



Subarachnoid Hemorrhage as a Complication of Cerebral Venous Thrombosis

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ABSTRACT

Cerebral venous sinus thrombosis (CVST) is an uncommon but important cause of stroke in young women. Subarachnoid hemorrhage (SAH) secondary to CVST is a rare presentation. We present a case of 40-year-old female who presented with hemicranial headache. Magnetic resonant imaging (MRI) showed CVST along with SAH and venous infarcts. The patient improved on anticoagulant therapy. This report highlights the value of early diagnosis of CVST through neuroimaging and the importance of immediate anticoagulation as a part of patient management.

Keywords: Anticoagulant therapy, Cerebral venous thrombosis, Neuroimaging, Subarachnoid hemorrhage.

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INTRODUCTION

Cerebral venous sinus thrombosis is a disease seen with increasing frequency in daily practice with a variety of nonspecific clinical symptoms. It most frequently presents as an acute onset throbbing type of headache which may or may not be associated with neurological deficit or seizures.¹ The acute stage of CVST may be associated with complications like venous infarction, SAH, and pulmonary embolism. The SAH is becoming more frequently recognized as a potential complication of CVST. The presentation of CVST sometimes greatly mimics SAH presenting as thunderclap headache in more than 10% of cases. Seizures and hemiparesis, possible manifestations of SAH, occur in about one-third of patients. However, CVST presenting with an associated SAH on computed tomography and MRI is infrequent.^{2,3} Thus, it is impor-

tant to establish whether SAH is due to CVST, as this requires a completely different treatment from SAH due to a leaking aneurysm.

CASE REPORT

A 40-year-old female was admitted to our ward with complaints of sudden-onset severe right hemicranial throbbing type of headache and six to seven episodes of vomiting since 5 days. Patient also complained of heaviness on the right side of the body. She had no other complaints. She had no significant past medical history. On admission, she was afebrile with a pulse of 58 bpm and blood pressure of 140/80 mm Hg. Rest of physical and neurological examination was normal with no evidence of any focal neurological deficit or signs of meningism. Magnetic resonance imaging was done which showed venous infarcts in both parietal lobes, SAH over right parietal lobe, and thrombosis of posterior aspect of superior sagittal sinus, right transverse sinus, right sigmoid sinus, and proximal aspect of right internal jugular vein (Figs 1 and 2). The magnetic resonance angiography was normal with no evidence of any aneurysm (Fig. 3).

She had no history of irregular periods and no history of taking oral contraceptive pills. Routine lab investigations were normal. The patient underwent a complete coagulation profile testing including prothrombin time, activated prothrombin time, antiphospholipid, and anticardiolipin antibody titer, protein C, protein S, antithrombin III, and homocysteine levels. All the results were in the normal range except for high homocysteine level, which was 35 $\mu\text{mol/L}$ (normal value $<15 \mu\text{mol/L}$). Treatment was started with analgesics, mannitol, and anticonvulsants. In addition, subcutaneous low-molecular-weight heparin 60 mg/day was started and later raised to 120 mg/day on 3rd day which was continued for 1 week. Patient was closely monitored. She was subsequently started on oral anticoagulation with warfarin, maintaining international normalized ratio between 2.0 and 3.0, after it was confirmed that there was no further SAH. Patient showed marked clinical improvement in 6 weeks. Repeat MRI done after 6 weeks also showed regression of SAH and resolving venous sinus thrombosis (Fig. 4). Subsequently, she was followed up in the outpatient department every 2 weeks for 12 months.

