Journal of Neurological Disorders

J Neurol Disord
Volume 11:5, 2023

Case Report Open Access

Arteriovenous Fistula Leading into Early-Onset Cerebral Venous Thrombosis

Man Bahadur Paudyal[1*], Neha Mehta[1], Niraj Gautam[2], Bijay Sharma[3], Bikas Baral[1] and Kusha Kc[1]

[1]*Department of Medicine, Tribhuvan University, Nepal*
[2]*Department of Neuro Medicine, Tribhuvan University, Nepal*
[3]*Department of Internal Medicine, Tribhuvan University, Nepal*

Abstract

Prothrombotic conditions generally ranging from acquired to genetic, oral contraceptives, malignancy, puerperium, infection, head injury are the common risk factors for cerebral venous thrombosis. Here, we present a case of 18 years males who developed recurrent cerebral thrombosis on the background of the presence of Arteriovenous Fistula.

Keywords: Thrombosis • Arteriovenous fistula • Protrombotic

Introduction

Cerebral venous thrombosis is an uncommon disease of the venous system. It is usually undiagnosed due to its varied presentation ranging from new-onset headache, seizure, altered mental status, and focal neurological deficit [1]. It is usually more common in females than males [2]. It may be due to increased risk of thrombosis during pregnancy, puerperium, and use of Oral contraceptive pills in females [3]. Risk factors for cerebral venous thrombosis vary from genetics, infections, inflammation use of medications with hematological malignancy [3]. Long-term cerebral venous thrombosis can lead to visual deficit [4]. We reported a case of chronic cerebral venous thrombosis due to an arteriovenous fistula with a visual deficit.

Case Report

18 Years old male known case of cerebral venous sinus thrombosis since 1 year presented to our hospital with a history of headache for 4 months and decreased vision on the right eye for the same duration. A patient complained of multiple episodes of headache associated with projectile vomiting, dull in nature, non-pulsatile persistent without seizure-like activity. Headache is associated with a decreased vision on the right side of the eye. A patient complained of more decreased vision sideways as compared to central vision. There was no history of double vision, difficulty in identifying color, deviation of angle of the mouth, hearing ability, and swallowing, altered sensorium. The patient was under anticoagulation therapy for the past events and also gave a history of warfarin free time for about 3 months duration before the onset of the symptom.

On Examination, he was well oriented to person, place, and time with a GCS score of 15/15. His pulse rate was 86 beats/minute, blood pressure was 100/70 mm of Hg, body temperature was 98°F, respiratory rate was 16 breaths/minute, and oxygen saturation was 95% in room air. All systemic examinations were unremarkable except bilateral papilledema on fundus examination.

Laboratory investigations showed hemoglobin 14.9 g/dl, and hematocrit 46.6%. The total leukocyte count was 11700/mm^3 neutrophils was 78%, and platelet count was 208000/mm [3]. The prothrombin time was 24.7 seconds and the International normalized ratio was 2.5. The protein S activity was 64% with a functional assay of 64% whereas Protein C activity in plasma was 90% with a functional assay of 90% The Antithrombin activity was 88% The D-dimer, VDRL, ANA, CRP, RA factor, Serology were negative. On CSF analysis, sugar was 70 mg%, the protein was 76 mg%, ADA was 8.1 U/L and LDH was 56 U/L.

A plain Computed Tomography (CT) scan of the brain shows bilateral hypodensity on the area of the transverse sinus. A Plain MRI study of the head shows abnormal hypointense signal along the course of bilateral transverse venous sinuses including torcular heterophil on T2/FLAIR while MRV shows heterogenous altered signal flow in bilateral transverse venous sinuses. Multiple collateral vessels are seen arising from bilateral transverse venous sinuses connecting to the different circulation. Cerebral Digital Subtraction Angiography (DSA) shows retrograde leptomeningeal venous drainage along with multiple feeders without intervening nidus. Superior sagittal sinus, inferior sagittal sinus, straight sinus, b/l transverse, and sigmoid sinus as well as visible cortical veins are grossly dilated suggestive of bilateral dural arteriovenous fistula with multiple collaterals (Figures 1-3).

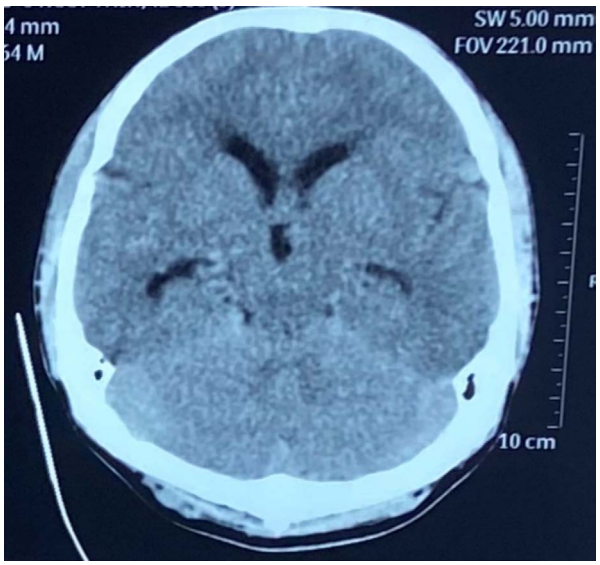

Figure 1. NCCT Head shows hypodensity on bilateral transverse sinus

**Address for Correspondence:* *Man Bahadur Paudyal, Department of Medicine, Tribhuvan University, Nepal, Email: paudyalman2023@gmail.com*

