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Case Report

Multimodality Imaging of Cerebral Venous Sinus Thrombosis - A Case Report and Short Review of Literature

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Abstract

This is a Case Report of Vein of Trolard and superior sagittal sinus thrombosis with hemorrhagic stroke, a known but rare entity. Dural sinus thrombosis [DST] related hemorrhagic strokes are well known though they are rarer when compared to arterial brain infarctions. When compared to thrombosis of dural sinuses like superior sagittal sinus, thrombosis of the other smaller cerebral veins cause an even rarer form of stroke, with an incidence of 5 per million people each year. The interpreting radiologist has to be aware of multiple findings of cerebral venous sinus thrombosis [CVST], which are described in this current Case Report using various modalities along with literature review. The current case report also reminds one to look at that which is not seen, e.g., the non-enhancing right cortical vein of Trolard in the CT Venogram of this patient, which aids in further confirming the diagnosis. The cortical veins can vary in their location and can be asymmetric when compared to the other side. However, in a setting of non-arterial distribution hemorrhages with unclear etiology, focused evaluation of nonenhanced and enhanced venous structures can be very helpful in timely diagnosis leading to prompt patient care and good outcome.

Keywords: *Superior Sagittal Sinus Thrombosis; Dural Sinus Thrombosis [DST]; Cerebral Venous Sinus Thrombosis [CVST]; Thrombosis of Cortical Vein of Trolard; Hemorrhagic Venous Stroke; Empty Delta Sign; Dense Cord sign*

Abbreviations

DST: Dural Sinus Thrombosis; CVST: Cerebral Venous Sinus Thrombosis; CT: Computerized Tomography; MRI: Magnetic Resonance Imaging; HU: Hounsfield Unit; CTV: CT Venography; MRV: MR Venography; DSA: Digital Subtraction Angiography.

Introduction

Dural sinus thrombosis (DST) is an atypical cause of stroke [less than 1%] that can affect patients of any age and gender but is more commonly associated with young females. The peak age is younger than patients with arterial strokes. Cerebral venous sinus thrombosis

[CVST] incidence is 5 per million people each year. It has to be considered in the differential diagnosis of non-arterial distribution infarctions without clear etiologies.

Case Report

47-year-old female presented with a sudden onset of left hemiparesis. The patient presented to the emergency department with complaints of acute-onset of left-sided weakness, decreased sensation, and slurred speech. She also reported progressively worsening daily headaches over the past week. There was no history of trauma. Prior medical history is significant for diabetes mellitus, hypertension, and hyperlipidemia. Social history is significant for smoking 1.5 PPD for >20 years. Physical exam revealed left upper extremity weakness and numbness. Systolic blood pressure was 152. GCS = 15, NIH stroke scale=1, and ICH score = 0. Other Diagnostic testing revealed LDL 226, HbA1C 8.5%, B12 deficiency and blood glucose 202 mg/dL. Patient was worked up for hypercoagulable causes, but none were identified. Patient was anticoagulated with heparin and once CT was stable, patient was started on apixaban. She progressively improved without incident.

Methods

CT head without contrast was performed in a Somatom Definition Edge Siemens scanner [Siemens Medical Systems, USA]. Standard 1mm axial CT soft tissue algorithm images were obtained followed by reconstructed sagittal and coronal images along with axial bone window images.

MRI of the brain was performed on a 3T Siemens Spectra scanner [Siemens Medical Systems, Malvern, Pennsylvania, USA] using a standard head coil. Standard 4 mm thick axial T1, axial T2, fat suppressed axial T2 FLAIR, axial DWI and ADC brain pulse sequences were acquired without contrast. After administration of 20 ml of Multihance axial, sagittal and coronal post contrast sequences were obtained.

CT venogram of the head was performed in a Somatom Definition Edge Siemens scanner [Siemens Medical Systems, USA] following administration of 100ml of Isovue 370, in the axial plane with 35 sec delay. The axial, sagittal and coronal images were reconstructed with overlapping slices.

Imaging findings

Nonenhanced CT [NECT] of the brain (Figure 1) shows right superior frontoparietal parenchymal hyperdensity with peripheral hypodensity suggestive of hemorrhage with surrounding vasogenic edema. 'Dense vein sign' of right vein of Trolard and dense superior sagittal sinus are suggestive of thrombosis.

