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Review Article

Internal Medicine Section

Cerebral Venous Thrombosis Presenting as Cortical Subarachnoid Haemorrhage- A Case Report and Review

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ABSTRACT

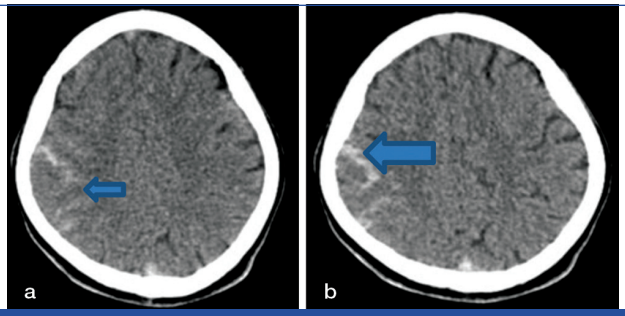
In the presence of Subarachnoid Haemorrhage (SAH), diagnosis of underlying Cerebral Venous Thrombosis (CVT) is challenging as there is no difference in clinical presentation and therapeutically it is important because CVT needs to be treated with anticoagulant, unlike SAH. This article is about a 50-year-old male presenting with headache, right hemiparesis, and recurrent seizures. Computed Tomography (CT) head was suggestive of SAH in right posterior parietal region. But Magnetic Resonance Imaging (MRI) venogram showed cerebral venous sinus thrombosis. Hence, he was treated with anticoagulants. The patient showed significant clinical improvement. SAH secondary to underlying CVT is a relatively rare entity. After reviewing medical literature of such cases, 42 case reports and case series forming 95 cases of SAH secondary to CVT were found.

Keywords: Anticoagulation, Cerebral venous infarction, Non-aneurysmal subarachnoid haemorrhage

CASE REPORT

A 50-year-old male presented with complaints of headache and giddiness for two days, followed by generalised tonic clonic seizures for 2-3 minutes and post ictal drowsiness, which lasted for 20 minutes. Same day in the afternoon, he had second episode of convulsion after which, his relatives brought him to the hospital. There was no history of seizures, diabetes or hypertension. Patient was non alcoholic.

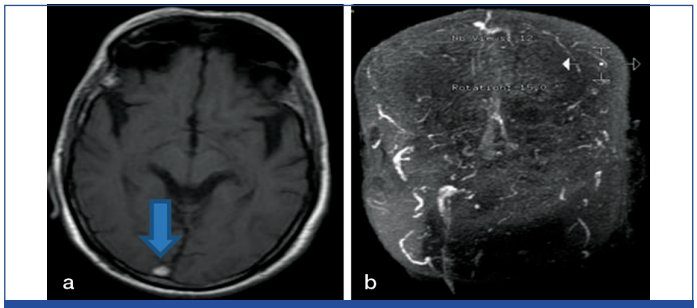
On clinical examination, the patient was afebrile, his pulse 72/minute regular, Blood Pressure (BP) 140/80 and Oxygen Saturation (SpO_2) was 98% on room air. He was drowsy, pupils were normal in size, reacting to light. Motor examination showed right sided hemiplegia. Planters were bilaterally extensor. Fundus examination showed bilateral papilledema. His non contrast Computed Tomography (NCCT) head was suggestive of SAH in right posterior parietal region [Table/Fig-1].



[Table/Fig-1]: CT head showing right parietal cortical subarachnoid haemorrhage.

The patient was admitted to Intensive Care Unit (ICU), and was started on intravenous levetiracetam 1 g twice daily and phenytoin 100 mg three times daily; but he continued to have recurrent right focal clonic seizures on second day too. So, intravenous lacosamide 200 mg, twice daily, was added and further he was put on midazolam infusion for two days. Finally, on the fourth day of admission, the seizures were controlled. In view of recurrent seizures and presence of SAH on CT head, MRI brain with MR angiogram and MR venography was done, which was suggestive of severe attenuation of superior sagittal sinus and bilateral transverse sinuses [Table/Fig-2]. The cerebral angiogram was normal. In view of venous sinus thrombosis with SAH, the patient was started on injection enoxaparin 0.6 mL subcutaneous once daily, on the fourth day of admission.

