

Cortical venous thrombosis presenting with subarachnoid haemorrhage

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BRIEF REPORT

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ABSTRACT

Background

Our study retrospectively reviewed the presentation, neuro-radiological findings, and outcomes of eight adult patients presenting at our institution with subarachnoid haemorrhage (SAH), which was subsequently proven to be due to cortical venous thrombosis (CVT).

Methods

We reviewed the case records and neuroimaging findings of eight patients diagnosed with SAH and CVT over a span of two years at our institution, a tertiary care centre in Western India. All details pertaining to their presentation, clinical findings, neuroimaging, management, and outcome following therapy with anticoagulants were collected until patient discharge.

Results

There were a total of eight patients, with the average age being 34 years (range 25–42). Only one patient was female. Six patients had a history of recent binge drinking. None of the patients had a past or family history of common risk factors for thrombosis. All patients presented acutely, with headache (n=6) and seizures (n=6) being the most common

presenting features, occurring in three-quarters of the patients examined. Non-contrast computed tomography (NCCT) was the initial imaging study for all but one of the patients and showed cortical SAH (cSAH) without basilar haemorrhage. Magnetic resonance imaging/magnetic resonance venography (MRI/MRV) confirmed the underlying CVT. Unfractionated heparin was used in all cases. Seven patients improved and were discharged on oral anticoagulation. The eighth patient died.

Conclusion

Localised cSAH with sparing of basal cisterns can be a presentation for CVT. In patients with cSAH, MRI/MRV can be useful to make a diagnosis of CVT. Anticoagulation for CVT, even in the presence of SAH was related to seven out of eight patients being discharged.

Key Words

Cortical venous thrombosis, subarachnoid haemorrhage, non-contrast CT

What this study adds:

1. What is known about this subject?

Subarachnoid haemorrhage is most commonly traumatic or associated with aneurysmal rupture.

2. What new information is offered in this study?

Our paper describes the presentation and neuroradiological features of eight patients presenting with SAH and CVT.

3. What are the implications for research, policy, or practice?

Recognising cSAH as an important presentation of CVT is clinically important as anticoagulation is recommended in this clinical setting.

Background

Subarachnoid haemorrhage (SAH) is an emergent condition, which can cause critical neurological dysfunction from both

primary and secondary brain insult.¹ Despite recent advances in medicine and treatment modalities, mortality and morbidity due to SAH is high.² Epidemiological data indicates that the incidence of SAH increases with age peaking in the sixth decade of life; SAH affects women more than men in the ratio of 3:2.³ Patients without an identifiable aetiology for their SAH have better long-term prognosis than patients with an aneurysmal SAH.⁴

Non-aneurysmal SAH can be further characterised as perimesencephalic (involving the basal spaces) or convexal; i.e., distributed over the cerebral convexities, characteristically sparing the basal cisterns. This distinction that can be made on initial CT scans.⁵ These differences have important implications in determining the underlying aetiology, treatment, and prognosis.⁶

Diverse aetiologies such as cerebral amyloid angiopathy, posterior reversible encephalopathy syndrome, cortical venous thrombosis (CVT), vasculitis, and vascular malformation are responsible for convexal subarachnoid haemorrhage (cSAH).⁷ Numerous cases have been described in the literature of SAH as a presentation of CVT.^{8–12} Diagnosis of CVT may be delayed due to its protean manifestations and subtle findings on CT imaging as well as a lack of suspicion for this rare but treatable condition.

We present the experience at our centre of recognising and treating eight cases of CVT presenting with SAH. This paper aims to outline the importance of diagnosing cases of CVT presenting as SAH as there are significant diagnostic and therapeutic implications especially with regard to the use of anticoagulants.

Method

We reviewed the case records and neuroimaging findings of eight patients diagnosed with SAH associated with CVT presenting over a span of two years at our institution, a tertiary care centre in Western India. No patient was on anticoagulation (for CVT or any other indication) when SAH was diagnosed. All details pertaining to their presentation, clinical findings, neuroimaging, management, and outcome following therapy with anticoagulants until patient discharge were collected. Only adult cases with all the pertinent details available were included. Comparisons were made with cases and studies in prior literature.

Results

There were a total of eight patients, with a mean age of 34 years (range 25–42). Only one patient was female. Six patients consumed alcohol daily, and reported frequent

binge drinking. None of these six patients had abnormal liver function tests and their international normalised ratio (INR) was normal. None of the patients had a past or family history of thrombosis, recent surgery, trauma, or use of oral contraceptive pills (OCPs). Smoking status of the patients cannot be determined. Other common risk factors such as pregnancy or post-partum state, central nervous system infections, or cancer were also not found in any of the patients.

