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SUBARACHNOID HAEMORRHAGE ASSOCIATED WITH CEREBRAL SINUS THROMBOSIS – A RARE CASE

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Abstract

Cerebral venous thrombosis is a condition that requires greater clinical and radiological experience due to the significantly wider range of clinical signs and significant radiological variability.

When the patient presents with SAH (subarachnoid haemorrhage), the challenge can be great.

We present a case of cerebral venous thrombosis that radiologically presented with SAH without initial involvement of the parenchyma, which is why early diagnosis was important.

We present a 36-year-old patient, with clinical manifestation of acute headache, elements for SAH on CT (computerised tomography) without initial involvement - haemorrhage of the brain parenchyma.

CT angiography and venography using MRI (magnetic resonance imaging) demonstrated extensive thrombosis of the jugular vein, sigmoid and transverse sinus, with no other cause for SAH. Complete recanalization of the venous sinuses and significant improvement of the clinical diagnosis were reached after adequate anticoagulation therapy.

The findings indicated that venous sinus thrombosis may initially manifest as unilateral SAH without the involvement of the brain parenchyma.

Keywords: cerebral venous thrombosis, subarachnoid haemorrhage, CT and MRI venography, acute headache

Introduction

Cerebral venous thrombosis is a condition often difficult to define because of the wide range of clinical signs with which it presents. One of its rarest manifestations can be subarachnoid haemorrhage, which is considered the hardest to be recognized [1, 2].

The most common presentation is a headache that is usually acute, diffuse, initially minor, but gradually intensifying. A headache with SAH characteristics occurs in a small number of patients, but it is also characteristic for patients with migraines[3]. Other clinical presentations may include seizures, papilloedema, focal neurological deficiency, and impairment of consciousness.

We present a patient with signs of deep venous thrombosis associated with SAH.

Case report

A 36-year-old patient was brought in by emergency medical services for suspicion of epileptic seizure. The patient had briefly lost consciousness, and had full-body spasms. On

admission to the hospital, he was conscious, responsive, visibly distraught, pulse 90, blood pressure 100/70.

Clinical examination demonstrated generalised enhanced reflexes, slight weakness of the right side, lightly lowered right oral angle, Babinski negative on both sides. No smoking and no alcohol consumption. The patient reported a sharp headache that persisted for a whole week, diffuse, initially mild but gradually increasing in intensity. Laboratory analyses indicated presence of D-dimers 3000.

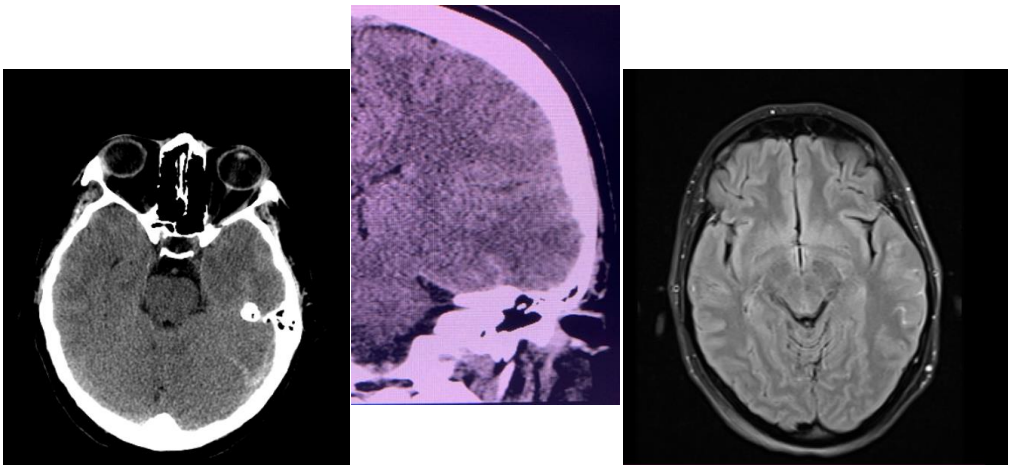
CT examination was performed immediately on admission to the hospital and MRI after 24h. The native CT scan, as well as the FLAIR pulse sequence, indicated gyral linear hyperdensities most compatible with SAH with hyperdense assigned left transverse sinus (figure 1).