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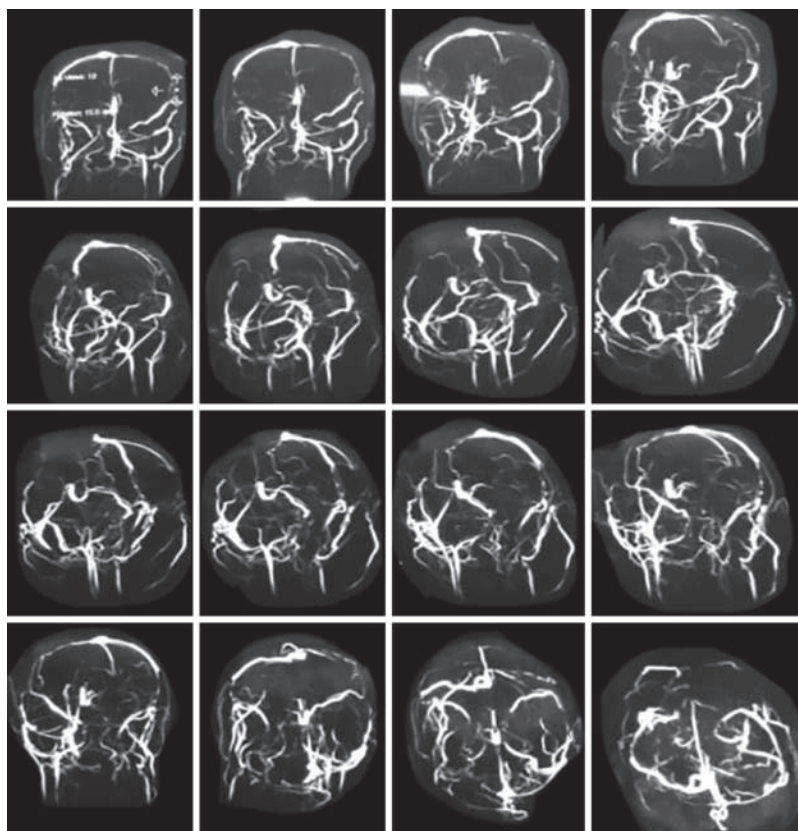


Fig. 1: MRI showing venous thrombosis involving posterior aspect of superior sagittal sinus, right transverse sinus, right sigmoid sinus, and proximal aspect of right jugular vein