After three days of once daily enoxaparin doses, repeat NCCT head was done which showed organised parasagittal parieto-occipital



[Table/Fig-2]: Thrombosed sagittal and right transverse venous sinuses on T1 and MRV.

bleed with no evidence of SAH. The patient was continued on subcutaneous injection enoxaparin 0.6 mL twice a day for further one week. He showed significant improvement at the end of two weeks, as his level of consciousness improved, there was no headache and right-side motor power improved from grade 1/5 to 3/5. He had no seizures since day four of admission. The patient was discharged on antiepileptics and anticoagulant drugs. He was reassessed on follow-up after 15 days of discharge when his right-side motor power was 4-5/5, and he was completely seizure-free.

DISCUSSION

Rupture of an intracranial aneurysm is usually the most common cause of spontaneous SAH causing SAH in 85% cases. But in almost 15% of patients of spontaneous SAH, bleeding source of subarachnoid bleed cannot be identified despite repeated neuroimaging [1].

CVT is one of the causes of spontaneous non aneurysmal SAH though considered rare [2]. [Table/Fig-3] shows some of the causes of SAH. The reported cases SAH secondary to CVT, seems to be increasing over the years. Panda S et al., reported 10 (4.3%) of 233 patients of CVT having SAH [3]. In a retrospective review, Oda S et al., found 3% of CVT cases with SAH [4]. Boukobza M et al., reported 22 cases (6.63%; 22/332) of CVT presenting as SAH without haemorrhagic brain lesion [5]. The most likely reason for increasing number of cases appears to be the technological advances in radiological diagnosis and widespread availability in the last few years [6]. This entity is diagnostically and therapeutically distinct because of better prognosis and needs treatment with anticoagulants unlike aneurysmal SAH.

Literature review for SAH, secondary to CVT, was conducted on PubMed, MedLine and Google Scholar. The keywords were

Aneurysmal/Non-perimesencephalic (85%)	Non-aneurysmal/ Mesencephalic (10%)	Cortical SAH (cSAH) (5%)
Trauma	Trauma	Reversible cerebral vasoconstriction syndrome
Saccular aneurysm	Non-aneurysmal/ venous	Cerebral venous thrombosis
Non saccular aneurysm	Saccular aneurysm	Vascular malformation (Superficial)
Vascular malformation	Malformation or tumour mostly spinal	Cerebral amyloid angiopathy
Arterial dissection	Arterial dissection	Septic emboli, septic aneurysm
		Posterior reversible encephalopathy syndrome

[Table/Fig-3]: Causes of SAH according to location [1,2].

subarachnoid haemorrhaged, cerebral venous thrombosis, cortical SAH, and non aneurysmal SAH. There were 51 articles where these terms were present in abstract/title published from 1995 to 2021. These articles were screened, and the reference lists were also checked to find out relevant articles. Finally, 41 journal articles including case reports and reviews were compiled, which included a total of 95 cases. These cases were reviewed for their available demographic data, clinical and neuroimaging findings.

Diagnosis of CVT in presence of SAH poses diagnostic and therapeutic challenges [1]. CVT should be considered in the differential diagnosis of patients presenting with SAH without evidence of an aneurysm.

Approximately 80% of cases of SAH occur in people aged 40-65 years of age [2], and though CVT in males has uniform age distribution, females suffer from CVT at younger age of 20-35 years [6]. The present review included total 95 cases of CVT complicated with SAH at presentation. Mean age of patients was 43.6 years (range 14-83 years). There were 54 females (54/95) with a mean age of 43 years, and 41 males with a mean age of 44 years. Thus, there was no significant age difference. CVT is seen in females routinely (F:M 1.29:1) [6] and a female dominance is also found in cases of aneurysmal SAH (F:M 3:2) [2]. In the present review, the F:M ratio was 1.31: 1 in cases of SAH secondary to CVT, which is not different from that observed in the cases of aneurysmal SAH or CVT.