MRI non-enhanced images of the brain (Figure 2 and 3) show an isointense T1 lesion in the right superior frontoparietal region with corresponding hyperintense T2 FLAIR signal, which correlate with the parenchymal hemorrhage on CT. There is slight vasogenic edema but no midline shift. Axial GRE images show a hypointense right superior frontoparietal 'blooming' hemorrhagic lesion with surrounding edema and hypointense thrombosed 'blooming' vein of Trolard extending from the right frontoparietal parenchymal lesion to the hypointense 'blooming' thrombosed superior sagittal sinus. Axial, coronal and sagittal T1-weighted MRI images through the brain after IV Multihance administration (Figure 4) show heterogenous gyriform enhancement surrounding the area of hemorrhage, a triangular filling defect [empty delta sign] within the superior sagittal sinus, normal enhancing left vein of Trolard and a long segment filling defect within the right vein of Trolard extending to the thrombosed superior sagittal sinus.

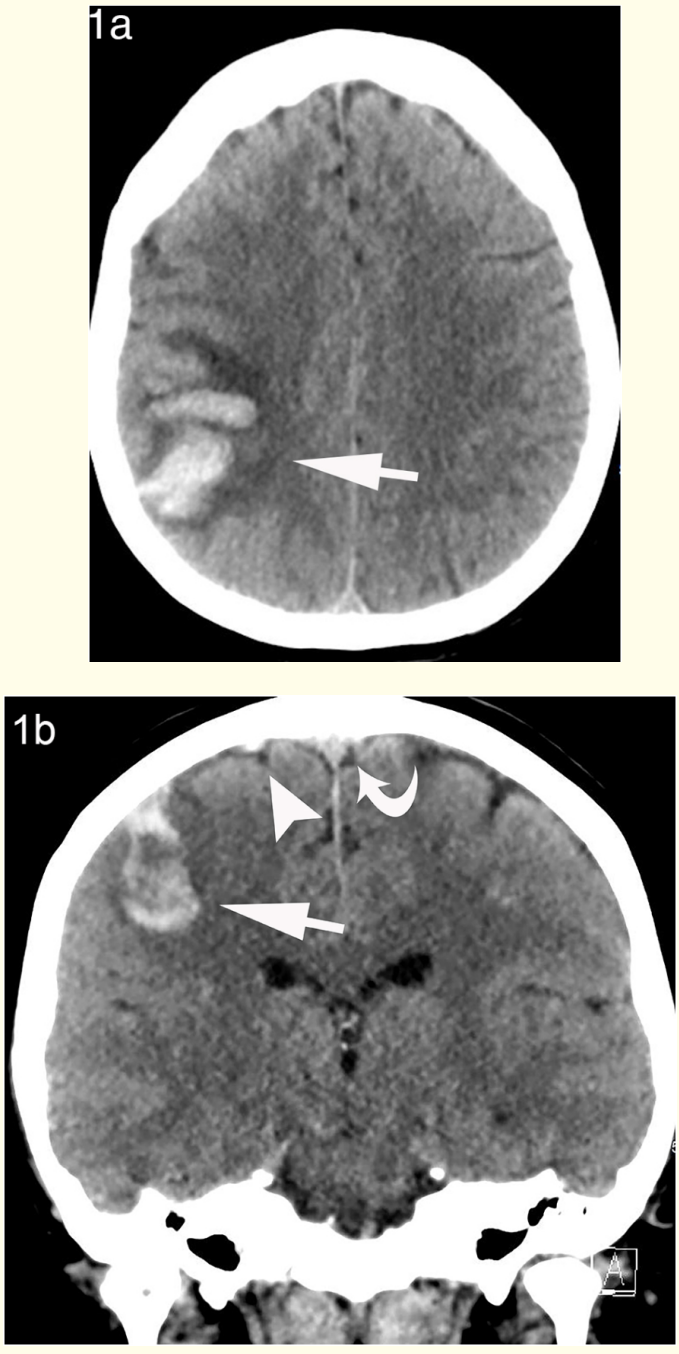


Figure 1: *Non-enhanced CT Head [a-b]. Axial (a) and coronal (b) non contrast CT of the brain show right superior frontoparietal parenchymal hemorrhagic hyperdensity (arrow in a, b) with peripheral edematous hypodensity, 'dense vein sign' of thrombosed right vein of Trolard (arrowhead in b) and dense thrombosed superior sagittal sinus (curved arrow in b).*