Received: 29-March-2023, Manuscript No. jnd-23-98332; **Editor assigned:** 31-March-2023, PreQC No. P-98332 (PQ); **Reviewed:** 14-April-2023; QC No. Q-98332; **Revised:** 19-April-2023; Manuscript No. R-98332 (R); **Published:** 26-October-2023, **DOI:** 10.4172/2329-6895.11.5.548

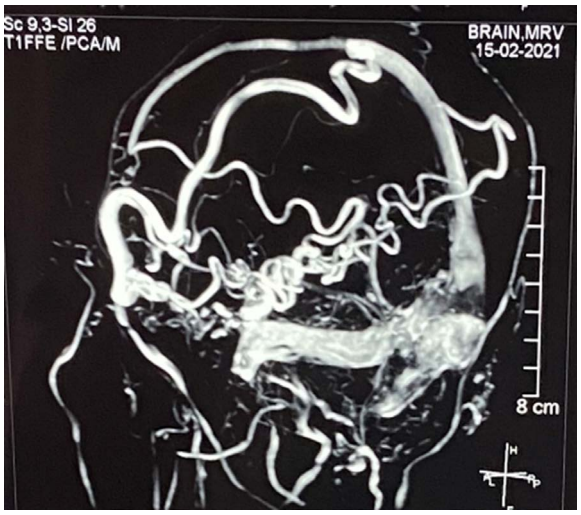

Figure 2. MR Venography shows heterogenous altered signal flow in bilateral transverse Venous sinuses

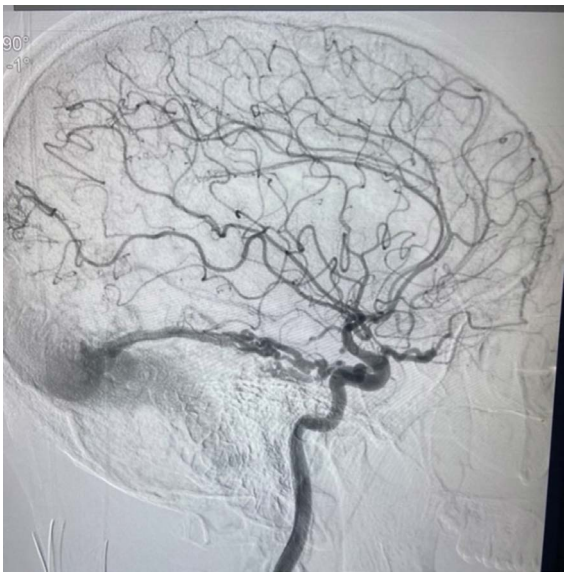

Figure 3. DSA shows bilateral giant arteriovenous fistula with multiple collaterals

Results and Discussion

Dural venous and cerebral venous thrombosis is less common and difficult to diagnose. With increasing clinical awareness along with the use of dynamic radiological investigation, there are increasing cases of Venous thrombosis. It usually affects middle-aged patients with more predominance of females than males [5]. Risk factors range from reversible to irreversible ones. It includes hereditary protein C and Protein S deficiency, dehydration, use of oral contraceptives pills, substance abuse, thrombophilias, head trauma, infections, connective tissue disorder, cancer, and many more [5].

Clinical presentation of the CVT is non-specific so it's difficult to diagnose [5]. Variable clinical presentation depends upon the extent, location, recanalization, and hypertension [6]. Most common clinical presentation of cerebral venous thrombosis is a headache (75%-95% pts) [7]. Focal neurological deficits, seizures, altered mental status, papilledema are the other common presenting complaints [1]. Focal neurological symptoms are more commonly seen with the patient with parenchymal changes as evidenced by imaging [6]. It has various clinical presentations varies from asymptomatic ones to serious clinical effects such as subarachnoid hemorrhage [8].

It's difficult to diagnose only based on history and examination basis. Urgent Neuroimaging is necessary to diagnose Cerebral Venous Thrombosis. We preferred to use Brain MRI and Magnetic Resonance (MR) venography or CT along with Cranial CT Venography if MRI is not available [1]. Most of the time CT head is non-specific. In some cases, CT demonstrates direct signs of CVT ranging from dense triangle sign-hyperdense triangular shape area seen on the posterior part of superior sagittal sinus on non-contrast CT. Empty delta sign is a triangular shape also seen in the posterior part of sagittal sinus on contrast CT with lacking central contrast enhancement. Cord sign is also seen in contrast CT which is linear hyperdensity on the cerebral cortex [9]. CT head is generally normal in most cases so CT venography might be helpful to aid in the diagnosis in the absence of MR venography. CT venography helps us by giving information about filling defects, sinus wall enhancement, and collateral venous drainage system [6].

