Content available at: https://www.ipinnovative.com/open-access-journals

IP Indian Journal of Neurosciences

Journal homepage: https://www.ijnonline.org/

Case Report

Drying up the drip- A case report

Poornima Nair[1]*, Sivakumar K[2]

[1]*Dept. of Neurology, Government Medical College, Thiruvananthapuram, Kerala, India*
[2]*Dept. of Internal Medicine, Coimbatore Medical College, Coimbatore, Tamil Nadu, India*

ARTICLE INFO

Article history:
Received 02-10-2023
Accepted 27-12-2023
Available online 22-04-2024

Keywords:
CSF Rhinorrhea
Venous sinus thrombosis
Meningoencephalitis
Cerebral venous

ABSTRACT

Cerebral Venous sinus thrombosis is an uncommon entity, which is usually associated with thrombosis of the dural venous sinuses due to local and clotting mechanism abnormalities. CVT secondary to CSF rhinorrhea has been recorded in literature but the relationship is uncertain.
Here we report a case of a 28yr old male with a past history of road traffic accident who presented with features suggestive of meningoencephalitis. He was diagnosed to have Cortical Venous Thrombosis, on further evaluation he was found to have CSF rhinorrhea.

1. Introduction

Cerebral venous sinus thrombosis is not an uncommon etiology. In any patient presenting with unexplained diffuse or unilateral headache, with new onset seizures, the possibility of CVT should be ruled out. There are multiple causes for CVT which include infection, trauma, surgery, hypercoagulable states, intracranial hypotension, lumbar puncture, drugs, collagen vascular diseases.[1] Isolated intracranial hypotension is a rare but significant cause of CVT. In a study, Schievnik and Maya found that CVT was present only in 3 out of 141 patients with spontaneous intracranial hypotension. Traumatic CSF Rhinorrhea is a rare but potentially devastating condition that leads to significant mortality and morbidity to the patient. Most (90%) are by penetrating and closed head trauma, which can be early or delayed. Early occurs within 48hrs & delayed which is more common, occurs within 3 months of the injury. Delayed leak beyond 3 months is very rare <5%. Traumatic leaks usually stop spontaneously- within a week in 70% of patients, and within 2-3 months in 20-30% patients and within 6 months in most patients.[2]

2. Case Report

A 28yr old male was admitted with a history of multiple episodes of generalized tonic clonic seizures and vomiting, one day history of high grade fever and severe headache. There were no other complaints. Past history revealed a road traffic accident four years back with traumatic brain injury. He had sustained fracture of the frontal bone, nasal bone, with bilateral maxillary and frontal hemosinus. There were no documented seizures at that time, however he had taken antiepileptics for one month and discontinued later.

Patient noted that he had recurrent episodes of unilateral watery discharge from right nostril while stooping forward since then. There were no episodes of fever/ altered behavior or seizures. He also had recurrent episodes of bifrontal headache not associated with any red flag symptoms for the past three years.

In the present admission, he was drowsy and disoriented, febrile with sinus tachycardia. Central nervous system examination revealed normal cranial nerves, normal fundus without papilledema with bilateral plantar mute. He had

* *Corresponding author*.
E-mail address: pnairbhavana@gmail.com (P. Nair).

https://doi.org/10.18231/j.ijn.2024.008

signs of meningeal irritation including neck stiffness and Brudzinski's leg sign. Other system examinations were within normal limits.

Investigations showed a hemoglobin of 14.7 mg/dl, total count of 16500, (L4/N89) and platelet count of 4.58lakhs/ cubcmm.

Blood sugars (146 mg/dl) and renal function test (Urea-22mg/dl, Creatinine- 0.9mg/dl, serum sodium- 144mEq/ dl, serum potassium -3.4mEq/ dl)were normal.

Liver function test (-Bilirubin-total-0.5;direct/indirect-0.4mg/dl, SGOT/SGPT- 47/33, ALP 99.Total protein- 7.7, A/G- 4.6/3.9) was normal. Serum Cholesterol was 229 mg/ dl, Triglycerides- 114 mg/ dl. Bleeding time was 30 secs, Clotting time was 5min, Prothrombin time was 14.1sec & INR was 1.01.