A cortical hypersignal of T2 and FLAIR with a slight restriction of diffusion was observed, which indicated a venous infarction without elements of bleeding (figure 2).

Venography on MRI demonstrated absence of the left transverse sinus and jugular vein **(**figure 3).

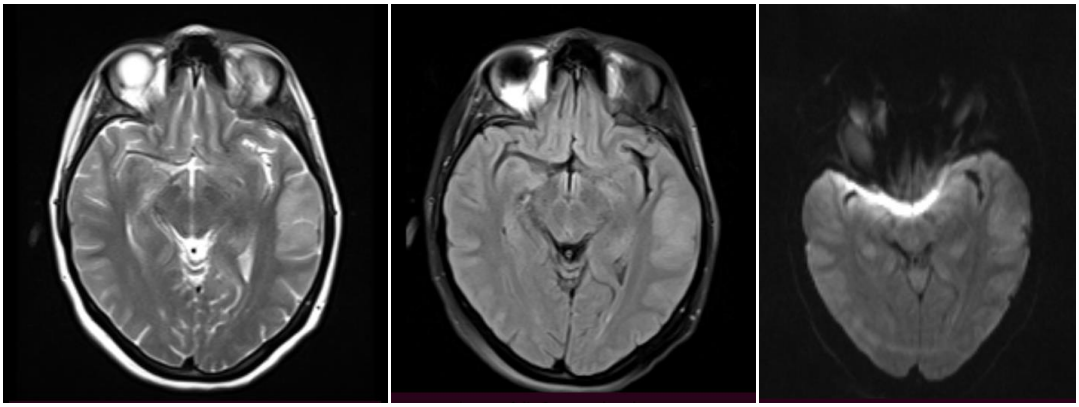
Extensive filling defects were seen at the level of the left jugular vein, the left sigmoid sinus, as well as the left transverse sinus as a result of massive venous thrombosis **(**figure 4).

After adequate anticoagulation therapy, at the check-up MRI, complete recanalization of the venous flow was indicated **(**figure 5).



A **B** **C**

Figure 1. Native CT scan – **(A, B)** as well as FLAIR **- (C)** pulse sequence, showed gyral linear hyperdensities most compatible with SAH with a hyperdense assigned left transverse sinus.



A **B** **C**

Figure 2. Cortical hypersignal area of the T2 pulse sequence **– (A)** and FLAIR - (**B)** was observed with light restriction of DWI-diffusion - (**C),** which indicates venous infarction without elements of bleeding.

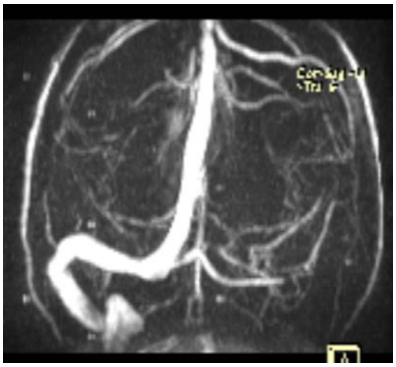


Figure 3. Venography on MRI demonstrates absence of the left transverse sinus and jugular vein.

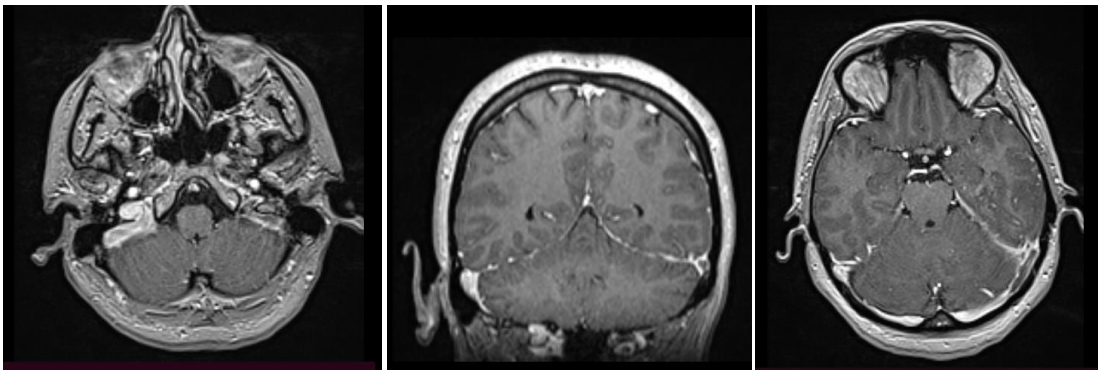


Figure 4. Extensive filling defects at the level of the left jugular vein, the left sigmoid sinus, as well as the left transverse sinus as a result of massive venous thrombosis.