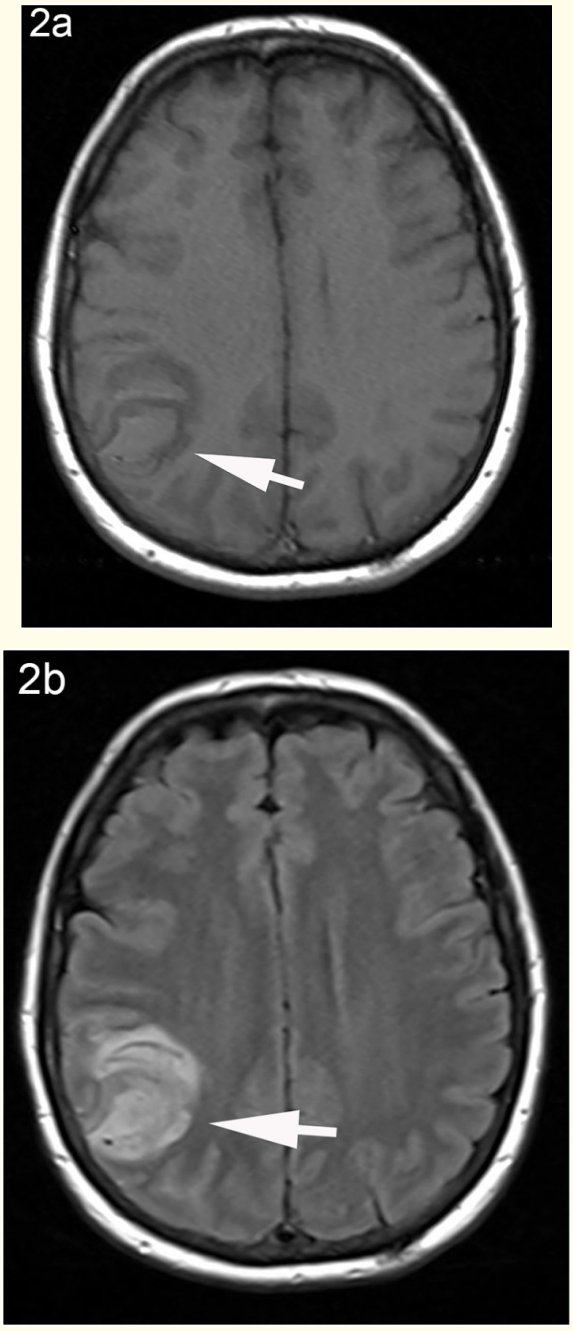


Figure 2: *3T Siemens Spectra scanner MRI Brain images without contrast [a, b]. Axial T1W (a), axial T2W FLAIR (b) MRI images without IV gadolinium through the brain show isointense T1 lesion in the right superior frontoparietal lobe (arrow in a) and hyperintense T2 FLAIR signal in the right superior frontoparietal lobe (arrow in b).*

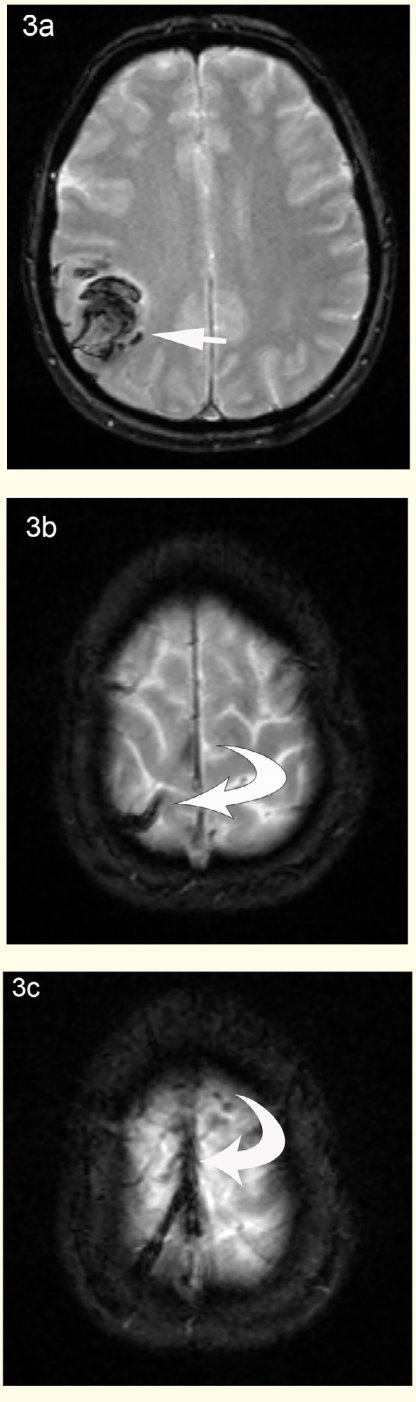
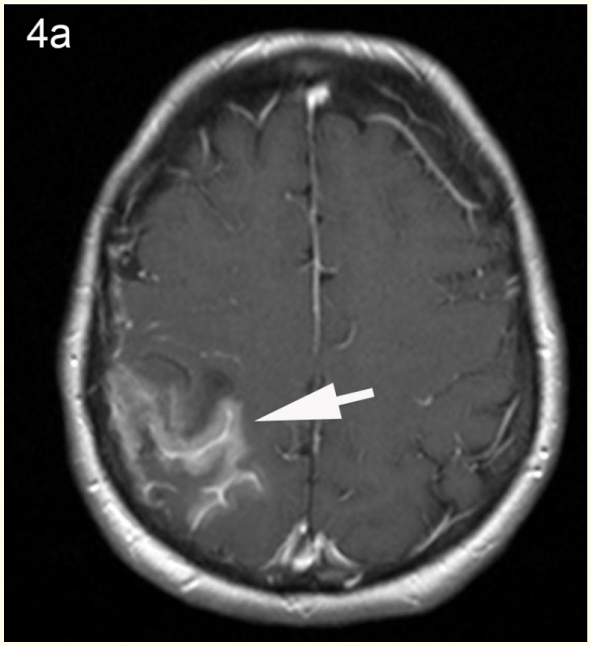