Amongst the two patients who did not have any history of alcohol intake, one had sickle cell trait and the other (the only female patient) had diabetes. No obvious predisposing factors for CVT were evident amongst the patients. All patients presented acutely, that is, within the first seven days. Six patients presented with severe headache and seizures. Focal deficits such as hemiparesis were seen in two patients. Non-contrast computed tomography (NCCT) was the initial imaging study for all but one of the patients and showed cSAH without basilar haemorrhage; “cord sign” (cordlike hyperattenuation within a dural venous sinus due to dural venous thrombosis) was seen in only one patient. All patients underwent magnetic resonance imaging/magnetic resonance venography (MRI/MRV), which confirmed the underlying CVT. The superior sagittal sinus was involved in all cases. Sigmoid and transverse sinuses were also involved in two cases.

All patients were treated with unfractionated heparin with close clinical monitoring. One patient died on day two despite being managed on the intensive care unit on mechanical ventilation and given a mannitol infusion; the cause of death was deemed to be raised intracranial pressure (based on the clinical findings of papilloedema and decorticate rigidity). The other patients improved and were discharged on oral anticoagulation with regular INR monitoring for six months. Results are shown in Table 1, with representative images in Figure 1.

Discussion

CVT is a relatively rare condition, predominantly affecting women, accounting for 0.5–1 per cent of all strokes.¹³ The diagnosis of CVT often proves to be elusive due to the large spectrum of clinical manifestations. SAH is being increasingly recognised as one of the presentations of CVT in recent years most likely due to strides made in diagnosing CVT with improved and non-invasive radiology.^{14,15} Retrospective studies of large CVT series at tertiary neurology institutes have shown an incidence of SAH in 3 per cent¹⁶ to 4.3 per cent⁹ of cases reviewed.

CVT may be a more common condition in the Indian scenario than in the Western world.¹⁷ There was a striking majority of males among our case series. No precipitating cause may be seen in up to one-third of cases; in our series, apart from a single case of sickle cell trait, no other underlying causes (including dehydration) could be found. However, the possible role of alcohol intoxication with subsequent dehydration and increased platelet reactivity has previously been suggested,^{18,19} therefore, it is notable that three-quarters of the patients we examined had a history of recent heavy bouts of intake. This raises a question regarding the role of alcohol as a risk factor for CVT or cSAH that needs to be further explored.

There is considerable overlap between the clinical features of CVT and SAH. Headache, altered mentation, focal neurological deficits, and features of raised intracranial pressure may be frequently seen in both entities. Although cephalgia is classically described as hyper-acute and severe in SAH compared to CVT, “thunderclap” headache has been described in CVT as well, even without associated SAH. Seizures may be seen in SAH (regardless of the aetiology) and CVT.¹⁴ Nuchal rigidity is an often described classical finding in aneurysmal SAH, but was seen in only one of our patients, perhaps due to the localised form and limited amount of bleeding. The onus of diagnosis thus falls on neuroimaging.

NCCT is usually the first step for patients presenting acutely with neurological symptoms. Reviews have shown that sensitivity of initial CT studies for SAH in patients with CVT and concomitant SAH are as high as 86–91 per cent.^{14,15} However, various characteristic radiological findings described for CVT on NCCT are not common and much less specific features such as oedema, haemorrhage, or infarction alone or in combination are the norm.¹⁵ The gold standard today for CVT diagnosis is an MRI that visualises the thrombosis along with MRI venography to document the absent flow in the occluded sinus.¹³ Circumscribed cSAH along the cerebral convexities with sparing of basal cisterns is usually localised to the region of dural sinuses or cortical veins.

Several mechanisms causing SAH in CVT have been suggested: (1) secondary rupture of cortical venous haemorrhagic infarcts into the adjacent subarachnoid space; (2) increased vascular permeability caused by local inflammatory response to cortical venous thrombosis leading to the localised extravasation of blood; and/or (3) dilation and rupture of thin-walled cortical veins due to secondary venous hypertension.¹⁴ Cortical irritation due to

the localised bleeding with or without underlying infarction is a likely explanation for the increased occurrence of seizures and focal deficits in these patients, especially when compared to perimesencephalic SAH.³

Anticoagulation is recommended in most patients of CVT along with symptomatic treatment, i.e., antiepileptic medication, measures for raised intracranial pressure, and visual failure.¹³ Although evidence is limited, most guidelines recommend antithrombotic treatment despite evidence of intracerebral haemorrhage²⁰ and several case reports^{8–12,14–16} attest to this practice even in the presence of SAH. Thus in the unique setting of SAH associated with CVT, anticoagulation is indicated despite known intracranial haemorrhage.