Aneurysmal SAH presents with seizure in 10-25% of cases in the acute phase [7]. On the other hand, seizures are more common and recurrent in CVT at presentation, occurring in about 40% of patients [8]. Ferro JM et al., found that about 39.2% (245/624) patients had seizures and 58 patients (9.3%) had focal seizures [9]. In the present review of 95 cases of SAH secondary to CVT, 36 (37.9%) patients had seizures which included generalised tonic clonic seizures or focal seizures [Table/Fig-4]. Focal neurological deficit was reported in 10.3% of 213 patients of SAH in a case series [10]. While in case of CVT, focal deficit was seen in upto 44% of patients which include motor weakness, aphasia and ataxia [8]. Aphasia is also a common neurological defect, that is observed in 19-24% of CVT patients. [6]. In the present case review as well, focal neurological deficit was reported in 35.8% (34/95) which included hemiparesis, monoparesis, sensory deficit, aphasia, dysarthria. Signs of meningism like Kernig's Sign, Brudzinski sign or neck stiffness was reported in 19/95 cases (20%). Small number of patients had dysarthria, aphasia or visual symptoms like blurring or aura [Table/Fig-4].

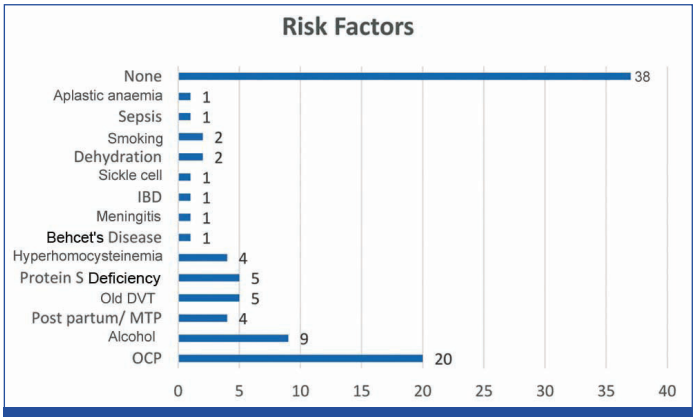
The SAH commonly presents as thunderclap headache, "the worst headache of life"- striking suddenly like a clap of thunder [7]. But thunderclap headaches have been reported in 5-13% of CVT cases too [8]. It is difficult to distinguish a thunderclap headache in CVT from that of subarachnoid haemorrhage. In this review, the most common presenting symptom was moderate to severe headache (76.8%), of which, 20% patients presented with thunderclap headache, which continued as mild to moderate ache. CVT presents with altered consciousness in 20-30.6% of patients [8]. SAH patients also present with loss of consciousness in 45% of cases,

Clinical presentations		Number	Percentage
Symptoms	Headache	73	76.8
	Seizure	36	37.9
	Focal neuro deficit	34	35.8
	Nausea/Vomiting	32	33.7
	Altered sensorium	23	24.2
	Meningism	19	20
	Thunderclap headache	19	20
	Papilledema	13	13.7
	Visual symptoms	6	6.31
	Aphasia	3	3.1
	Dysarthria	2	21
SAH Location	Peri mesencephalic	5	5.26
	Aneurysmal site	3	3.16
	Cortical SAH (cSAH)	87	91.58
CVT Location	Cortical vein	7	7.37
	SSS+TS	84	88.42
	Couldn't be assessed	4	4.21

[Table/Fig-4]: Clinical presentation of cases.

due to increased intracranial tension and 10% patients may remain comatose for several days [2]. In the present review, 24.2% (23/95) cases had altered level of consciousness in the form of reduced level of vigilance, drowsiness, or disorientation. Papilledema is a common manifestation of CVT that was observed in 28-67.5% of CVT patients [8]. In cases of SAH, papilledema and sub-hyaloid haemorrhage may be evident in 20-30% of patients [2]. In the present review, 13.7% patients with SAH secondary to CVT had papilledema. Though papilledema was reported in these cases of SAH secondary to CVT, it is difficult on clinical grounds solely to suspect that SAH visible on neuroimaging is secondary to CVT.

