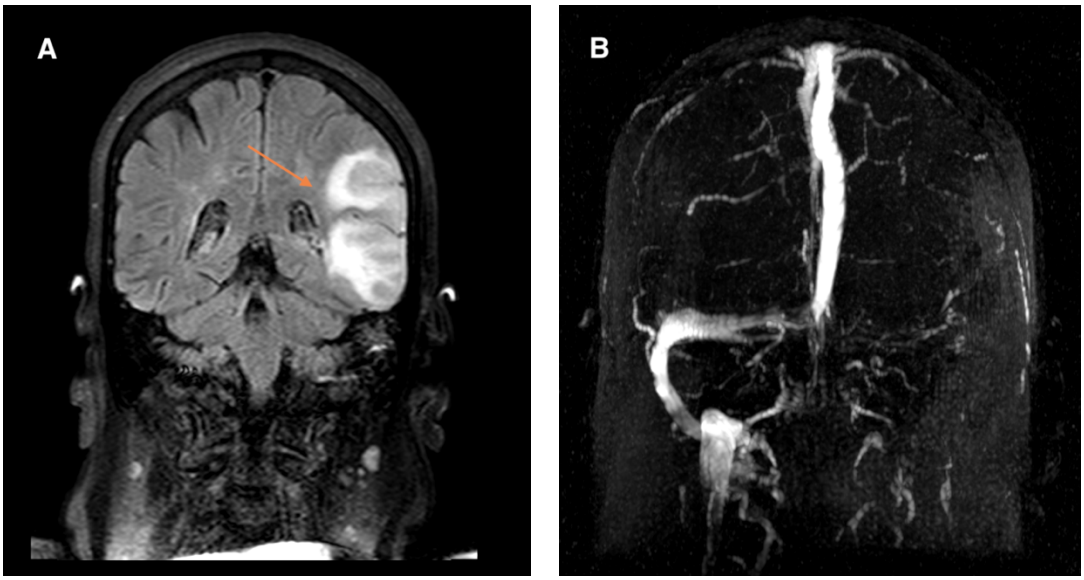

Figure 3.6: 56 years old female presented with weakness. Coronal FLAIR (a), and MR venogram (b) shows left transverse sinus thrombosis with venous infarction (yellow arrow) within the left temporal lobe.

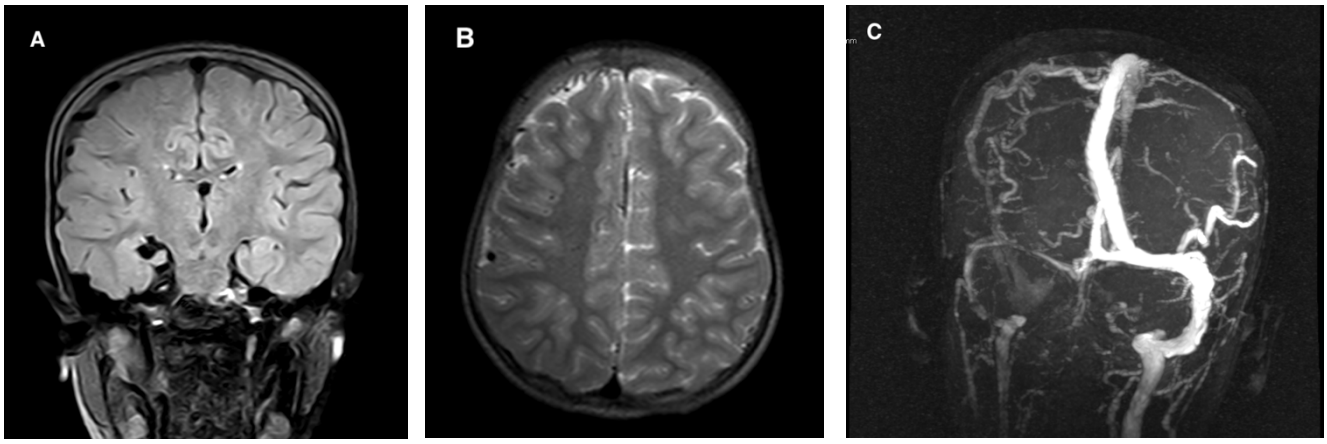

Figure 3.7: 7 years old female presented with headache. Coronal FLAIR (a), axial T2 (b) and MR venogram (c) was thought to be CVT but subsequently diagnosed as hypoplasia of the right transverse sinus.