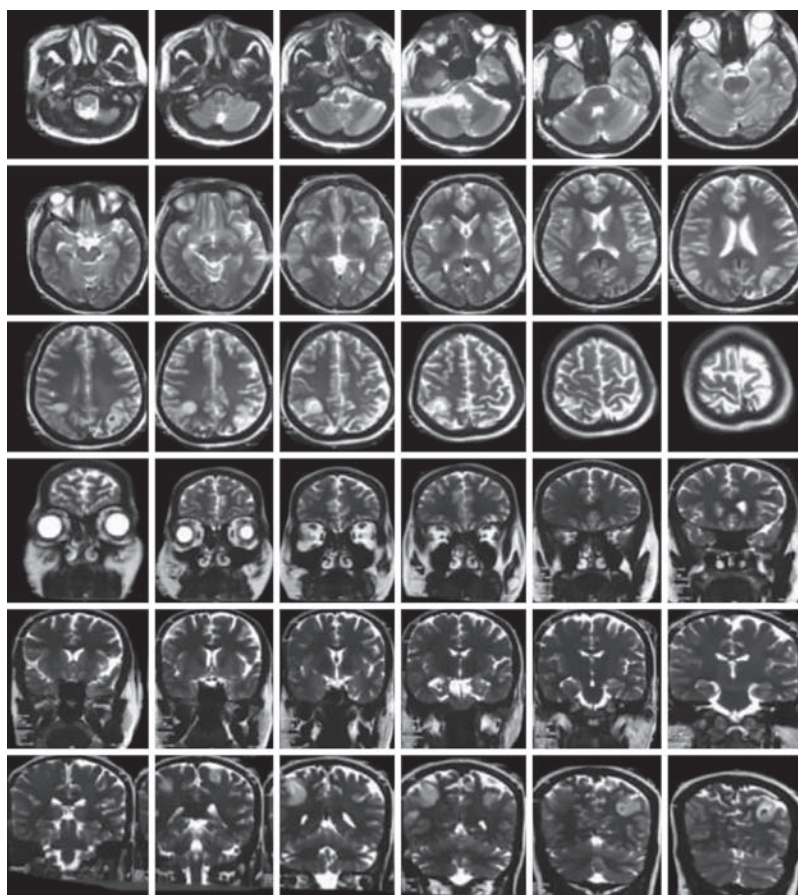


Fig. 2: MRI showing venous infarcts and SAH over right parietal lobe

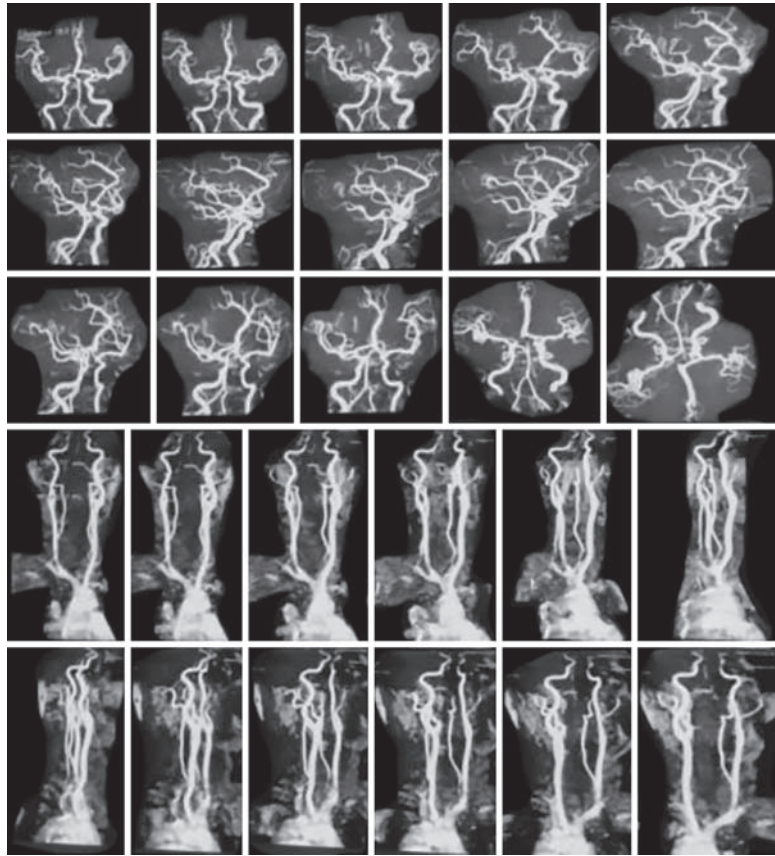


Fig. 3: MR angiogram shows no evidence of any aneurysm