Smoking and heavy alcohol consumption are strong risk factors for SAH [2]. Risk of SAH also increases during pregnancy [1]. Though there are very few case reports showing smoking as a risk factor for CVT by causing significant polycythemia, a case-control study showed no relationship [12]. Though alcohol consumption has not been mentioned as independent risk factor for CVT [6], some studies had suggested associated dehydration and hyperviscosity related to alcoholism as predisposing factor for CVT [13,14]. Oral Contraceptive Pills (OCP), hyper-homocysteinemia, hypercoagulable states are not risk factors for subarachnoid haemorrhage [2]. In the present case review, 9 (9.5%) patients had a history of alcoholism and two patients were smokers. The most common predisposing condition for CVT was OCP (21.05%) in females, and alcoholism was the most common predisposing factor in male patients (9.5%). In majority of the patients, no predisposing factor was found (38.95%). Thus, the present review of cases of SAH secondary to CVT revealed risk factor profile which are similar to risk profile of CVT cases as compared to SAH. This may raise suspicion of underlying CVT in case of non-aneurysmal SAH [Table/Fig-5].



[Table/Fig-5]: Risk factors associated with CVT presenting as cSAH.

S. No.	Author, publication year, reference	Number of cases	Age	Sex	Headache	Seizure	Thunderclap headache	Nausae/ Vomiting	FND	Aphasia	Dysarthria	Alt Senso	Blurring Vision	Gait imbalance	ICT raised	Meningism	Type of SAH	SAH location	Venous sinus involved	Risk factors	Treatment given
1	Boukobza M et al., 2016 [5]	1	52	F	Y	Y		Y									cSAH	FTP, Parasagittal	SSS, Rt TS	OCP	LMWH BD
		2	49	F	Y		Y		Y		Y		Y		Y		cSAH	Temporal, Sylvian Fissure	Lt TS Sigmoid Sinus	OCP	LMWH BD
		3	27	F	Y	Y	Y										cSAH	Rt Frontal	SSS, Rt TS, Sigmoid Sinus	OCP	LMWH BD
		4	28	M	Y	Y											cSAH	Rt Temporal	RtTs Sigmoid Sinus	IBD	LMWH BD
		5	37	F	Y			Y				Y					cSAH	Rt Temporal	SSS, Rt TS Sigmoid Sinus	OCP, Abortion	LMWH BD
		6	35	M	Y	Y			Y							Y	cSAH	Rt Frontal	SSS	Meningitis	LMWH BD
		7	49	F	Y				Y								cSAH	Lt Temporal	Lt TS Sigmoid Sinus	Thrombocytemia	LMWH BD
		8	32	F	Y			Y	Y			Y					cSAH	Rt Frontal	SSS, Rt TS Sigmoid Sinus	OCP	LMWH BD
		9	33	F	Y												cSAH	Fronto Parietal		OCP, CSF Low P	LMWH BD
		10	42	F	Y	Y	Y										cSAH	Rt Frontal	SSS, Lt TS sigmoid	OCP	LMWH BD
		11	24	F	Y	Y		Y	Y			Y			Y		cSAH	Bilateral Frontal	SSS, Lt TS sigmoid	OCP	LMWH BD
		12	54	F	Y												cSAH	Lt Temporal	Lt TS & Sigmoid Sinus, Rt TS	Thrombocytemia	LMWH BD
		13	44	F	Y					Y		Y					cSAH	Lt Temporal	SSS Lt TS Sigmoid	OCP	LMWH BD
		14	43	F	Y												cSAH	Lt Frontal	SSS	APLA, OCP	LMWH BD
		15	31	M		Y											cSAH	Rt Frontal	SSS, Rt TS, Sigmoid Sinus	CVT old	LMWH BD
		16	28	F	Y								Y		Y		cSAH	Rt Temporal	Rt TS, Sigmoid Sinus	OCP	LMWH BD
		17	43	F	Y			Y					Y				cSAH	Rt Temporal	SSS, Rt TS	OCP, Factor V	LMWH BD
		18	43	F	Y							Y					cSAH	Rt Frontal B/L TemporoOccipetal	SSS, Rt TS	Past PE, DVT	LMWH BD
		19	49	M	Y			Y							Y		cSAH	Rt Temporal	SSS, Rt TS	None	LMWH BD
		20	38	F	Y				Y								cSAH	Rt Parietal	SSS	Postpartum	LMWH BD
		21	44	F	Y			Y									cSAH	Rt Temporal	SSS, Rt TS Sigmoid Sinus	Bachets	LMWH BD
		22	38	F	Y												cSAH	Rt Temporal	SSS, Rt TS Sigmoid Sinus	None	LMWH BD