VDRL was non-reactive. Peripheral smear study and USG abdomen were normal

CSF analysis showed opening pressure 150mmCSF, cells 20/cu.mm (only lymphocytes), sugar 94 mg/dl(GRBS- 134 mg/dl); protein 18 mg/dl,LDH 34 U/L. CSF AFB and gram stain were negative. CSF culture and sensitivity revealed no growth.

He was provisionally diagnosed as acute meningoencephalitis and was treated with empirical antibiotics and mannitol.

Over a few daystime, the patient's conscious level and irritable behavior improved, there were no further episodes of seizures/vomiting.

MRI Brain with MRA and MRV revealed Superior sagittal sinus thrombosis with Significant leptomeningeal enhancement and dehiscence of right cribriform plate. Noncontrast CT brain showed the presence of Old fracture with dehiscence of cribriform plate on the right side. Patient was started on Inj.heparin 5000u iv q8h and later on Tab Acitrom 2mg OD.

With the unusual site of thrombosis and presence of cribriform plate dehiscence, possibility of ascending infection as an etiology for SSS thrombosis was thought of.

Within this period, the patient had 2 more episodes of CSF rhinorrhea.CECT brain was done which showed venous hemorrhagic infarct right frontal lobe.

CT CSF CISTERNOGRAM was done which showed a defect in right Cribriform plate 0.8 cm x 0.4 mm, with fluid at the nasal roof suggestive of active CSF leak in the right cribriform plate.

He underwent Trans-nasal endoscopic CSF leak closure under GA. Patient was stable after the procedure and was discharged with antiepileptics and anticoagulants. He was reviewed 2 weeks after discharge. Till date,the patient has no further episodes of rhinorrhea.

3. Discussion

Cerebral venous sinus thrombosis is not an uncommon etiology. In any patient presenting with unexplained diffuse or unilateral headache, with new onset seizures, the possibility of CVT should be ruled out. There are multiple causes for CVT which include infection, trauma, surgery, hypercoagulable states, intracranial hypotension, lumbar puncture, drugs, collagen vascular diseases.[1] Isolated intracranial hypotension is a rare but significant cause of CVT. In a study, Schievnik and Maya found that CVT was present only in 3 out of 141 patients with spontaneous intracranial hypotension.

Traumatic CSF Rhinorrhea is a rare but potentially devastating condition that leads to significant mortality and morbidity to the patient. Most (90%) are by penetrating and closed head trauma, which can be early or delayed. Early occurs within 48hrs & Delayed which is more common, occurs within 3 months of the injury. Delayed leak beyond 3 months is very rare <5%. Traumatic leaks usually stop spontaneously- within a week in 70% of patients, and within 2-3 months in 20-30% patients and within 6 months in most patients.[2]

In our case, the presentation was that of acute meningoencephalitis. The patient gives a clear history of prolonged headache and persistent watery discharge from the right nostril for the past 4 years. Hypercoagulability risk factors such as protein S, protein C, factor V Leiden mutation, antithrombin III and prothrombin G20210A mutation were all negative in our case.

This gives 2 possible etiology for the cause of CVT. First one, is an ascending infection via the fractured cribriform plate in the background of head injury four years back. Second one, persistent csf leakage leading to intracranial hypotension.

As per literature, superior sagittal sinus thrombosis due to ascending infection is rare, especially in young adults. This narrows down the cause to intracranial hypotension as the cause for CVT in the light of other investigations. The relationship between these two entities is uncertain.

3.1. Why CVT in intracranial hypotension?

Pathophysiology of dural venous sinus thrombosis in patients with intracranial hypotension is multifactorial. Etiologies include venous stasis, vascular distortion secondary to brain sagging, and CSF depletion leading to increased blood viscosity in the dural venous sinuses.

SIH is a risk factor for CVT, and approximately 2% of SIH patients develop CVT. A change in headache pattern is not a reliable predictor of the development of cerebral venous thrombosis in patients with SIH.