Figure 3: *3T Siemens Spectra scanner MRI Brain GRE images without contrast [a-c]. Axial GRE MRI images (a-c) without IV gadolinium through the brain show hypointense right superior frontoparietal lobe blooming hemorrhagic lesion (arrow in a) with minimal surrounding edema, hypointense blooming thrombosed vein of Trolard (curved arrow in b) extending from the right frontoparietal parenchymal lesion to the hypointense blooming thrombosed superior sagittal sinus (curved arrow in c).*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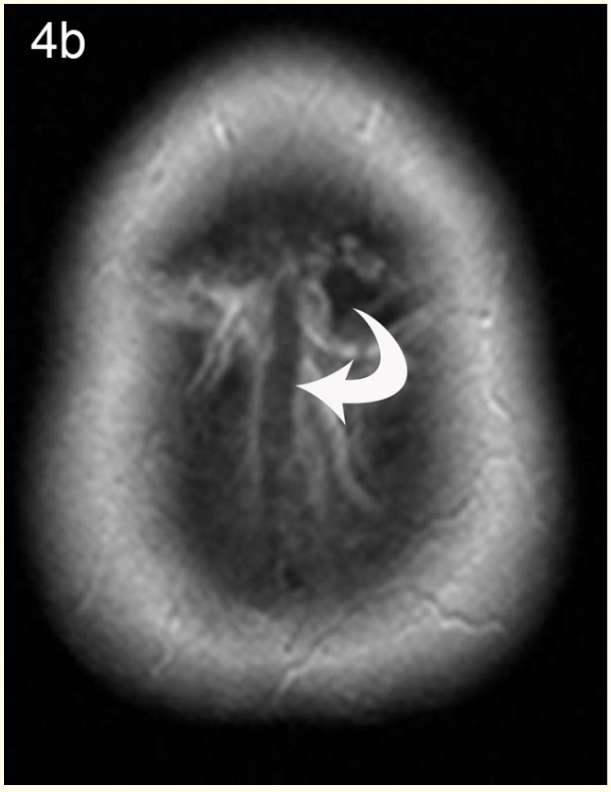
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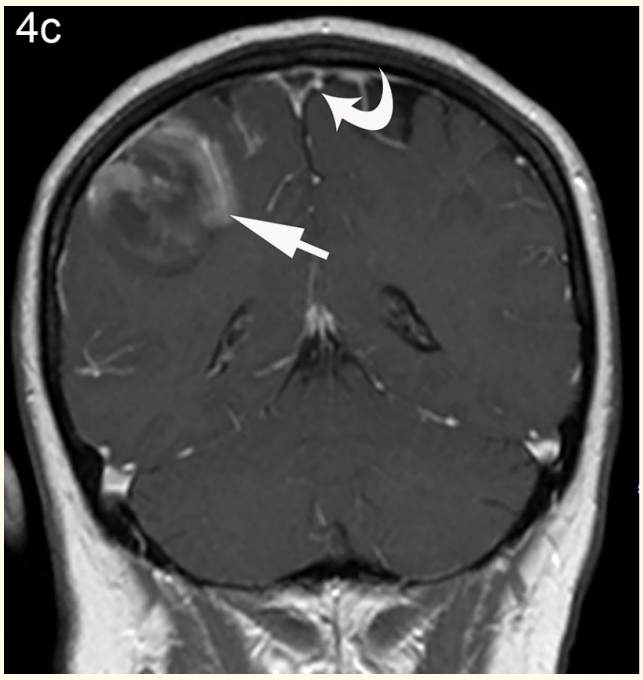