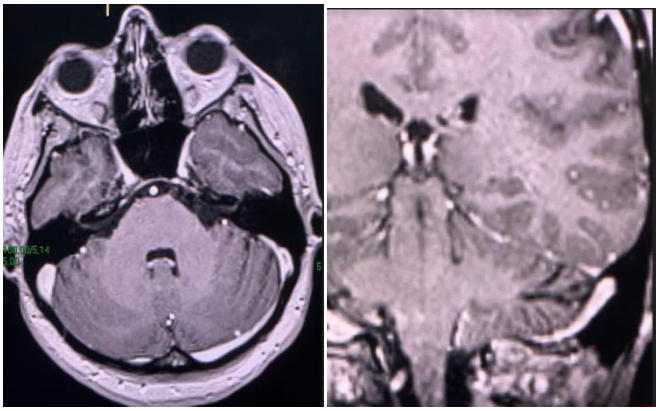


Figure 5. Complete recanalization of venous sinuses after adequate treatment with anticoagulation therapy.

Discussion

Venous cerebral thrombosis represents 1-2% of strokes in adults [4]. There are more than 100 etiological factors associated with this condition, among which the most common are the use of oral contraceptives, postoperative conditions, coagulopathies, dehydration and malignancy [5]. Clinical manifestations are highly variable, most commonly presented as headaches (95%), seizures (47%), focal motor deficits (43%), papilloedema (41%), altered state of consciousness (39%), intracranial hypertension (20%), or coma (15%) [6].

Globally, mortality rates range somewhere between 5-30%, but several studies have shown a high mortality rate of up to 50% in untreated patients [7, 8, 9], so early diagnosis and treatment are crucial and of extreme importance.

Subarachnoid haemorrhage is considered a very rare manifestation of cerebral venous thrombosis, described in the literature in a small number of cases and is a factor of significant difficulty when establishing the diagnosis [10, 11].

The exact cause of its association with cerebral venous thrombosis is subject to many hypotheses, but most theories suggest a slight disruption of the small cortical veins wall with secondary sequential haemorrhagic infarction or venous hypertension [12].

The main distribution is along the cortical sulci on the convexity, with the preservation of the basal cisterns [13].

In patients with cerebral venous thrombosis, dural sinuses are affected as much as 98%, with the involvement of cortical veins most often secondary to retrograde propagation of the primary thrombus [14]. Clinical manifestations and risk factors are not well established, but isolated cases suggest they are analogous to venous sinus thrombosis.

When symptoms are not strongly expressed and the clinical diagnosis is easy, the diagnosis can often be omitted or underestimated, due to frequent anatomical variations and cortical vein distribution.

The association of deep vein thrombosis and SAH is a very rare condition. In the literature, only a few cases with this condition are described. In a study by Panda *et al*., about 4.4% of patients with dural venous thrombosis were registered to have SAH [15].

Another study by Oda *et al*. showed that DVST was associated with 3% of SAH [16].

In one of the mentioned cases, the literature describes a connection with deep venous thrombosis where CT scan detected frontal gyral linear hyperdesities, but those changes were interpreted as dilatation and cortical venous stasis.

The lumbar sphincter in patients with CVT most often shows the presence of red blood cells. In a series of 32 patients with CVT, 50% had more than 100 erythrocytes/mm 3sub CSF, however, no subarachnoid haemorrhage was shown on the CT scans [17, 18].

When reviewing world literature, to date a very small number of patients who have isolated cortical vein thrombosis presented on CT scan with visible SAH in the absence of parenchymal involvement have been analysed and published [19].

In our case, the initial native non-contrast scan showed signs of left temporal cortical SAH without elements of bleeding at the level of the parenchyma, probably as a result of thrombosis of the sigmoid or transverse sinus. CT angiography, venography and post-contrast series confirmed the diagnosis.

Magnetic resonance imaging and venography on the magnetic resonance imaging are pivotal imaging techniques for establishing the diagnosis and monitoring of patients with venous thrombosis.