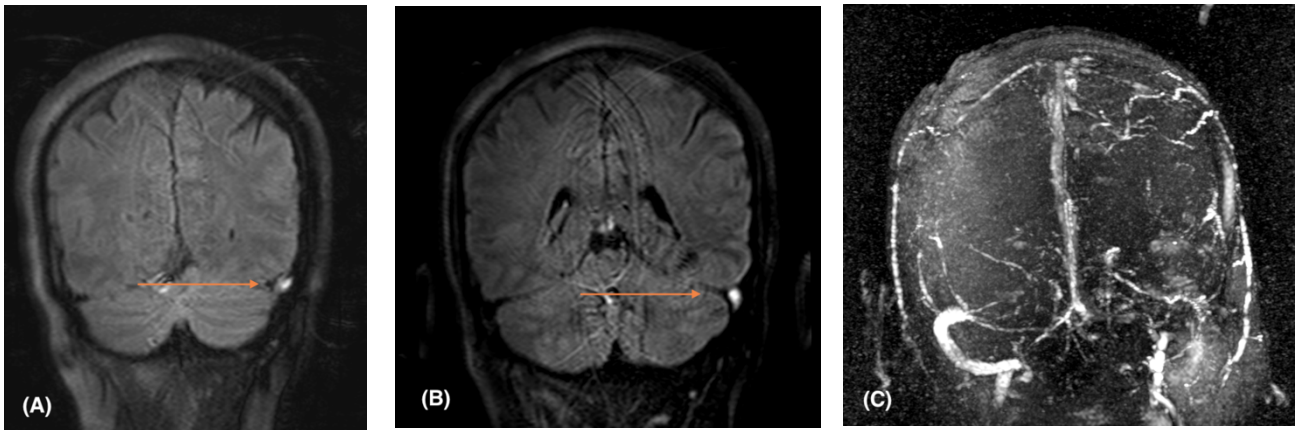

Figure 3.8: 48 years old female presented with weakness. Coronal T2 (a), Coronal FLAIR (b) and MR venogram (c) shows abnormal signal intensity (orange arrow) within left transverse sinus due to intraluminal clot with no apparent secondary cerebral parenchymal changes

