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<u>*Original Research*</u>

Prevalence of subarachnoid hemorrhage among patients with cranial venous sinus thrombosis in the presence and absence of venous infarcts

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ABSTRACT

Introduction: In patients with cranial venous sinus thrombosis (CVST), occurrence of subarachnoid hemorrhage (SAH) in association with hemorrhagic venous infarcts (HVI) is a well-described phenomenon. However, presence of SAH in patients with CVST in the absence of a HVI is exceedingly rare.

Methods: We retrospectively reviewed charts and scans of all patients who had CVST confirmed by magnetic resonance venography (MRV) at our hospital between September, 2004 and May, 2015. Presence of SAH was ascertained on FLAIR, SWI and/or unenhanced CT scans by a single experienced neuroradiologist. Statistical analysis was performed using Statistical Package for Social Sciences (SPSS) version 20. Differences in proportion of HVI among patients with SAH *vs.* those without SAH were compared using Chi-square (χ^2) test. A *P* value of less than 0.05 was considered significant.

Results: A total of 138 patients who had CVST were included in the study. Seventy three (52.9%) were female and median age of subjects was 35 (inter-quartile range [IQR]: 22–47) years. Venous infarcts and HVI were noted in 20/138 (14.5%) and 62/138 (44.9%) cases respectively. SAH was present in 15/138 (10.9%) cases and, in 3 cases, SAH occurred in the absence of a venous infarct. Hemorrhagic venous infarcts were more prevalent (*P* = 0.021) among patients with SAH (11/15) than those without SAH (51/123).

Conclusion: In patients with CVST, SAH can occur even in the absence of a HVI. Recognition

of CVST as the underlying cause of SAH is important to avoid misdiagnosis and inappropriate management.

Word count: 246

Key words: dural sinus thrombosis, cranial sinus thrombosis, subarachnoid

hemorrhage, cerebral infarction, stroke

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Introduction

Cranial venous sinus thrombosis (CVST) accounts for 1% of all cases of stroke.[1] While this disease can affect patients of any age, young and middle-aged women are most commonly affected.[2] Most patients are found to have a pro-thrombotic state (such as an inherited thrombophilia) or a risk factor for thrombosis (the most common being oral contraceptive pills).[3] Clinical manifestations are often varied and previous studies have reported headache, vomiting, altered mental status, visual disturbances, seizures or focal neurologic deficits as the notable clinical features.[4] These signs and symptoms result from impaired cranial venous drainage due to CVST, which leads to raised intracranial pressure and impaired reabsorption of cerebrospinal fluid into the superior sagittal sinus.[5] In some cases, CVST can lead to venous infarction of the cerebral parenchyma, which often develops hemorrhagic transformation.[6,7]

Non-traumatic subarachnoid hemorrhage (SAH) is secondary to rupture of an intracranial aneurysm in 85% of cases.[8] Sudden onset of a severe headache ("thunderclap headache") is the presenting clinical feature in most cases.[9] If not diagnosed and managed promptly, patients can rapidly progress to develop drowsiness, stupor and coma.[10] Aneurysmal SAH portends a worse prognosis and population-based studies have reported mortality rates to be as high as 45%.[11] Apart from aneurysmal rupture, other reported causes of non-traumatic SAH include arteriovenous malformations, intracranial vascular dissection, intracranial vasculitis, dural venous fistula, metastatic disease and bleeding diathesis.[12,13] CVST as a cause of SAH has also

been reported in the literature, although it usually occurs in conjunction with the presence of a hemorrhagic venous infarct (HVI).[14,15]

Occurrence of SAH in patients with CVST in the absence of venous infarcts (VI) has been rarely reported.[14] The importance of recognizing CVST as the underlying cause of SAH is that its management is radically different from that of aneurysmal SAH. CVST is a potentially treatable condition and requires prompt anti-coagulation to prevent long-term neurologic sequelae.[3,16] On the other hand, anti-coagulation in cases of aneurysmal SAH can have disastrous consequences.[8] From a theoretical standpoint, CVST can lead to the development of VI, which may undergo hemorrhagic transformation and progress to SAH.[17] However, occurrence of SAH in patients with CVST in the absence of VI remains an obscure entity.[18] In the present study, our aim was to determine the frequency of SAH in patients with diagnosed CVST, both in the presence and absence of HVI.