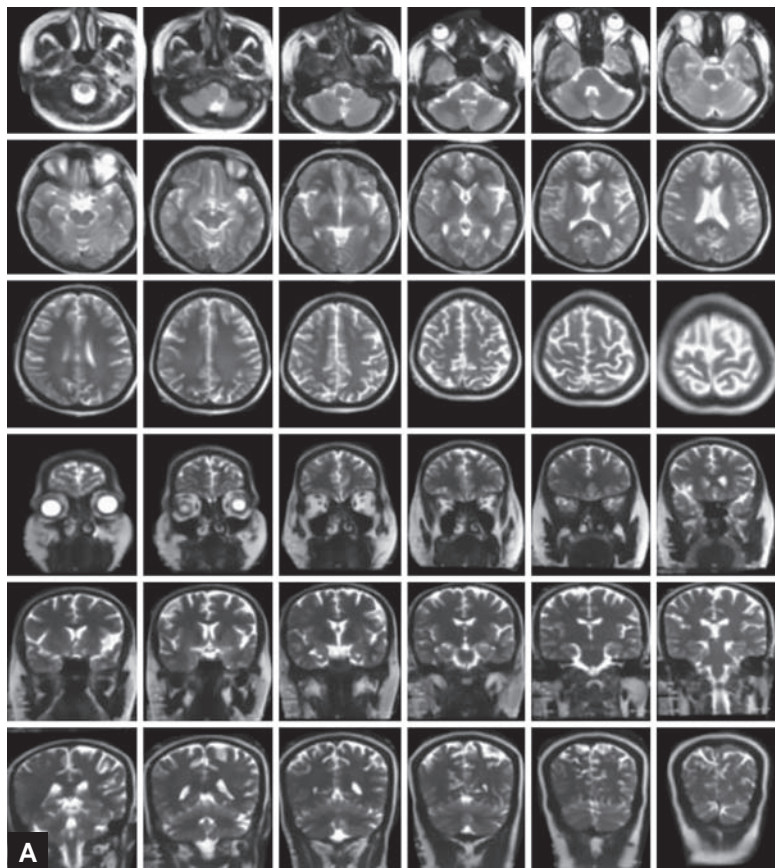
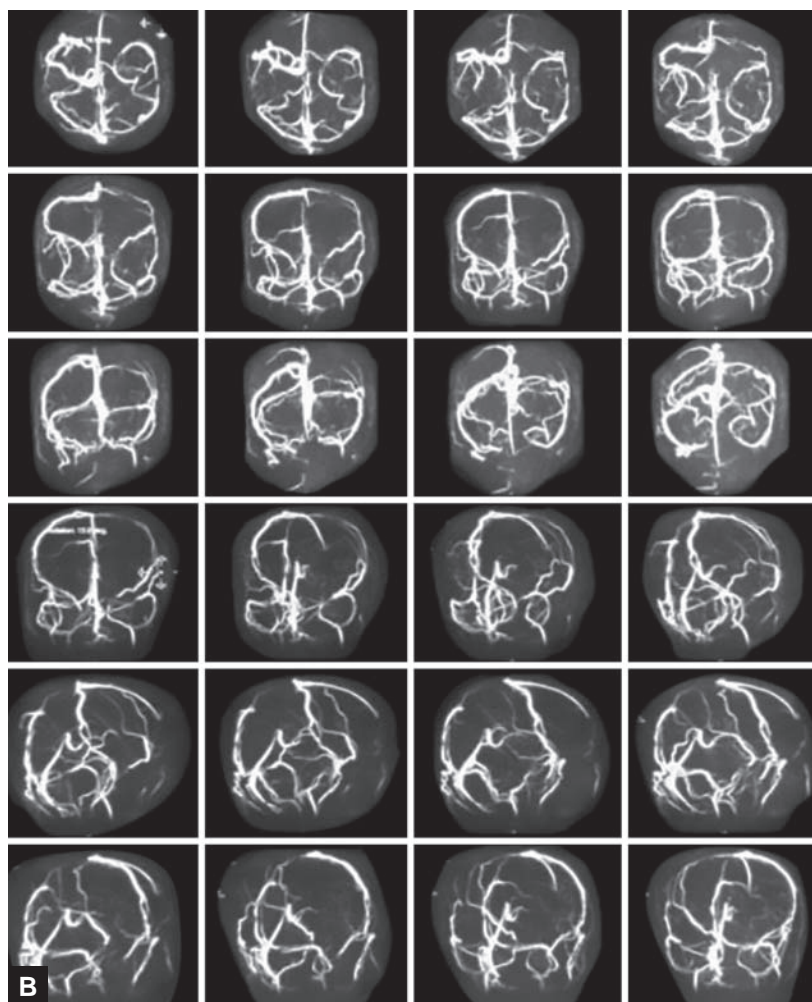


Fig. 4A:



Figs 4A and B: (A and B) Repeat MRI after 6 weeks of starting oral anticoagulants showing regression of SAH and resolving venous sinus thrombosis