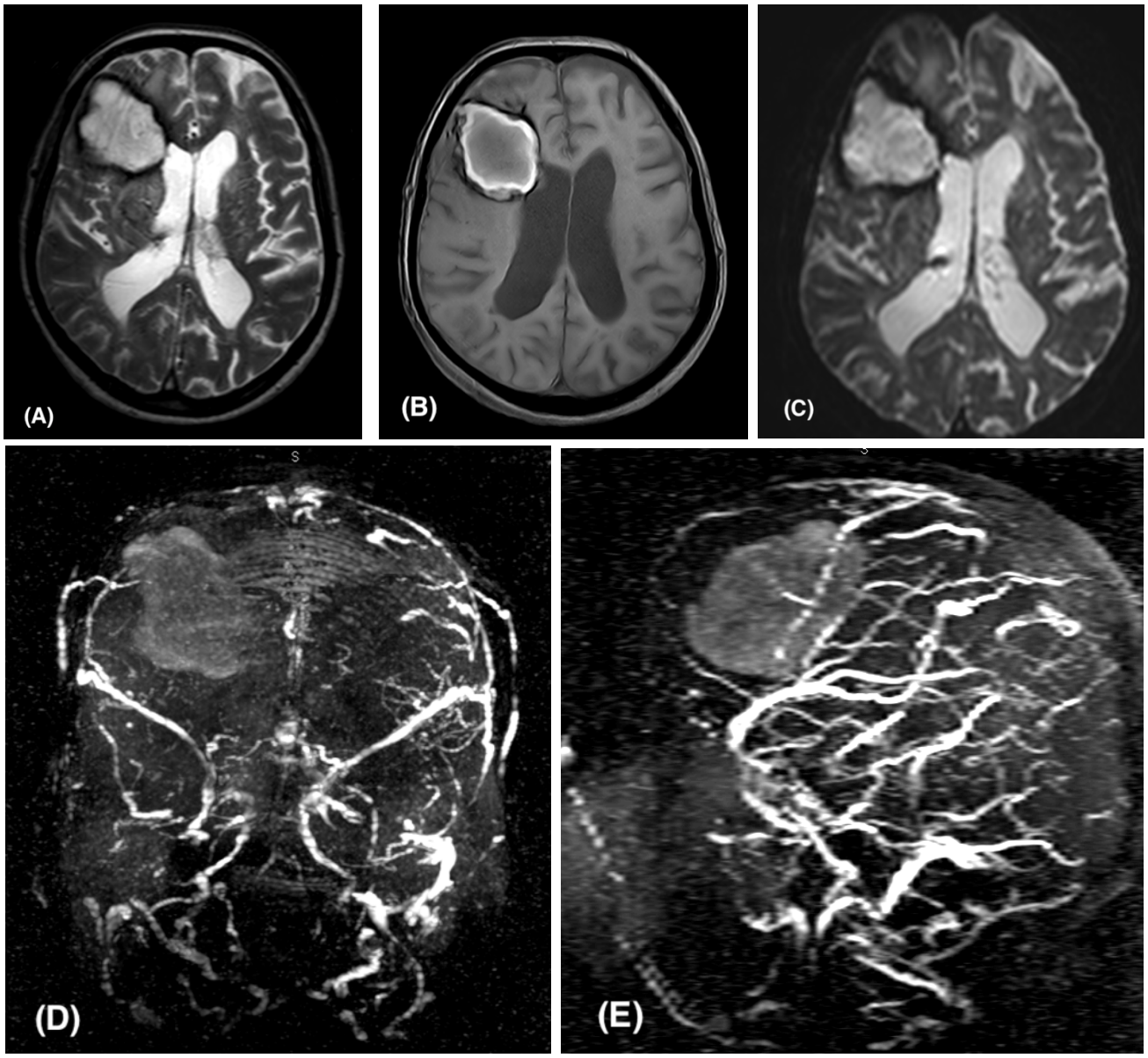

Figure 3.9: 52 years old female presented with weakness. Axial T2 (A), Axial T1 (B), DWI (C) and MR venogram (C) shows a hemorrhagic infarct within the frontoparietal territory, with midline shift, due to superior sagittal sinus thrombosis as shown in the MR venogram, with collaterals formation.

Chapter Four

Discussion

4.Discussion

Cerebral venous sinus thrombosis (CVST) is considered a rare disease refers to complete or partial occlusion of either the main sinus/sinuses or the feeding cortical veins leading to secondary effects of vascular congestion and focal or generalized neurological deficits. [49]

In this research, we found that Cerebral sinus thrombosis predominantly affects young adults, with mean age of 37 years in affected patients, and females are more commonly involved than males with male: female ratio of 2.2:1 (68%), this was in agreement with the results reported by Kalita et al. [47]

According to site distribution, we found that the superficial sinuses involvement was the most common type encountered. The transverse sinus was most commonly involved (93%) the left was even more commonly involved than the right, followed by the sagittal sinus (7%), these results were different from those reported by Raizer et al [48] in their study, which might be attributed case selection involving factors related to epidemiological causes.

MRI was performed on 16 patients. Abnormalities, excluding sinus thrombosis, included intraparenchymal hemorrhages (6%), infarction (25%), and hemorrhagic infarctions was seen in (25%). In addition, no parenchymal changes were noticed in (43%) of patients, which is different from that of Raizer et al [48] which might be due case selection involving cancer patients.

Regarding risk factors assessment in this study, 11 patients had risks of developing cerebral sinus thrombosis, while 5 patients had no identifiable risks, and these were in agreement with the results reported by Kalita et al. [47]

Chapter Five

Conclusion

5.Conclusion

1. Magnetic resonance imaging is the modality of choice to diagnose the cerebral sinus thrombosis and detect its complications.

2. Superficial sinuses involvement was the most common type encountered. The transverse sinus was most commonly involved.

3. Infarctions with hemorrhage was the most common abnormal secondary cerebral parenchymal changes found on MRI due to venous thrombosis.

Recommendation

Recommendation

1- Extending the study period to include more patients and expand the study population and the range of MRI findings that can be obtained.
2- Correlating CT findings of the patients with the MRI findings.