Methods

Our hospital is a 522-bedded tertiary care center located in the city of Karachi (Pakistan) with an estimated population of almost 27.5 million. A retrospective cross-sectional study was performed after obtaining exemption from formal approval by the institutional ethics review committee (ERC). We retrieved all magnetic resonance venography (MRV) reports from the institutional Radiology Information System (RIS) containing the words "dural sinus thrombosis", "cerebral venous sinus thrombosis", "cranial sinus thrombosis" or "cranial venous sinus thrombosis." Medical records for all patients with a diagnosis of CVST confirmed by MRV between September, 2004 and May, 2015 were retrieved. Patients with untraceable medical records or those who were not managed at our institution were excluded from the study. We also intended

to exclude patients with CVST who had an alternative diagnosis as the underlying cause of SAH (such as a ruptured aneurysm), although we did not encounter any such cases. Using a pre-designed, structured pro forma, each patient's chart was systematically reviewed and data relating to demographics, clinical features and subsequent work-up was collected. Personal identifiers or other identifiable information was not recorded. All scans were de-identified prior to re-interpretation for the purpose of this study.

All patients included in the study had undergone MRI and MRV scans on a 1.5 Tesla MRI scanner (MAGNETOM® Avanto; Siemens AG, Munich, Germany) with a dedicated head-coil. Gadolinium-based contrast (Magnevist®; Bayer AG, Leverkusen, Germany) in a dose of 0.5 ml/kg was used for performing contrast-enhanced MRV scans. MRV sequences were performed with the following parameters: TR / TE: 3.48 / 1.22, field of view: 20 cm × 20 cm, matrix: 320 × 128, bandwidth: 380 Hz/pixel and slice thickness: 1.1 mm. A consultant neuroradiologist with more than 5 years of experience was responsible for re-interpreting the scans and he was blinded to the actual report of the scans at the time of interpretation. Presence of VI, HVI or SAH was specifically noted on MRI scans. For the purpose of this study, SAH was diagnosed using two MRI sequences viz. fluid attenuated inversion recovery (FLAIR) and susceptibility weighted imaging (SWI) sequences. Hyperintense signals in the subarachnoid space on FLAIR sequences along with corresponding signal dropouts on SWI sequences were considered positive for SAH. CVST was diagnosed on contrast-enhanced MRV images as filling defects in the dural venous sinuses and/or cortical or deep veins provided such filling defects were not accounted for by arachnoid granulations or hypoplastic sinuses. Moreover, site and extent of CVST on each MRV scan was also recorded for each patient. If a patient had undergone head computed tomography (CT) during the course of hospitalization, these scans were also interpreted for the presence of

SAH and other associated findings. SAH on CT scans was defined as the presence of high attenuation streaks within the subarachnoid space. Cohen's kappa (κ) statistic was calculated as a measure of agreement between the findings reported in the official MRV report and the findings noted by the neuroradiologist re-interpreting scans (for the purpose of this study).

Statistical Package for Social Sciences (SPSS) version 20.0 was used for performing statistical analysis. Frequencies were calculated for qualitative variables, while median (inter-quartile range [IQR]) were computed for quantitative variables. For all proportions, 95% confidence intervals (CI) were also computed. Chi-Square (χ^2) or Fisher's exact test was used for comparison of proportions. Pearson product-moment correlation coefficient was computed to determine the correlation between SAH and VI or HVI. For all comparisons, a *P* value of less than 0.05 was considered statistically significant.

Results

Our initial search of the institutional RIS identified 144 patients with CVST on MRV. Of these, 4 were excluded as they were managed at another hospital and only underwent follow-up imaging at our center. Another 2 were excluded due to untraceable records and missing data. This left a total of 138 patients for inclusion in the final analysis. Among these, 73/138 (52.9% [95% CI: 44.4% to 61.4%]) were female and 65/138 (47.1% [95% CI: 38.6% to 55.6%]) were male. The median age of study subjects was 35 (IQR: 22–47) years. The most common clinical presentations were headache (n=47/138, 34.1% [95% CI: 26.0% to 42.2%]), seizure (n=43/138, 31.2% [95% CI: 23.3% to 39.1%]) and motor weakness (n=29/138, 21% [95% CI: 14.1% to 27.9%]) as given in Table 1. The most common underlying cause of CVST was malignancy (n=21/138, 15.2% [95% CI: 9.1% to 21.3%]) followed by meningoencephalitis (n=17/138,

12.3% [95% CI: 6.7% to 17.9%]) and post-partum status (n=16/138, 11.6% [95% CI: 6.1% to

17.1%]). In another 52/138 (37.7% [95% CI: 29.4% to 46.0%]) patients, no underlying cause

could be identified (Table 1). Superior sagittal sinus (n=86/138, 62.3% [95% CI: 54.0% to