The major limitation of the present study was its retrospective nature. However, only those cases that had complete clinical, laboratory, and imaging data were included in the series. Screening for the commonly known causes of CVT was done on the basis of history, clinical examination, and investigations in all the patients. Daily alcohol use with history of recent binge drinking (without a diagnosis of chronic liver disease) was present in 75 per cent of the cases, suggesting a possible association. However, this study was not designed to demonstrate a causal relationship, although it has been postulated before.^{18,19,21}

Conclusion

Localised cSAH with sparing of basal cisterns may provide an early clue to underlying CVT. CVT is a potentially fatal condition making it important for clinicians and radiologists to be aware of this perhaps under-recognised association, to ensure timely and accurate diagnosis. MRV should be obtained for the confirmation of diagnosis. Anticoagulation is used in cases of CVT complicated with cSAH.

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PEER REVIEW

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CONFLICTS OF INTEREST

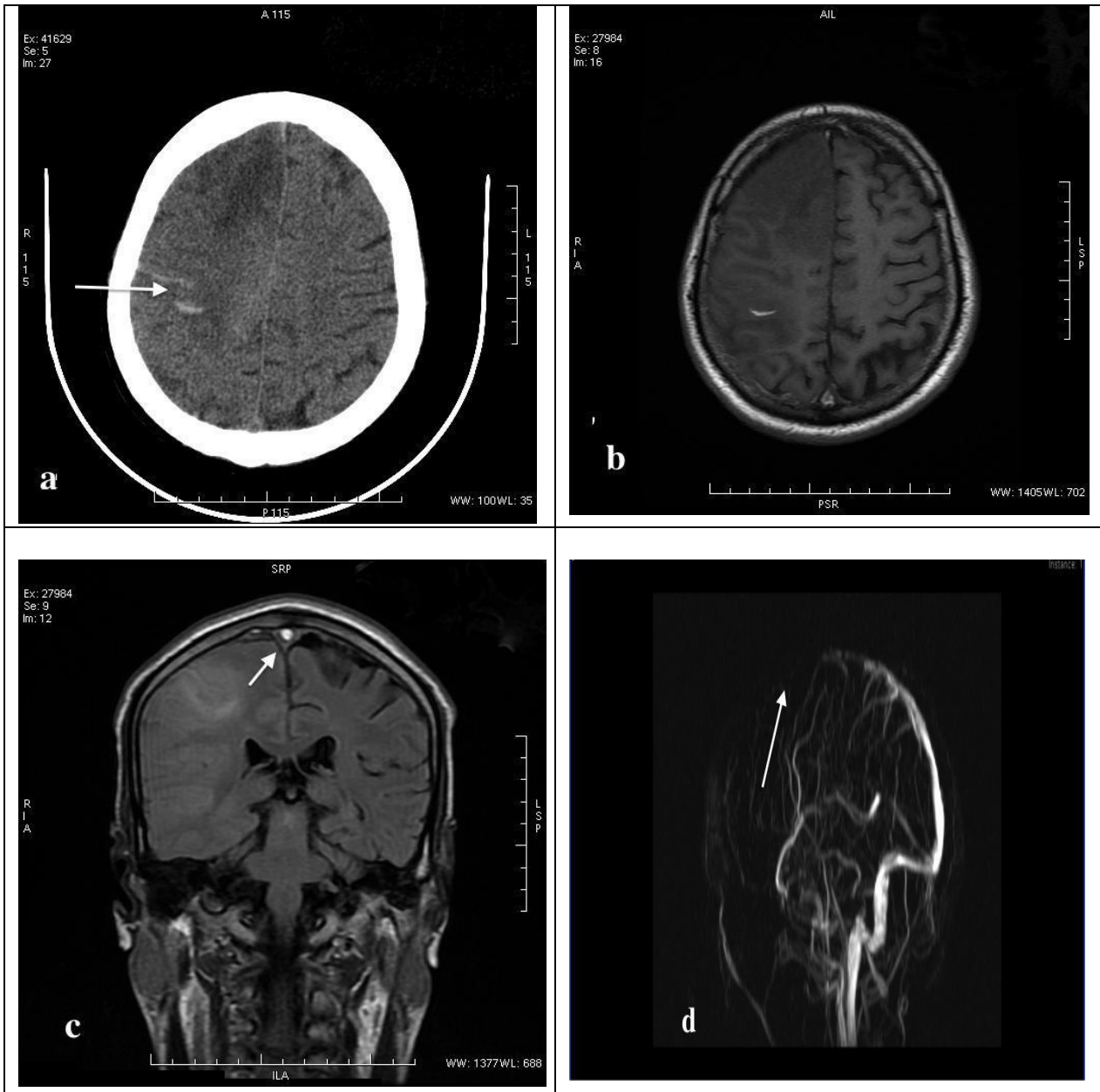
The authors declare that they have no competing interests.