References

References

1. Bousser MG and Ferro JM: Cerebral venous thrombosis: an update. Lancet Neurol 6: 162-170, 2007.
2. Haacke EM, Reichenbach JR. Susceptibility weighted imaging in MRI: basic concepts and clinical applications. Hoboken, NJ: John Wiley & Sons; 2011.
3. Bousser MG, Russell RR. Cerebral venous thrombosis. In: Major Problems in Neurology, Warlow CP, Van Gijn J (Eds), WB Saunders, London 1997. p.27, 104.
4. Bousser MG, Chiras J, Bories J, Castaigne P. Cerebral venous thrombosis--a review of 38 cases. Stroke 1985; 16:199.
5. Ferro JM, Canhão P, Stam J, et al. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). Stroke 2004; 35:664.
6. Coutinho JM, Ferro JM, Canhão P, et al. Cerebral venous and sinus thrombosis in women. Stroke 2009; 40:2356.
7. Stam J. Thrombosis of the cerebral veins and sinuses. N Engl J Med 2005; 352:1791.
8. de Bruijn SF and Stam J: Randomized, placebo-controlled trial of anticoagulant treatment with low-molecular-weight heparin for cerebral sinus thrombosis. Stroke 30: 484-488, 1999.
9. lsensee C, Reul J and Thron A: Magnetic resonance imaging of thrombosed dural sinuses. Stroke 25: 29-34, 1994.
10. Dentali F, Crowther M and Ageno W: Thrombophilic abnormalities, oral contraceptives, and risk of cerebral vein thrombosis: a meta-analysis. Blood 107: 2766-2773, 2006.
11. Kenet G, Sadetzki S, Murad H, Martinowitz U, Rosenberg N, Gitel S, et al. Factor V Leiden and antiphospholipid antibodies are significant risk factors for ischemic stroke in children. Stroke 2000 Jun;31(6):1283–8.
12. Strater R, Vielhaber H, Kassenbohmer R, von Kries R, Gobel U, Nowak-Gottl U. Genetic risk factors of thrombophilia in ischaemic childhood stroke of cardiac origin. A prospective ESPED survey. Eur J Pediatr 1999 Dec;158(Suppl 3):S122–5.
13. Brown DC, Livingston JH, Minns RA, Eden OB. Protein C and S deficiency causing childhood stroke. Scott Med J 1993 Aug;38(4):114–15.
14. Coutinho JM. Cerebral venous thrombosis. J Thromb Haemost 2015; 13 Suppl 1:S238.
15. Saposnik G, Barinagarrementeria F, Brown RD Jr, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare

professionals from the American Heart Association/American Stroke Association. Stroke 2011; 42:1158.
16. Ferro JM, Correia M, Pontes C, et al. Cerebral vein and dural sinus thrombosis in Portugal: 1980-1998. Cerebrovasc Dis 2001; 11:177.
17. Coutinho JM, Zuurbier SM, Aramideh M, Stam J. The incidence of cerebral venous thrombosis: a cross-sectional study. Stroke 2012; 43:3375.
18. Gotoh M, Ohmoto T, Kuyama H. Experimental study of venous circulatory disturbance by dural sinus occlusion. Acta Neurochir (Wien) 1993; 124:120.
19. deVeber G, Andrew M, Adams C, et al. Cerebral sinovenous thrombosis in children. N Engl J Med 2001; 345:417.
20. Ferro JM, Canhão P, Bousser MG, et al. Cerebral vein and dural sinus thrombosis in elderly patients. Stroke 2005; 36:1927.
21. Zuurbier SM, Hiltunen S, Lindgren E, et al. Cerebral Venous Thrombosis in Older Patients. Stroke 2018; 49:197.
22. 24.Lafitte F, Boukobza M, Guichard JP, Hoeffel C, Reizine D, Ille O, et al. MRI and MRA for diagnosis and follow-up of Cerebral Venous Thrombosis (CVT). Clin Radiol 1997; 52(9): 672-9.
23. Rodallec MH, Krainik A, Feydy A, Helias A, Colombani JM, Julles MC, et al. Cerebral venous thrombosis and multidetector CT angiography: tips and tricks. Radiographics 2006; 26(Suppl 1): S5-18.
24. Nabil Sheri M, Kashif A. Chronic deep venous thrombosis of the petrosal sinus causing multiple cranial nerve palsy. Iran J Neurol 2012; 11(2): 74-6.
25. Ferro JM, Canhão P, Stam J, et al. Delay in the diagnosis of cerebral vein and dural sinus thrombosis: influence on outcome. Stroke 2009; 40:3133.
26. Ferro JM, Falcao F, Melo TP, Campos JG. Dural sinus thrombosis mimicking "capsular warning syndrome". J Neurol 2000; 247:802.
27. Lancon JA, Killough KR, Tibbs RE, et al. Spontaneous dural sinus thrombosis in children. Pediatr Neurosurg 1999; 30:23.
28. Wasay M, Bakshi R, Bobustuc G, et al. Cerebral venous thrombosis: analysis of a multicenter cohort from the United States. J Stroke Cerebrovasc Dis 2008; 17:49.
29. Girot M, Ferro JM, Canhão P, et al. Predictors of outcome in patients with cerebral venous thrombosis and intracerebral hemorrhage. Stroke 2007; 38:337.
30. Ohene-Frempong K, Weiner SJ, Sleeper LA, Miller ST, Embury S, Moohr JW, et al. Cerebrovascular accidents in sickle cell disease: rates and risk factors. Blood 1998 Jan 1;91(1):288–94.
31. Cakmak S, Hermier M, Montavont A, et al. T2*-weighted MRI in cortical venous thrombosis. Neurology 2004; 63:1698.