2	Shastri M et al., 2015 [18]	23	25	M	Y	Y			Y			Y					cSAH	B/L Cerebral Convexity	SSS	Alcoholism	LMWH BD
		24	30	M		Y											cSAH	B/I High Parietal	SSS, Rt TS Sigmoid Sinus	Alcoholism	LMWH BD
		25	36	M	Y	Y											cSAH	B/I FP SAH	SSS	Sickel cell Trait	LMWH BD
		26	35	M	Y	Y									Y		cSAH	Left Parietal SAH	SSS	Alcoholism	LMWH BD
		27	25	M		Y			Y								cSAH	Left FP SAH	SSS	Alcoholism	LMWH BD
		28	42	F	Y							Y					cSAH	B/I FP SAH	SSS	None	LMWH BD
		29	40	M	Y			Y	Y								cSAH	Rt Parietal	SSS, Rt TS Sigmoid Sinus	Alcoholism	LMWH BD
		30	42	M		Y			Y								cSAH	FP SAH Left	SSS	Alcoholism	LMWH BD
3	Oda S et al., 2011 [4]	31	83	M								Y					cSAH	B/I Occipital Cortex	Transverse Sinus, Cortical Veins	Dehydration	LMWH BD
		32	67	F			Y										cSAH	Left Sylvian Fissure	Insular Cortical Vein	None	LMWH BD
		33	57	M			Y										cSAH	Left Convexity	Cortical Vein	None	LMWH BD
		34	31	F	Y												cSAH	Rt Convexity	Cortical Vein	Postpartum	LMWH BD
4	Oppenheim C et al., 2005 [19]	35	69	M	Y		Y										cSAH	Rt Frontal Convexity	SSS; Both TS	Old DVT	LMWH BD
		36	55	F	Y	Y	Y									Y	Aneurysmal	Bihemisperic Insular SAH	SSS, Lt TS	None	LMWH BD
		37	32	F	Y	Y		Y									cSAH	Ant Interhemispheric SAH	SSS, Rt TS	OCP	LMWH BD
		38	51	F	Y				Y						Y	Y	cSAH	Diffuse B/I Cortical SAH	SSS Cortical Vein Draining SSS	HRT, F/H/O DVT	LMWH BD
5	Chang R and Friedman DP, 2004 [15]	39	29	F			Y	Y	Y				Y				cSAH	Rt Cortical Convexity	Cortical Vein Trolard	OCP	LMWH BD
		40	46	F	Y								Y				cSAH	Rt Central Sulcus	Cortical Vein Trolard	None	LMWH BD
		41	63	F	Y										Y	Y	cSAH	Rt Convexity SAH	Cortical Vein	Sepsis	LMWH BD
6	Rice H and Tang Y, 2006 [20]	42	56	F	Y										Y	Y	cSAH	Rt FP Cortex	SSS, Lt TS	None	LMWH BD
7	Singh H et al., 2012 [13]	43	35	M	Y	Y						Y					cSAH	B/I FP SAH	SSS	Alcoholism	LMWH BD
8	Verma R et al., 2012 [21]	44	65	F	Y			Y							Y		cSAH	Perisylvian Fissure SAH	SSS	None	LMWH BD
9	Pradhan S et al., 2007 [22]	45	47	F	Y			Y	Y			Y				Y	cSAH	RtSylvian Fissure SAH	Straight Sinus, Lt TS, Inf SS	Protein S Deficiency	Conservative
10	Pangariya A et al., 2010 [23]	46	40	M	Y				Y							Y	cSAH	Lt FP, Rt Frontal	SSS, Rt TS Sigmoid Sinus	Alcoholism	LMWH BD