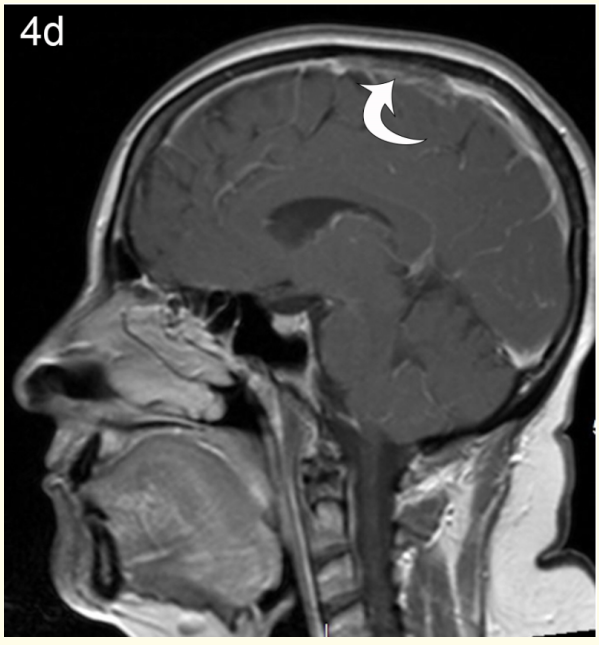
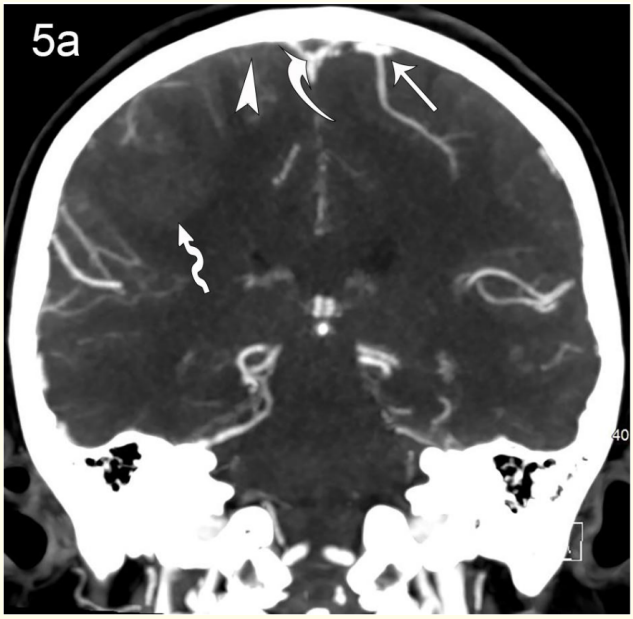
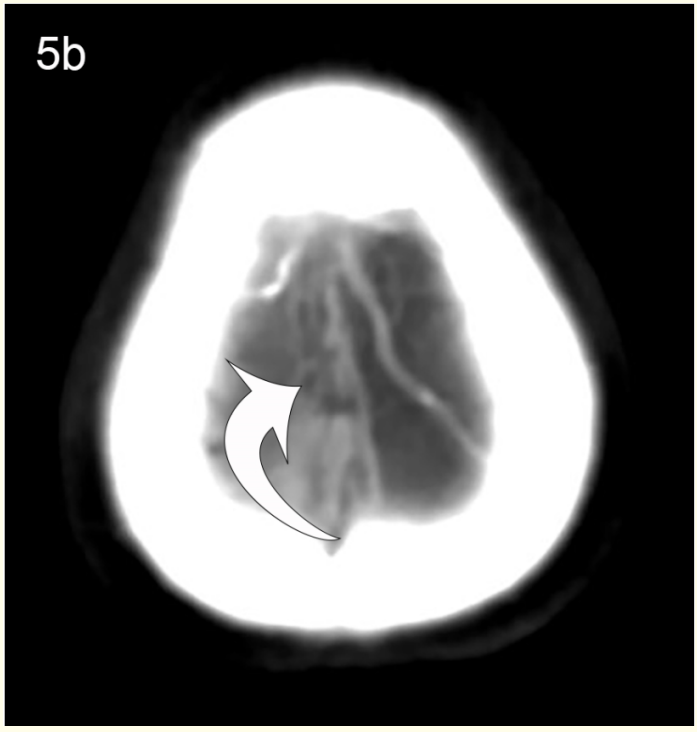