Conclusion

SAH is a very rare initial presentation of venous cerebral thrombosis. Recognition of radiological signs, presence of SAH and venous sinus thrombosis is essential, thus examination continues with a venography for accurate identification of the size of the thrombotic mass. Adequate and timely treatment with anticoagulant therapy significantly improves the clinical outcome.

References

1. Oppenheim C, Domigo V, Gauvrit JY, Lamy C, Mackowiak-Cordoliani MA, Pruvo JP, et al. Subarachnoid hemorrhage as the initial presentation of dural sinus thrombosis. Am J Neuroradiol 2005;26(3):614-617.
2. Adaleti I, Sirikci A, Kara B, Kurugoglu S, Ozer H, Bayram M. Cerebral venous sinus thrombosis presenting with excessive subarachnoid hemorrhage in a 14-year-old boy. Emerg Radiol 2005;12(1-2):57-59.
3. Zare M, Mirabdolbaghi P. Cerebral venous thrombosis presenting as subarachnoid hemorrhage and treated with anticoagulants. JRMS 2005;10:251-254.
4. Chang R, Friedman DP. Isolated cortical venous thrombosis presenting as subarachnoid hemorrhage: a report of three cases. AJNR 2004;25(10):1676-1679.
5. Bousser MG. Cerebral venous thrombosis: nothing, heparin or local thrombolysis. Stroke 1999;30(3):481-483.
6. Kimber J. Cerebral venous sinus thrombosis. QJM 2002;95(3):137-142.
7. Van Gijn J. Cerebral venous thrombosis: pathogenesis, presentation and prognosis. J R Soc Med 2002;93(5):230–233.
8. Lafitte F, Boukobza M, Guichard JP, Reizine D, Woimant F, Merland JJ. Deep cerebral venous thrombosis: imaging in eight cases. Neuroradiology 1999;41(6):410–418.
9. Yuh WT, Simonson TM, Wang AM, et al. Venous sinus occlusive disease: MR findings. AJNR Am J Neuroradiol 1994;15(2):309–316.
10. Chakraborty S, terBrugge K, Farb R, Mikulis D. Case of the month. Isolated cortical vein thrombosis of left vein of Labbe. Can Assoc Radiol J 2008;59(5):271–274.
11. Urban PP, Mülle-Forell W. Clinical and neuroradiological spectrum of isolated cortical vein thrombosis. J Neurol 2005;252(12):1476–1481.
12. Sakaki T, Matsuyama T, Nagata K, Nakase H, Hirabayashi H, Morimoto T. Delayed intracerebral haemorrhage after intracranial surgery. J Clin Neurosci 1999;6(1):54-57.
13. Bittencourt LK, Palma-Filho F, Domingues RC, Gasparetto EL. Subarachnoid hemorrhage in isolated cortical vein thrombosis: are presentation of an unusual condition. Arq Neuropsiquiatr 2009;67(4):1106–1108.
14. Miranda V H, Mellado T P, Sandoval R P, Huete L I. Isolated cortical vein thrombosis: report of two cases. Rev Med Chil. 2007;135(10):1313-1317.
15. Panda S, Prashantha DK, Ravi Shankar S, Nagaraja D. Localized convexity subarachnoid haemorrhage - a sign of early cerebral venous sinus thrombosis. Eur J Neurol. 2010;17(10):1249-1258.
16. Oda S, Shimoda M, Hoshikawa K, Osada T, Yoshiyama M, Matsumae M. Cortical subarachnoid hemorrhage caused by cerebral venous thrombosis. Neurol Med Chir (Tokyo). 2011;51(1):30-36.
17. Leach JL, Strub WM, Gaskill-Shipley MF. Cerebral venous thrombus signal intensity and susceptibility effects on gradient recalled-echo MR imaging. AJNR 2007;28(5):940-945.
18. Spitzer C, Mull M, Rohde V, Kosinski CM. Non-traumatic cortical subarachnoid haemorrhage: diagnostic work-up and aetiological background. Neuroradiology 2005;47:525-531.
19. Wang YF, Fuh JL, Lirng JF, Chang FC, Wang SJ. Spontaneous intracranial hypotension with isolated cortical vein thrombosis and subarachnoid haemorrhage. Cephalalgia 2007;27(12):1413-1417.