11	Yamamoto J et al., 2013 [24]	47	32	F	Y											Y	Aneurysmal	Basal Cistern, Sylvian Fissure	SSS, Rt TS		Craniot-omy
12	Sun J et al., 2018 [25]	48	57	F		Y		Y	Y	Y							cSAH	Rt FP Cortex, Temporal	Lt TS		Conserv-ative
13	Benabu Y et al., 2009 [26]	49	56	M	Y			Y									cSAH	Biparietal SAH	SSS	Hyperhomocyst	LMWH BD
14	Adaletli I et al., 2005 [27]	50	14	M	Y			Y						Y		Y	Aneurysmal	Basal Cistern, Sylvian Fissure	SSS, Straight sinus		LMWH BD
15	Sharma S et al., 2010 [28]	51	59	M	Y	Y		Y								Y	cSAH	Rt frontal parasagital	SSS, TS		LMWH BD
16	Mathew T et al., 2007 [29]	52	40	M	Y			Y	Y			Y					cSAH	Lt Parietal SAH,B/L SDH Parench	SSS, B/L TS	Alcoholism	LMWH BD
17	Glikstein et al., 2009 [26] (cross ref)	53	56	M	Y			Y									cSAH	Parietal SAH	SSS Rt TS	Smoker	LMWH BD
18	Rau CS et al., 2001 [30]	54	60	M	Y	Y		Y	Y						Y	Y	cSAH	FP SAH and Intraparenchymal Bleed	SSS, Rt TS	Thyrotoxicosis	LMWH BD
19	Tidahy E et al., 2004 [31]	55	27	F	Y			Y									cSAH	Temporal and Parietal region	SSS Rt TS	MTP OCP	LMWH BD
20	Shukla R et al., 2006 [32]	56	40	M	Y	Y		Y	Y			Y				Y	cSAH	Lt Sylvian Fissure	SSS, TS	None	LMWH BD
21	Spitzer C et al., 2005 [33]	57	41	F	Y				Y								cSAH	Rt Frontal	SSS	None	LMWH BD
		58	65	M		Y											cSAH	Rt Pre central SAH	SSS	None	LMWH BD
		59	42	M	Y							Y					cSAH	Bifrontal SAH	SSS, Rt TS	None	LMWH BD
22	Sztajzel R et al., 2001[34]	60	58	F			Y										cSAH	Rt cerebellar SAH	Rt TS	Old DVT	LMWH BD
23	Lin JH et al.,2006 [35]	61	44	M		Y	Y										cSAH	Rt Parietal SAH	SSS, Rt TS		LMWH BD
24	Ciccone A et al., 2000 [36]	62	36	F		Y	Y	Y	Y			Y					cSAH	Sylvian Fissure Lt, Post Temporal	Lt Sylvian Vein	OCP	LMWH BD
25	de Bruijn S et al., 1996 [37]	63	37	F	Y			Y								Y	Perimesen-cephalic	Infratentorial SAH	B/L TS, Sigmoid	OCP	LMWH BD
26	WidjajaE et al 2003 [38]	64	33	F	Y			Y	Y				Y				cSAH	Rt Sylvian Fissure SAH	SSS, middle cerebral vein	OCP	LMWH BD
		65	22	F			Y					Y					cSAH	Inter hemisperic SAH	SSS	None	LMWH BD
		66	43	F			Y				Y	Y					cSAH	Lt Sylvian Fissure Temporal Sulcus	SSS, Lt TS	OCP	LMWH BD
27	Kasuga K et al., 2006 [39]	67	39	M	Y				Y	Y							cSAH	Lt Sylvian Fissure	SSS	Greaves	LMWH BD
28	Ohta H et al., 1998 [40]	68	54	F								Y					cSAH	Rt Sylvian Fissure SAH	SSS	Aplastic Anaemia	LMWH BD