Figure 4: *3T Siemens Spectra scanner MRI Brain post contrast images [a-d]. Axial (a-b), coronal (c) and sagittal (d) T1-weighted MRI images after IV Multihance administration show heterogenous gyriform enhancement of the lesion (arrow in a,c). A triangular filling defect [empty delta sign] within the superior sagittal sinus (curved arrow in c)) and ropey filling defect in superior sagittal sinus (curved arrow in b, d) are consistent with thrombosis.*

CT venogram reconstructed MIP images of the brain obtained post intravenous contrast (Figure 5) show a long filling defect in the superior sagittal sinus, normal enhancing left vein of Trolard, lack of a normal symmetrical enhancing right vein of Trolard and subtle hyperdense right frontoparietal parenchymal hemorrhagic venous infarct.





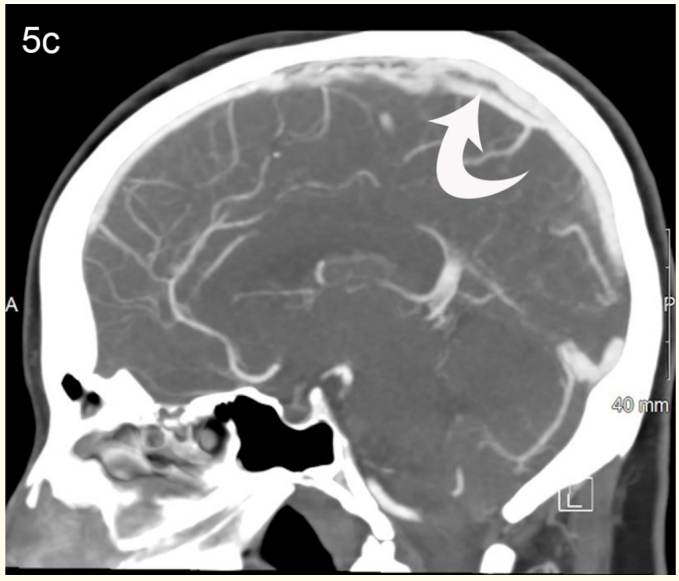


Figure 5: *CT venogram [a-c]. Coronal (a), axial (b) and sagittal (c) post IV contrast reconstruction MIP images of CTV show filling defect in the superior sagittal sinus (curved arrow in a-c), enhancing left cortical vein of Trolard (straight arrow in a), region of non-enhancing right cortical vein of Trolard (arrowhead in a) and subtle hyperdense right frontoparietal parenchymal lesion (squiggly arrow in a).*

Discussion

Cerebral venous sinus thrombosis (CVST) is a rare form of stroke. It can affect patients of any age and gender but is more commonly associated with young females. The risk factors include acquired hypercoagulable states (pregnancy, early postpartum state, antiphospholipid syndrome), previous thrombotic events, prothrombotic drugs (oral contraceptive pills, hormone replacement therapy), genetic coagulopathies (e.g. protein C and S deficiency, antithrombin III deficiency, and factor V Leiden mutation), malignancy, collagen vascular diseases (e.g. Lupus, Wegener granulomatosis, and Behcet syndrome) and inflammatory bowel disease (e.g. Crohn disease and ulcerative colitis). Other entities like obesity, anemias, nearby infection (e.g. mastoiditis), intracranial hypotension, etc. can also contribute to it [1-3].

Pathophysiology

The thrombus in the cortical veins and dural sinuses blocks venous drainage and thereby increases venous pressure with subsequent decrease in perfusion and causes infarction. In the re-perfusion phase, the recruited immature friable capillaries cause hemorrhage in the involved area. Approximately 50% of patients will develop a venous infarct [4,5]. Cerebral cortical vein thrombosis are commonly found in conjunction with dural sinus thrombosis, although they can occur in isolation [6].

Clinical presentation

Clinical manifestations vary depending on the location of thrombus. Signs and symptoms include headache, blurred vision, hemiparesis, seizures and coma. Papilledema and altered consciousness may also be present [1]. The diagnosis is often made late due to the high variability of clinical presentation and relatively rare incidence [7].