Table 1: Clinical and neuroimaging details of eight patients, presenting with cortical subarachnoid haemorrhage (cSAH) as a complication of cortical venous thrombosis (CVT)

Sr. No	Age/Sex	History	Presenting Signs and Symptoms	Clinical Findings	Imaging	
					NCCT	Magnetic Resonance
1	25/M	Chronic daily alcoholic	Headache, GTC and altered sensorium x 1 day	Right hemiparesis	Haemorrhagic venous infarct in the left frontoparietal lobe. Mild SAH over bilateral cerebral convexities	Anterior 2/3 rd superior sagittal thrombosis
2	30/M	Chronic alcoholic, recent binge	Status epilepticus x 3 hours	Generalised hypotonia. Bilateral Babinski's response	Intra cerebral haemorrhages in bilateral parietal lobes and SAH in bilateral high parietal lobe sulci. Cord sign present.	Haemorrhagic infarct in left parietal lobe. SAH in bilateral parietal regions with superior sagittal sinus and right sigmoid and transverse sinus thrombosis
3	36/M	k/c/o sickle cell trait	Severe 5-day headache. GTC since 2 days.	Bilateral Babinski's response	Bilateral frontoparietal SAH.	Superior sagittal sinus thrombosis
4	35/M	Acute alcohol binge on night prior to symptoms in a chronic alcoholic.	Severe 7-day headache. GTC since 2 days.	Drowsy, signs of raised intracranial tension and Bilateral Babinski's response	Left parietal subcortical venous infarct and left parietal sulci SAH.	Thrombosis of anterior ½ of superior sagittal sinus.
5	25/M	Acute alcohol binge	2-day severe headache. Single episode of GTC.	No significant findings.	Right frontal cortical SAH.	Superior sagittal thrombosis
6	42/M	Chronic daily alcoholic	GTC followed by right-sided hemiparesis.	Diminished attention. Rt sided limb weakness, hypotonia and upgoing plantar.		Acute left fronto-parietal lobar haemorrhage with perilesional cytotoxic oedema, subarachnoid extension and mass effect. Acute complete venous sinus thrombosis in superior sagittal sinus and adjacent cortical veins.
7	42/F	Diabetes mellitus	3-day severe headache, altered sensorium x 1-day	Unconscious, bilateral upgoing plantars. No focal deficits. Papilledema +. Neck rigidity +.	Bilateral frontoparietal cerebral oedema with overlying cortical subarachnoid haemorrhage	Superior sagittal sinus thrombosis with bilateral cytotoxic oedema of bilateral frontal and parietal lobes.
8	40/M	Chronic daily alcoholic	Severe headache, vomiting, altered sensorium and left-sided hemiparesis x1 day.	Unconscious left-sided weakness and hypotonia, bilateral upgoing plantars.	Right parietal lobar haemorrhagic infarct with localised overlying sulcal SAH	Thrombosis of superior sagittal, right transverse and right sigmoid sinus. Haemorrhagic infarct with perilesional infarct in right parietal-frontal lobe.

M-Male, F-Female, GTC-Generalised tonic clonic seizures, SAH-Subarachnoid haemorrhage

Figure 1: Neuroimaging findings of cortical venous thrombosis (CVT) presenting with circumscribed subarachnoid haemorrhage



(a) Non-contrast CT image cortical-subcortical, wedge-shaped hypodensity in bilateral frontal lobes suggestive of ischemic infarcts, with linear gyriform hyper-densities in right parietal region (arrow) suggestive of cortical subarachnoid haemorrhage; (b) MRI shows hyperintensities suggestive of acute ischemic infarcts in right frontoparietal region with haemorrhage in left frontal region; (c) coronal view shows absence of flow void (arrow) suggestive of sagittal sinus thrombosis; (d) MRV displaying superior sagittal sinus thrombosis (arrow).