29	Arevalo-Lorido JC and Carretero-Gomez J 2015 [16]	69	70	M	Y		Y	Y									cSAH	Rt Parietal SAH	Rt TS	Hyperhomo-cysteinemia	LMWH BD
30	Panda S et al., 2010 [3]	70	32	M	Y	Y										Y	cSAH	Left Sylvian, Ant Hemispheric fissure, B/L FP convexities	SSS Rt TS	None	LMWH BD
		71	50	M	Y	Y			Y			Y					cSAH	Right Sylvian, posterior interhemispheric	SSS Left TS, Straight Sinus	Dehydration	LMWH BD
		72	27	F	Y				Y								cSAH	right frontal convexity, Rt cigulate, Ant Hemispheric fissure	SSS B/l TS, Straight Sinus	OCP	LMWH BD
		73	33	M	Y	Y										Y	cSAH	B/L FP convexities, interhemispheric fissure	SSS	None	LMWH BD
		74	32	F	Y	Y			Y			Y					cSAH	Right Sylvian, Empty Delta sign	Not Done	Postpartum state	LMWH BD
		75	25	M	Y	Y			Y								cSAH	Left frontal convexity	SSS	APLA Syndrome	LMWH BD
		76	38	M	Y	Y			Y								cSAH	Right Frontal Convexity, Left frontal Haemorrhagic infarct	SSS, B/L TS	None	LMWH BD
		77	30	M	Y	Y											cSAH	Right Sylvian fissure	SSS	Hyperhomocy-steinemia	LMWH BD
		78	38	M	Y	Y			Y						Y	Y	cSAH	Right Sylvian	SSS, B/l TS, Sigmoid Sinus	None	LMWH BD
31	Zare M and Mirabdolbaghi P, 2005 [41]	79	45	F	Y	Y	Y		Y		Y				Y	Y	cSAH	Right Sylvian fissure, posterior temporal sulci	Left Sigmoid, TS, SSS	Low Protein C, S, Migraine	LMWH BD
32	Sahin N et al., 2014 [42]	80	48	F	Y		Y							Y			cSAH	B/lFronto-Parietal Convexity	SSS	None	LMWH BD
33	Kato Y et al., 2010 [43]	81	52	F	Y	Y		y									cSAH	Rt Temporal Sulci + B/L cerebellar sulci	SSS+ Straight + TS + Right Sigmoid	None	LMWH BD
34	Sangra MS et al., 2008 [44]	82	25	M	Y		Y	Y									Perimese-ncephalic	Perimesencephalic Haemorrhage	B/L Jugular	None	LMWH BD
35	Abbas A et al., 2018 [45]	83	58	M		Y		Y	Y			Y					cSAH	Left Temporo-Parietal	Straight Sinus + SSS	None	LMWH BD
36	Amer RR, Bakhsh EA 2018 [46]	84	44	F	Y		Y		Y			Y				Y	Perimese-ncephalic	Prepontine Cistern	IJV + TS	None	LMWH BD
37	Lee J et al., 2009 [47]	85	72	F	Y		Y	Y	Y								Perimesen-cephalic	Rt Thalamic	Straight + Right TS + Sigmoid	None	LMWH BD

38	Sayadnasiri M et al., 2012 [48]	86	42	F							Perimesen-cephalic			TS +SS		LMWH BD
		87	36	M							cSAH			SSS + T + SS		LMWH BD
39	Renou P et al., 2012 [49]	88	74	F							cSAH					LMWH BD
		89	36	F							cSAH					LMWH BD
40	Hegazi MO et al., 2010 [17]	90	38	F	Y			Y			cSAH	Y		SSS + TS	None	LMWH BD
		91	70	M	Y			Y			cSAH			Rt TS		LMWH BD
41	Tang PH et al., 2008 [50]	92	23	M		Y					cSAH	Y		SSS		LMWH BD
		93	30	M		Y					cSAH		SSS	SSS	None	LMWH BD
		94	48	M	Y						cSAH	Y	SSS	SSS	None	LMWH BD
		95	37	F		Y					cSAH			SSS + T	None	LMWH BD