Venous thrombosis related infarcts should be considered in the setting of atypical hemorrhagic infarcts which cross arterial territories, or spares cortex. Venous hemorrhagic infarcts are typically seen in parasagittal brain structures from sagittal sinus thrombosis and appear in temporoparietal regions following transverse/sigmoid sinus thrombosis. The vein of Trolard is a large cortical vein which drains the parietal lobe into the superior sagittal sinus. The vein of Labbe is a large cortical vein which drains the lateral temporal lobe towards the transverse or sigmoid sinus. Thrombosis of these large cortical veins causes infarctions in their corresponding draining areas. 50% of venous thrombosis show infarct with cortical and subcortical hemorrhages, which typically extend from central to periphery of the affected area.

Imaging findings

Non-enhanced CT

The venous infarct presents as an edematous hypodense lesion in that portion of the brain that it drains. Associated hemorrhage related hyperdensity is seen during the reperfusion phase of the venous infarct. These findings are similar to arterial strokes, only with change in locations. The thrombosed vein itself may not be seen in many cases. One notable feature of CVST is the "cord" sign, named due to the shape of a tubular venous structure caused by hyperdense thrombosed cortical vein. There is literature on Hounsfield unit (HU) related to the hyperdensity of thrombus. Hyperdense dural sinus with a HU greater than 70 is associated with a high degree of specificity in diagnosing DST [1,4,8]. A thrombosed vein can be differentiated from non-thrombosed hemoconcentrated blood containing hyperdense vein by the presence of high hematocrit. The Hounsfield unit (HU): hematocrit (HCT) ratio of 2 or more is strongly associated with thrombus. In the proper clinical context HU:HCT ratio of 1.8 - 1.9 may require further evaluation [8].

Contrast enhanced CT

The venous infarct is seen with or without hemorrhage as a hypodense lesion with varying degrees of hyperdensity related to the hemorrhage. Subacute to chronic infarct can enhance. Thrombosis in DST and cortical vein demonstrate filling defects within the enhancing dural sinus or vein. The "empty delta" sign is seen in thrombosis of Superior sagittal sinus. It results from enhancing contrast surrounding a non-enhancing clot-within the lumen [9,4,10]. However, lack of this sign should not be used to rule out the diagnosis as it is present in only 30% of cases. Due to clot organization and vascularization, the thrombus can sometimes enhance and a filling defect may not be seen within a dural sinus or cerebral vein in a post-contrast study. CT (especially without contrast) can be normal over 25% of the time [11,12].

MRI

Similar to arterial infarct, venous infarct is seen with or without hemorrhage with similar signal characteristics in a distribution different than expected for arterial distribution. Acute stroke is edematous and it will demonstrate hypointense signal on T1 and hyperintense signal on T2 spin echo sequences with restricted diffusion [1]. Hemorrhage superimposed on infarct shows varying signal characteristics based on the blood products related to its evolution. DSTs can show various imaging features based on duration of the thrombus (acute vs. subacute vs. chronic). T1 weighted imaging shows an isointense thrombus in acute and chronic settings, but the thrombus is hyperintense if subacute. T2 weighted imaging reveals hyperintense thrombus if subacute or chronic, and hypointense thrombus if acute [13]. A significant finding on T2*GRE is "blooming" hypointense thrombus. Cortical vein thrombosis on T2*GRE is hypointense and cord-like. The empty delta sign of superior sagittal sinus thrombosis can be seen on T1 weighted post contrast images due to filling defects caused by clots. However, it has to be noted that chronic clots can themselves enhance [4].

Angiography

CT venography (CTV), MR Venography (MRV), and Digital Subtraction Angiography (DSA) with intravenous contrast demonstrate thrombus as a filling defect within the dural sinuses and cortical veins in these contrast enhanced studies.

Prognosis and treatment

Prognosis is typically promising. 81% of women and 71% of men make a full recovery. The most frequently noted complications are seizures and new thrombotic events. The recurrence rate at one year is 2.2% [10]. Poorer outcomes were associated with male gender, age over 37, ICH upon admission, deep venous thrombosis, and CNS infection [5,14]. Early anticoagulation is the treatment of choice in venous thrombosis despite the hemorrhage. It's unlike the arterial infarcts where anticoagulation is withheld in case of associated hemorrhage. Treatment consists of low molecular weight or unfractionated heparin in acute setting, followed by usage of long-term anticoagulants such as warfarin [1,5]. Neuro-interventional treatment may be considered for patients who are refractory to treatment or where anticoagulants are contraindicated [1,15].