According to the Monroe-Kellie hypothesis, the intracranial volume is always constant. Therefore, any decrease in CSF volume is compensated by an increase in the blood volume. As the hydrostatic pressure within the dural venous sinuses increases, the meninges become engorged, followed by transudation of fluid into the subdural and subarachnoid space by means of increased

hydrostatic gradient. Further decrease in CSF may lead to increased microvascular permeability and result in blood in the subdural space.

A proposed mechanism of this condition is that a low CSF pressure causes a downward shift of the brain with increased pressure on the cerebral veins and sinuses. The increased pressure compresses the venous walls, thereby increasing the risk of thrombosis. Use of myelography or epidural injection has confirmed this theoretical possibility.[3]

3.2. CT Cisternogram in CVT

Patients with intermittent CSF rhinorrhea may have false negative CT cisternography. CT cisternography can also miss cribriform or ethmoid sinus defects. Surgical correction is the first line management in delayed traumatic leaks with two approaches- transcranial and extracranial. Over the past few years, endoscopic repair has gained importance and currently Functional Endoscopic Sinus Surgery is the first line surgical option for correction of cribriform plate defect.[4,5]

There is currently insufficient evidence to support definitive treatment guidelines of CVT in SIH. Since the development of CVT is closely related to the pathophysiological changes after CSF leak, the primary focus of treatment should be the treatment of SIH with Extradural blood patch(EBP).[6,7] Effective and prompt EBP, even without anticoagulation therapy, might lead to a good prognosis in selected cases. It could be possible that anticoagulation might increase the risk of intracranial hemorrhage (particularly subdural hemorrhage) in SIH patients, although a firm conclusion could not be drawn based on the limited number of patients currently available. The use of anticoagulation therapy should be weighed against the intracranial hemorrhage risk and should be monitored carefully when initiated.[8]

4. Conclusion

We report this case to highlight the rarity of its presentation. Patient had initially presented with features of meningoencephalitis later found to have SSS thrombosis, which was due to CSF rhinorrhea.

CSF leak leading into CVT is not much reported in literature and most of the cases reported have been presented within two months of the inflicting event. Our case was presented 3 years after trauma.

We also want to high-light that since a definite surgery is available for cribriform plate defect leading to CVT, a timely intervention can prevent further complications. CSF rhinorrhea should be taken with caution and a proper history should be elicited.

5. Source of Funding

None.

6. Conflict of Interest

None.

7. Acknowledgement

My sincere thanks to Dr. Geetha T, Professor of Medicine and my colleagues for their constant support.

References

1. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol*. 2007;6(2):162–70.
2. Coutinho JM. Cerebral venous thrombosis. *J Thromb Haemost*. 2015;13(Suppl 1):238–44.
3. Mehdiratta M, Kumar S, Selim M, Caplan L. Cerebral venous sinus thrombosis. In: Caplan L, editor. Uncommon Causes of Stroke, 2nd Edn. New York: Cambridge Univer- sity Press; 2008. p. 497–504.
4. Leach JL, Fortuna RB, Jones BV, Gaskill-Shipley MF. Imaging of cerebral venous thrombosis: current techniques, spectrum of findings, and diagnostic pitfalls. *Radiographics*. 2006;26(Suppl 1):19–41.
5. Luo Y, Tian X, Wang X. Diagnosis and treatment of cerebral venous thrombosis: a review. *Front Aging Neurosci*. 2018;10:2. doi:10.3389/fnagi.2018.00002.
6. Mokri B. Spontaneous Intracranial Hypotension. *Continuum (Minneap Minn*. 2015;21(4 Headache):1086–8.
7. Schievink WI, Maya MM. Cerebral venous thrombosis in spontaneous intracranial hypotension. *Headache*. 2008;48(10):1511–9.
8. Wasay M, Khan M, Rajput HM, Farooq S, Memon MI, Alrukn SA, et al. New Oral Anticoagulants versus Warfarin for Cerebral Venous Thrombosis: A Multi-Center, Observational Study. *J Stroke*. 2019;21(2):220–3.

Author biography

Poornima Nair, Senior Resident

Sivakumar K, Professor of Medicine

Cite this article: Nair P, Sivakumar K. Drying up the drip- A case report. *IP Indian J Neurosci* 2024;10(1):43-45.