[Table/Fig-6]: Shows cases reviewed [3-5,13,15-50].
LMWH: Low molecular weight heparin; OD: Once daily; OCP: Oral contraceptive pills; IBD: Inflammatory bowel disease; APLA: Antiphospholipid; CVT: Cerebral venous thrombosis; DVT: Deep venous thrombosis; SSS: Superior sagittal sinus; cSAH: Cortical subarachnoid haemorrhage

Spontaneous SAH is caused by ruptured cerebral aneurysm in 85% of patients and non aneurysmal peri mesencephalic haemorrhage accounts for 10% of cases [1]. In these cases, SAH mainly involves the skull base [2]. In the present case review, the most common location of SAH was cortical, involving convexity of cerebral hemisphere in 87 patients (91.58%), while 5 patients (5.26) had peri mesencephalic SAH. Typical aneurysmal distribution was also reported in 3 cases (3.16%) [Table/Fig-4]. Therefore, when SAH is localised at the cerebral convexity, CVT should be ruled out as underlying cause.

In majority of these reported cases, most commonly, superior sagittal sinus was thrombosed with variable involvement of other major venous sinuses (84/95) while seven cases had only isolated cortical vein thrombosis. There were four cases in which venography was not available [Table/Fig-4]. In patients presenting with non aneurysmal SAH, the diagnosis of CVT is relatively straight when there was a major sinus thrombosis. By contrast, the diagnosis was challenging when there is an isolated cortical vein thrombosis [4,15]. Even in the absence of SAH, CVT is difficult to diagnose, because of the variable number and location [1]. The largest veins are detectable on MRV or CT venography [6]. When there is a SAH localised at the convexity T2 MR sequences are crucial. It shows thrombosed cortical vein as an hypointense tubular structure while SAH appears as a slight hemosiderin deposit [5]. So, review of neuroimaging findings of these cases shows that SAH localised at the cortical convexity area should raise the possibility of thrombosis of the cerebral venous sinuses or cortical vein and should be evaluated. Thus, the localisation of cortical SAH appears to be a good indicator of the involved venous structure. The diagnosis of CVT in a patient with SAH is crucial, because it needs to be treated with heparin, but it is contraindicated in all other causes of SAH [11]. Most of the reviewed cases were treated with either conventional heparin or low molecular weight heparin followed by oral anticoagulant. One case report by Arevalo-Lorido JC and Carretero-Gomez J mentioned that patient was initially treated with once daily dose of LMWH and after 3 days, the dose was increased to two times daily [16]. No other case report mentioned the dose of heparin used. Hegazi MO et al., reported a case of SAH secondary to CVT where they started anticoagulation after 3-4 days on evidence of reduced SAH component on repeat neuroimaging [17]. But none of these cases included in the present review showed worsening of SAH after starting anticoagulant therapy. [Table/Fig-6] shows a total of 95 cases which were reviewed for their available demographic data, clinical and neuroimaging findings [3-5,13,15-50].

Limitation(s)

The review had significant limitations because of variability of information reported in each case, and lack of clinical data in some papers or insufficient neuroimaging data in others. There is also a chance of selection bias, but this was not a statistical problem as the present data collected yields information on clinic-radiological features, and it is not intended to compare outcome morbidity or mortality.

CONCLUSION(S)

In patients with non aneurysmal SAH, MRI/ CT venography should be done to rule out cerebral venous sinus thrombosis. Presence of cortical pattern of SAH and risk factors for CVT should raise suspicion of underlying venous thrombosis. Diagnosing CVT in these cases is therapeutically important in view of starting anticoagulant treatment.

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