32. Selim M, Fink J, Linfante I, et al. Diagnosis of cerebral venous thrombosis with echo-planar T2*-weighted magnetic resonance imaging. Arch Neurol 2002; 59:1021.
33. Fellner FA, Fellner C, Aichner FT, Mölzer G. Importance of T2*-weighted gradient-echo MRI for diagnosis of cortical vein thrombosis. Eur J Radiol 2005; 56:235.
34. Wetzel SG, Kirsch E, Stock KW, et al. Cerebral veins: comparative study of CT venography with intraarterial digital subtraction angiography. AJNR Am J Neuroradiol 1999; 20:249.
35. Linn J, Ertl-Wagner B, Seelos KC, et al. Diagnostic value of multidetector-row CT angiography in the evaluation of thrombosis of the cerebral venous sinuses. AJNR Am J Neuroradiol 2007; 28:946.
36. Chu K, Kang DW, Yoon BW, Roh JK. Diffusion-weighted magnetic resonance in cerebral venous thrombosis. Arch Neurol 2001; 58:1569.
37. Dormont D, Anxionnat R, Evrard S, et al. MRI in cerebral venous thrombosis. J Neuroradiol 1994; 21:81.
38. Isensee C, Reul J, Thron A. Magnetic resonance imaging of thrombosed dural sinuses. Stroke 1994; 25:29.
39. Favrole P, Guichard JP, Crassard I, et al. Diffusion-weighted imaging of intravascular clots in cerebral venous thrombosis. Stroke 2004; 35:99.
40. Ozsvath RR, Casey SO, Lustrin ES, et al. Cerebral venography: comparison of CT and MR projection venography. AJR Am J Roentgenol 1997; 169:1699.
41. Khandelwal N, Agarwal A, Kochhar R, et al. Comparison of CT venography with MR venography in cerebral sinovenous thrombosis. AJR Am J Roentgenol 2006; 187:1637.
42. Rodallec MH, Krainik A, Feydy A, et al. Cerebral venous thrombosis and multidetector CT angiography: tips and tricks. Radiographics 2006; 26 Suppl 1:S5.
43. Celebisoy N, Gokcay F, Sirin H, Akyurekli O. Treatment of idiopathic intracranial hypertension: topiramate vs acetazolamide, an open-label study. Acta Neurol Scand 2007; 116:322–327
44. Rohr A, Dorner L, Stingele R, Buhl R, Alfke K, Jansen O. Reversibility of venous sinus obstruction in idiopathic intracranial hypertension. AJNR 2007; 28:656–659
45. Stam J, De Bruijn SF, DeVeber G. Anticoagulation for cerebral sinus thrombosis. Cochrane Database Syst Rev 2002; 4:CD002005
46. Anvekar D. Dr Balaji Anvekar's Neuroradiology Cases [Internet]. Neuroradiologycases.com. 2019 [cited 17 April 2019]. Available from: http://www.neuroradiologycases.com/search/label/Hypoplastic%20vs%20thrombosed%20sinus%20on%20MRI

47. Kalita J, Chandra S, Kumar B, Bansal V, Misra U. Cerebral Venous Sinus Thrombosis from a Tertiary Care Teaching Hospital in India. The Neurologist. 2016;21(3):35-38.
48. Rodas R, Raizer J, DeAngelis L. Cerebral sinus thrombosis diagnosed by MRI and MR venography in cancer patients. Neurology. 2000;55(6):903-903.
49. Khanal P, Thapa L, Shrestha A, Bhattarai S, Sapkota D, Sharma N et al. Cerebral Venous Sinus Thrombosis during Everest Expedition: A Case Report and Review of the Literature. Case Reports in Neurological Medicine. 2016; 2016:1-3.
50. Moharir, M., & Kenet, G. (2015). Cerebral sinovenous thrombosis in children and neonates. In N. Goldenberg & M. Manco-Johnson (Eds.), *Pediatric Thrombotic Disorders* (pp. 33-46). Cambridge: Cambridge University Press. doi:10.1017/CBO9781139028882.00
51. Souza D. Dural venous sinus thrombosis | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia.org. 2019 [cited 19 April 2019]. Available from: https://radiopaedia.org/articles/dural-venous-sinus-thrombosis?lang=us
52. Dixon A. Cord sign (dural sinus thrombosis) | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia.org. 2019 [cited 19 April 2019]. Available from: https://radiopaedia.org/articles/cord-sign-dural-sinus-thrombosis?lang=gb