70.6%]) was the most commonly thrombosed dural sinus followed by transverse (n=82/138,

59.4% [95% CI: 51.0% to 67.8%]) and sigmoid (n=62/138, 44.9% [95% CI: 36.4% to 53.4%])

sinuses. Extension of sigmoid sinus thrombosis into the internal jugular vein was noted in 24/138

(17.4% [95% CI: 10.9% to 23.9%]) cases. Cortical veins and deep veins were thrombosed in

12/138 (8.7% [95% CI: 3.9% to 13.5%]) and 9/138 (6.5% [95% CI: 2.3% to 10.7%]) patients

respectively (Table 1).

VI, HVI and SAH were noted in 20/138 (14.5% [95% CI: 8.5% to 20.5%]), 62/138 (44.9% [95%

CI: 36.4% to 53.4%]) and 15/138 (10.9% [95% CI: 5.6% to 16.2%]) patients respectively. Figure

1, Figure 2 and Figure 3 demonstrate examples of VI, HVI and SAH. None of these findings

were present in the other 56/138 (40.6% [95% CI: 32.2% to 49.0%]) patients. There was strong

agreement between the findings reported in the official MRV report and findings noted by the

neuroradiologist re-interpreting scans for the purpose of this study (κ=0.985 for VI, κ=1.000 for

HVI and κ=0.961 for SAH respectively). Reports of CT head were available for 93/138 (67.4%)

patients in total. Of the 15/138 (10.9% [95% CI: 5.6% to 16.2%]) patients with SAH noted on

MRI, all had undergone CT head and presence of SAH on CT was confirmed in 14/15 (93.3%

[95% CI: 89.0% to 97.6%]) patients. SAH was not noted in any of the remaining 79/138 (57.2%

[95% CI: 48.8% to 65.6%]) CT scans. Pearson product-moment coefficient revealed a

statistically significant, positive correlation between SAH and HVI ($P = 0.010$, $r = 0.198$). The

proportion of patients with SAH who had HVI (11/15, 73.3% [95% CI: 65.8% to 80.8%]) was

significantly higher ($P = 0.021$) than the proportion of patients without SAH who had HVI

(51/123, 41.5% [95% CI: 33.1% to 49.9%]) as given in Table 2. This is depicted more clearly

with a Venn diagram in Figure 4. Among patients with SAH (15/138), 3/15 (20%, [95% CI:

13.2% to 26.8%]) had no evidence of a VI or HVI. In all these cases, SAH was limited to the

cortical convexity with sparing of the basal cisterns.

Discussion

In this retrospective cross-sectional study, we systematically reviewed scans of patients with

CVST for evidence of SAH and found that SAH was noted in 10.9% of patients. Furthermore,

we noted that 44.9% of patients had a HVI and another 14.5% had VI without evidence of

hemorrhagic transformation. These results are of considerable interest and may suggest that SAH

in conjunction with CVST has been under-estimated in previous reports published in the

literature.[14,17,18] In a report from India, Panda and colleagues retrospectively reviewed records of

233 patients who were diagnosed with CVST at their center and found radiological evidence of

SAH in 10 cases (i.e. 4.3%).[19] On the other hand, we found a higher frequency of SAH in our

study (i.e. 10.9%). One reason for this observation may be that in our study, all scans were

reviewed and re-interpreted systematically to look for evidence of SAH. Subtle SAH in cases of

CVST may often be overlooked.[17,18]

Most previously published literature on SAH in conjunction with CVST is limited to a few small

case series. Kato *et al.* from Japan described the case of a 52-year-old lady who presented with

severe occipital headache and was found to have CVST involving the superior sagittal, straight,

transverse and bilateral sigmoid sinuses.[14] Her CT showed evidence of SAH involving bilateral

cerebellar sulci and right temporal sulcus. In another report by Jaiser *et al.* from the United

Kingdom, a 53-year-old lady presented with occipital headache and was found to have evidence

of small left frontal SAH on unenhanced CT.[18] CT angiography and MRV confirmed a diagnosis

of CVST (in the absence of a VI) and excluded intracranial aneurysm. In our study, headache

was the presenting feature in 47 (34.1%) cases and malignancy (n=21, 15.2%) was the most

common predisposing factor for thrombosis. No obvious pro-thrombotic state could be identified

in 52 (37.7%) cases in our study.