MRI with T2 weighted image in combination with MR venography is the most sensitive test to diagnose the CVT. The MRI signal depends upon the age of the thrombus. Initial 5 days show isointense on T1 and hypointense on T2. After 5 days it became apparent on both T1 and T2. After a month it may show the variable pattern of the signal. We must know the different stages and their appearance on the different techniques to diagnose the CVT. In the older thrombus, we may need intra arterial angiography.

Digital Subtraction Angiography (DSA) CT angiography is a last resort imaging technique to find out predisposing factors in CVT. Congenital anomalies like craniofacial venous abnormalities may predispose to the development of the CVT. Digital Subtraction Angiography (DSA) CT angiography should be the preferred noninvasive modality to evaluate the case of CVT. Besides neuroimaging, American Heart Association recommends obtaining complete blood count (CBC), chemistry panel, prothrombin time, and activated partial thromboplastin time for the suspected patient. Investigation may reveal a hypercoagulable state, infection, and inflammatory process. Elevated D-dimer level supports the diagnosis of CVT but normal D-dimer levels do not rule out the diagnosis. Meta-analysis of 14 studies with a total of 1134 patients shows D-dimer was good with a sensitivity of 93.9% (95% CI 87.5-97.1) and specificity of 89.7% (95% CI 86.5-92.2). Generally protein C, Protein S along anti thrombin screening should be done in patients with suspected CVT.

Due to the variable presentation of the CVT, it can mimic different diseases. For a patient presenting with headache with or without vomiting, it should be differentiated with idiopathic intracranial hypertension, meningitis. Patients presenting with focal neurological deficit should be differentiated with intracranial hemorrhage, ischemic stroke, meningitis, intracranial space-occupying lesion.

Treatment of CVT should be started as soon as possible once it's confirmed. It consists of reversal of known factors if present, control of intracranial hypertension and seizures along with anticoagulation therapy. With the aim of recanalizing veins along with propagation of thrombus to the nearby veins, others body structure and prevent recurrence of CVT. Anticoagulation therapy generally started with either low molecular weight heparin (LMWH) subcutaneously or unfractionated heparin intravenously. Along with the benefits there is a risk of anticoagulation therapy in the patient with CVT. There is an increased risk of hemorrhage in previously infarcted areas.

Endovascular treatment (EVT) like thrombolysis doesn't show significance in patients with CVT. The TO-ACT Randomized Clinical Trial showed that EVT with standard medical care did not appear to improve the functional outcome of patients with CVT.

Conclusion

Although CVT is uncommon we should always keep CVT as our differentials on young adolescent age group patients present with long-term headaches.

We should always try to find out the risk factors of CVT at the early stages so we can prevent long-term sequelae of this disease.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Dmytriw, AA, Song JSA, Yu E and Poon CS. "Cerebral Venous Thrombosis: State of the art Diagnosis and Management." Neuroradiology 60(2018): 669-685.
2. Coutinho, JM, Ferro JM and Canhao P. "Cerebral Venous and Sinus Thrombosis in Women." Stroke 40(2009): 2356-2361.
3. Liu, KC, Bhatti MT and Chen JJ. "Presentation and Progression of Papilledema in Cerebral Venous Sinus Thrombosis." Am J Ophthalmol 213(2020): 1-8.
4. Gordon, DL. "The Diagnosis and Management of Cerebral Venous Thrombosis."
5. Leach, JL, Fortuna RB, Jones BV and Gaskill-Shipley MF. "Imaging of Cerebral Venous Thrombosis: Current Techniques, Spectrum of Findings, and Diagnostic Pitfalls." Radiographics 26: 19-42.
6. Mehta, A, Danesh J and Kuruvilla D. "Cerebral Venous Thrombosis Headache." Curr Pain Headache Rep 23(2019): 1-6.
7. Benabu, Y, Mark L, Daniel S and Glikstein R. "Cerebral Venous Thrombosis Presenting with Subarachnoid Hemorrhage: Case Report and Review." Am J Emerg Med 27(2009): 96-106.
8. Virapongse, C, Cazenave C, Quisling R and Sarwar M, et al. "The Empty Delta Sign: Frequency and Significance in 76 Cases of Dural Sinus Thrombosis." Radiology 162(1987): 779-785.
9. Lee, EJY. "Signs in Imaging Radiology the Empty Delta Sign."

How to cite this article: Paudyal, MB, Mehta N, Gautam N and Sharma B, et al. "Arteriovenous Fistula Leading into Early-Onset Cerebral Venous Thrombosis." *J Neurol Disord*. 11 (2023):548.