Conclusion

Cerebral venous sinus thrombosis [CVST] is an unusual cause of stroke, particularly in younger patients. Because the outcome is generally fairly good, and the treatment is very different from hemorrhagic arterial stroke or uncomplicated parenchymal hemorrhage, it is important to make the distinction. Usually with intracranial hemorrhage related to causes such as arterial infarcts, hypertension, amyloid angiopathy, neoplasm or vascular malformation anticoagulation is withheld, but patients with venous hemorrhagic infarcts are anticoagulated. The interpreting physician must be alert to the presence of hyperdensity in cerebral venous structures on non-enhanced CT, parenchymal hemorrhages which do not correspond to arterial territories, empty delta sign in superior sagittal sinus and filling defects in cerebral venous structures in contrast enhanced studies. These findings help differentiate parenchymal hemorrhage related to CVST from a multitude of other potential etiologies. Though the cortical veins can be normally asymmetrical when compared to the other side and can differ in location, the current case report also reminds one to look for these enhancing normal structures as sometimes non-visualization of it along with pertinent other findings will lead to the diagnosis. In this study, the asymmetrically non-enhancing right cortical vein of Trolard with non-contrast hyperdense appearance of that vein, superior sagittal sinus filling defects and the hemorrhagic infarct in the drainage area, helped with the diagnosis when evaluated in conjunction with the MRI findings. Awareness and prompt identification of this entity will help in timely management of these patients leading to good patient care and outcome.

Conflict of Interest

None.

Bibliography

1. Osborn AG., *et al.* "Diagnostic Imaging Brain". 3rd edition. Philadelphia, PA: Elsevier (2016): 376-379.

2. Provenzale JM., *et al.* "Dural Sinus Thrombosis: Findings on CT and MR Imaging and Diagnostic Pitfalls". *American Journal of Roentgenology* 170 (1998): 777-783.

3. Provenzale JM and PG Kranz. "Dural Sinus Thrombosis: Sources of Error in Image Interpretation". *American Journal of Roentgenology* 196 (2011): 23-31.

4. Black DF., *et al*. "Cerebral Venous Sinus Density on Noncontrast CT Correlates with Hematocrit". *American Journal of Neuroradiology* 32.7 (2011): 1354-1357.

5. Patchana T., *et al*. "Superior Sagittal Sinus: A Review of the History, Surgical Considerations, and Pathology". *Cureus* 11.5 (2019).

6. Linn J and H Brückmann. "Cerebral Venous and Dural Sinus Thrombosis". *Clinical Neuroradiology* 20 (2010): 25-37.

7. Vogl TJ., *et al*. "Dural Sinus Thrombosis: Value of Venous MR Angiography for Diagnosis and Follow-Up". *American Journal of Roentgenology* 162 (1994): 1191-1198.

8. Bonneville F. "Imaging of Cerebral Venous Thrombosis". *Diagnostic and Interventional Imaging* 95.12 (2014): 1145-1150.

9. Dlamini N., *et al.* "Cerebral Venous Sinus (Sinovenous) Thrombosis in Children". *Neurosurgery Clinics of North America* 21.3 (2010): 511-527.

10. Al-Sulaiman A. "Clinical Aspects, Diagnosis and Management of Cerebral Vein and Dural Sinus Thrombosis: A Literature Review". *Saudi Journal of Medicine and Medical Sciences* 7.3 (2019): 137-145.

11. Wasay M and M Azeemuddin. "Neuroimaging of Cerebral Venous Thrombosis". *Journal of Neuroimaging* 15 (2006): 118-128.

12. Sadigh G., *et al.* "Diagnostic Performance of MRI Sequences for Evaluation of Dural Venous Sinus Thrombosis". *American Journal of Roentgenology* 206 (2016): 1298-306.

13. Dormont D., *et al*. "Gadolinium-Enhanced MR of Chronic Dural Sinus Thrombosis". *American Journal of Neuroradiology* (1995): 1347-1352.

14. Linn J., *et al*. "Noncontrast CT in Deep Cerebral Venous Thrombosis and Sinus Thrombosis: Comparison of Its Diagnostic Value for Both Entities". *American Journal of Neuroradiology* 30 (2009): 728-735.

15. Soleau SW., *et al*. "Extensive Experience with Dural Sinus Thrombosis". *Neurosurgery* 52.3 (2003): 534-544.

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