A number of hypotheses have been proposed to explain the occurrence of SAH in conjunction

with CVST. The most plausible explanation is that SAH in the setting of CVST can occur due to

hemorrhagic transformation of a venous cerebral infarction.[20] From the infarcted parenchyma,

red blood cells may leak into perivascular Virchow-Robin spaces and pool up in the

subarachnoid spaces between cortical gyri. However, in our study, 3 patients with SAH did not

have any evidence of VI on radiologic imaging. Likewise, in the series of Panda *et al.*, 9 patients

with CVST and SAH had no radiologic evidence of VI.[19] One reason may be that evolution of a

VI requires time and may not be evident on radiologic imaging early in the process. A better

explanation may be that SAH in conjunction with CVST is due to an alternative

pathophysiologic mechanism. Kato and colleagues suggested that CVST can lead to

development of venous hypertension, which may lead to rupture of fragile cortical veins.[14] As

cortical veins are valveless and lack smooth muscle in their tunica media, venous hypertension

can lead to rupture of these veins with leakage of blood into the subarachnoid space at points

where these veins enter into the dural venous sinuses.[21,22]

Published literature on CVST and SAH suggests that the distribution of SAH may provide a clue

to the underlying etiology of SAH. In a report of 4 cases, Oppenheim *et al.* reported that SAH

associated with CVST involved only the cortical sulci and spared the basal cisterns.[19] Chang and

Friedman described 3 cases of isolated cortical vein thrombosis involving the vein of Trolard and

all patients had SAH limited to the adjacent cortical sulci with sparing of basal cisterns.[23] This observation was also noted in the case series of Panda *et al.*, who suggested that localized convexity SAH sparing the skull base may be an early sign of CVST.[19] In our study, we noted that patients with thrombosis of superior sagittal, transverse and sigmoid sinuses had VI or HVI in the cerebral or cerebellar hemispheres. On the other hand, thrombosis of straight sinus or vein of Galen caused HVI in basal ganglia and thalamus. Among patients with SAH who had VI or HVI, SAH was limited to the vicinity of HVI. In patients with HVI of the basal ganglia or thalamus, SAH could involve the skull base and extend beyond the sulci of the cerebellar convexities. However, consistent with previous observations, SAH in cases without VI spared the basal cisterns and was limited to the cortical convexity. In all such cases, thrombosis involved the deep cortical veins, which may suggest that SAH was caused by pressure changes in the fragile microvasculature of the central nervous system.

Differentiation between SAH caused by CVST and SAH due to other causes is of considerable clinical importance. CVST is a potentially treatable condition, although delays in diagnosis and management have been shown to lead to a poor neurologic outcome.[24] Prompt anti-coagulation with either unfractionated heparin or low molecular weight heparin is recommended to prevent further clot propagation.[25] Recent studies have also explored the role of thrombolysis and endovascular therapies (such as thrombectomy) to reduce clot burden and hasten recovery.[26,27] Notable here is the fact that presence of HVI or SAH in the setting of CVST is not a contraindication to anti-coagulation as hemorrhage in such scenarios is caused by venous hypertension and is expected to improve with anti-coagulation.[28] On the other hand, anti-coagulation is detrimental in cases of SAH due to other causes (such as aneurysmal bleeding)

and may lead to a fatal outcome.[8] Therefore, the association between SAH and CVST needs to be recognized by clinicians and radiologists alike.

The limitations of this study merit attention as well. Firstly, this study was a retrospective cross-sectional review of patients at a single tertiary care center and this may have over-estimated the prevalence of SAH in patients with CVST. Secondly, our study included only Asian patients and we cannot generalize these findings to patients of other ethnicities. Thirdly, conventional cerebral angiography was not performed in all patients, although most patients had undergone either CT angiography or magnetic resonance arteriography to exclude an aneurysmal SAH. Additionally, all patients included in our study had undergone contrast-enhanced MRV. In clinical practice, a small (but significant) number of patients have contraindications to gadolinium-based contrast media and can only undergo MRV without contrast. Whether the results of our study can be generalized to those patients remains uncertain. Lastly, a single neuroradiologist with relevant expertise in the field was responsible for re-interpreting scans for all patients included in this study. This precluded an accurate assessment of the inter-rater reliability of these findings.

Conclusion

SAH occurs frequently in patients with CVST, especially in patients who develop VI or HVI. However, SAH may occur in conjunction with CVST even in the absence of a VI. Presence of a cerebral convexity SAH with sparing of the basal cisterns may be a useful differentiating sign in such cases.

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Conflict of interests

The authors have no conflict of interests to disclose.

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Ethical considerations

This retrospective study was granted exemption from formal approval by the institutional ethics review committee.