DISCUSSION

Cerebral venous sinus thrombosis is a well-known cause of cortical SAH. The CVST is seen in 3 to 4% of all strokes in adults. Cortical SAH without the involvement of the basal cisterns may suggest underlying CVST. The distribution of SAH due to CVST is usually localized to the cerebral convexity and spares the skull base and basal cisterns.⁴ The exact cause of mechanism of SAH secondary to CVST remains unknown. One possibility is rupture of venous parenchyma hemorrhagic infarcts into the subarachnoid space.^{5,6} Our patient showed no signs of hemorrhagic venous infarction. Another possible mechanism is venous hypertension and subsequent rupture of dilated, valveless, thin-walled, bridging subarachnoid cortical veins which lack smooth muscle.⁵ The third mechanism of SAH could be local inflammatory response caused by CVST, which would increase the vascular permeability, allowing for extravasation of blood into the subarachnoid space. This is the most likely cause in our patient, who, because of her altered metabolism of homocysteine, had a proinflammatory status.

Management of SAH secondary to CVST is quite different from that of arterial SAH. The usual treatment of

sinus thrombosis is anticoagulation or local thrombolysis. Systemic anticoagulation is the first line of treatment for CVST, as it is safe, effective, and not too costly.⁷ In our case, we preferred to initiate treatment with low doses of fractionated heparin, 60 mg once a day, which was increased to 120 mg/day on the 3rd day for 1 week and later followed by oral anticoagulants with effect from the 2nd week. During this period, we observed the patient carefully for any deterioration due to increase in SAH. We did not use intravenous heparinization, as we thought that it may cause more SAH. Review of literature shows that very early anticoagulation in SAH due to CVST is avoided due to fear of bleeding.⁸ More studies like ours are required to find out the appropriate timing of starting anticoagulation safely in a case of SAH secondary to CVST.

CONCLUSION

Subarachnoid hemorrhage is a rarely seen complication of CVST. All patients with neurological symptoms, even a simple headache, should be carefully evaluated using neuroimaging to analyze the underlying cause and initiate treatment accordingly as early as possible. Furthermore,

in cases of CVST, a complete study of thrombophilia and procoagulative profile work-up should be done to identify the possible cause and appropriate treatment should be given. Though initiation of early anticoagulation with low-molecular-weight heparin has given good results in our case, more studies are required before we can say the final word.

REFERENCES

1. Iurlaro S, Beghi E, Massetto N, Guccione A, Autunno M, Colombo B, DiMonda T, Gionco M, Cortelli P, Perini F, et al. Does headache represent a clinical marker in early diagnosis of cerebral venous thrombosis? A prospective multicentric study. *Neurol Sci* 2004 Oct;25(Suppl 3):S298-S299.
2. Martelli N, Colli BO, Assirati JA Jr, Machado HR. Cerebromeningeal hemorrhage. Analysis of autopsies performed over a 10-year period. *Arq Neuropsiquiatr* 1988 Jun; 46(2):166-175.
3. Benabu Y, Mark L, Daniel S, Glikstein R. Cerebral venous thrombosis presenting with subarachnoid haemorrhage. Case report and review. *Am J Emerg Med* 2009 Jan;27(1): 96-106.
4. Panda S, Prashantha DK, Shankar SR, Nagaraja D. Localized convexity subarachnoid haemorrhage – a sign of early cerebral venous sinus thrombosis. *Eur J Neurol* 2010 Oct;17(10): 1249-1258.
5. Cuvinciuc V, Viguier A, Calviere L, Raposo N, Larrue V, Cognard C, Bonneville F. Isolated acute nontraumatic cortical subarachnoid hemorrhage. *Am J Neuroradiol* 2010 Sep;31(8):1355-1362.
6. Kato Y, Takeda H, Furuya D, Nagoya H, Deguchi I, Fukuoka T, Tanahashi N. Subarachnoid hemorrhage as the initial presentation of cerebral venous thrombosis. *Intern Med* 2010 Jan;49(5):467-470.
7. Rodallec MH, Krainik A, Feydy A, Helias A, Colombani JM, Julles MC, Marteau V, Zins M. Cerebral venous thrombosis and multidetector CT angiography: tips and tricks. *Radiographics* 2006 Oct;26(Suppl 1):S5-S18.
8. 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 Jan;51(1):30-36.