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Legend to the figures

Figure 1: (a) Axial and (b) maximum intensity projection post-contrast images of magnetic resonance venography demonstrate a filling defect in the straight sinus consistent with thrombosis. (c) Coronal fluid attenuated inversion recovery (FLAIR) images show diffuse symmetrical hyperintense signals in bilateral basal ganglia and thalamic regions. (d) Diffusion weighted imaging (DWI) and apparent diffusion coefficient (ADC) mapped axial images reveal diffusion restriction in the corresponding areas consistent with acute venous infarcts.

Figure 2: (a) Axial and (b) maximum intensity projection post-contrast images of magnetic resonance venography demonstrate a filling defect in the superior sagittal sinus consistent with thrombosis. (c) Coronal fluid attenuated inversion recovery (FLAIR) and (d) axial T2-weighted images show diffuse, heterogeneous, predominantly hyperintense signals in bilateral parietal lobes. (e) Susceptibility weighted images (SWI) show signal dropout in the corresponding areas consistent with bilateral hemorrhagic venous infarcts.

Figure 3: (a) Axial and (b) maximum intensity projection post-contrast images of magnetic resonance venography reveal a filling defect in the superior sagittal sinus consistent with dural sinus thrombosis. (c) Coronal fluid attenuated inversion recovery (FLAIR) images show subtle hyperintensities in the left parietal lobe sulci representing subarachnoid hemorrhage, which was confirmed on (d) susceptibility weighted imaging (SWI). (e) No subarachnoid hemorrhage was noted involving the basal cisterns.

Figure 4: A Venn diagram depicting the relative proportion of patients with venous infarcts (VI), hemorrhagic venous infarcts (HVI) and subarachnoid hemorrhage (SAH).

Tables

Table 1. Descriptive characteristics of patients* included in our study (n = 138)

Variables	Values (n [%])	95% CI
Sex		
Female	73/138 (52.9%)	44.4% to 61.4%
Male	65/138 (47.1%)	38.6% to 55.6%
Clinical presentation		
Headache	47/138 (34.1%)	26.0% to 42.2%
Seizure	43/138 (31.2%)	23.3% to 39.1%
Motor weakness	29/138 (21.0%)	14.1% to 27.9%
Vomiting	28/138 (20.3%)	13.5% to 27.1%
Altered consciousness	25/138 (18.1%)	11.5% to 24.7%
Fever	18/138 (13.1%)	7.4% to 18.8%
Decreased vision	10/138 (7.2%)	2.8% to 11.6%
Predisposing factor		
None	52/138 (37.7%)	29.4% to 46.0%
Malignancy	21/138 (15.2%)	9.1% to 21.3%
Infection	17/138 (12.3%)	6.7% to 17.9%
Post-partum	16/138 (11.6%)	6.1% to 17.1%
CNS or inner ear infection	13/138 (9.4%)	4.4% to 14.4%
Trauma	9/138 (6.5%)	2.3% to 10.7%
Auto-immune disease	4/138 (2.9%)	0.0% to 5.8%
Other	6/138 (4.3%)	0.8% to 7.8%
Sinuses thrombosed†		
Superior sagittal sinus	86/138 (62.3%)	54.0% to 70.6%
Transverse sinus	82/138 (59.4%)	51.0% to 67.8%
Sigmoid sinus	62/138 (44.9%)	36.4% to 53.4%
Internal jugular vein	24/138 (17.4%)	10.9% to 23.9%
Straight sinus	20/138 (14.5%)	8.5% to 20.5%
Cortical veins	12/138 (8.7%)	3.9% to 13.5%
Deep veins	9/138 (6.5%)	2.3% to 10.7%

CI = confidence interval; *CNS* = central nervous system.

* Median age of included patients was 35 years (interquartile range: 22 to 47 years)

† Some patients had more than one venous sinus thrombosed

Table 2. Frequency of subarachnoid hemorrhage, venous infarct and hemorrhagic venous infarct on magnetic resonance venography among study subjects (n = 138)

Type of infarct	SUBARACHNOID HEMORRHAGE*		*Total*
	Present	Absent	
VENOUS INFARCT	1	19	20
HEMORRHAGIC VENOUS INFARCT	11	51	62
NO INFARCT	3	53	56
Total	15	123	138

* Subarachnoid hemorrhage on magnetic resonance imaging was defined as the presence of hyperintense signals in the subarachnoid space on FLAIR (fluid attenuated inversion recovery) sequences along with corresponding signal dropouts on SWI (susceptibility weighted imaging) sequences