Neurology Asia 2025; 30(4) : 1035 – 1040

Clinical and etiological spectrum of nontraumatic convexal subarachnoid hemorrhage in a South Indian tertiary care centre

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Abstract

Background: Nontraumatic convexal subarachnoid hemorrhage (cSAH) is a distinct subtype of intracranial hemorrhage confined to the cortical sulci. Given its diverse etiologies, accurate diagnosis requires careful clinical and radiological evaluation. The present study aims to characterize the clinical profile, risk factors, neuroimaging features, and etiological spectrum of cSAH in a cohort of South Indian patients. *Methods:* We prospectively studied 17 consecutive patients with cSAH presenting to our tertiary care centre in Chennai, India, from 2023 to 2024. Data on demographics, clinical features, risk factors, neuroimaging, and etiologies were collected. Findings were compared with international cohorts. *Results:* The mean patient age was 46 years, and 47% were female. Headache occurred in 88%, seizures in 41%, and focal deficits in 35%. Parietal (71%) and frontal (65%) lobes were the most frequent hemorrhage sites; 35% had bilateral involvement. Cerebral venous thrombosis (CVT) was the leading cause (71%), followed by posterior reversible encephalopathy syndrome (12%), reversible cerebral vasoconstriction syndrome (6%), and arteriovenous malformation (6%). Compared with other published cohorts, our patients were younger, had a higher seizure incidence, and a predominance of CVT.
Conclusion: In South Indian patients, cSAH most often results from CVT, especially in younger individuals. Prompt MRI with MR venography is critical for early diagnosis and management.

Keywords: Subarachnoid hemorrhage, cerebral venous thrombosis, posterior reversible encephalopathy syndrome, reversible cerebral vasoconstriction syndrome

INTRODUCTION

Nontraumatic convexal subarachnoid hemorrhage (cSAH) is defined by isolated bleeding within the cortical sulci, without extension into the basal cisterns, ventricular system, or significant intraparenchymal involvement.[1] It accounts for around 6% of all SAH cases and is distinct from aneurysmal SAH.[2] The increasing recognition of cSAH is largely attributable to advancements in neuroimaging, particularly the widespread availability and sophistication of MRI sequences like Fluid-Attenuated Inversion Recovery (FLAIR) and Susceptibility-Weighted Imaging (SWI).

The etiologies of cSAH are remarkably diverse, encompassing conditions ranging from benign to potentially life-threatening. These include, but are not limited to, reversible cerebral vasoconstriction syndrome (RCVS)[3], cerebral amyloid angiopathy[4,5], cortical vein thrombosis[6], coagulopathy, anticoagulant use[7], drug abuse, rupture of small arteriovenous malformations (AVM)[8], and carotid disease.[9] Given this broad spectrum, a meticulous diagnostic workup is essential to identify the underlying cause and guide appropriate management strategies.

cSAH presents with a range of symptoms, depending on its cause and the patient's age. Common symptoms include headache, impaired level of consciousness, seizures, confusion, transient focal neurological episodes, persistent focal neurological deficits, visual abnormalities, nausea, and vomiting.[1]

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Date of Submission: 18 August 2025; Date of Acceptance: 29 August 2025
https://doi.org/10.54029/2025jhx

While numerous studies have enhanced our understanding of the clinical presentation, etiological profile, and prognosis of cSAH, regional and demographic variations in its underlying causes are increasingly recognized. This study aims to comprehensively characterize the clinical and radiological features, risk factors, and etiological spectrum of nontraumatic cSAH in our patient cohort, representing one of the first Indian cohort studies on this entity, as existing literature from India is limited to isolated case reports.

METHODS

This prospective, single-center observational study enrolled 17 consecutive patients diagnosed with nontraumatic cSAH who presented to the Institute of Neurology, Madras Medical College, which is a tertiary care referral centre in South India, between 2023 and 2024. Inclusion criteria mandated clear radiological evidence of subarachnoid hemorrhage strictly localized to the cortical sulci, with exclusion of any history of head trauma, aneurysm rupture, or primary intraparenchymal hemorrhage.

Data collection

For each eligible patient, comprehensive data were collected. This included:

Demographic characteristics: Age at presentation, sex.

Clinical presentation: Nature of headache (e.g., thunderclap), presence of seizures (generalized tonic-clonic or focal), focal neurological deficits (e.g., motor or sensory deficits), altered sensorium, and other neurological symptoms.

Identified risk factors: Medical comorbidities (e.g., diabetes mellitus, hypertension, dyslipidemia, thyroid dysfunction, systemic lupus erythematosus), lifestyle factors (e.g., alcohol use, smoking), and specific physiological states (e.g., postpartum).

Neuroimaging findings: Findings from non-contrast Computed Tomography (CT), Magnetic Resonance Imaging (MRI) sequences (including FLAIR, SWI, T1, T2), Magnetic Resonance Angiography (MRA), and Magnetic Resonance Venography (MRV). Digital Subtraction Angiography (DSA) results were also recorded, if performed.

cSAH localization: Detailed description of the affected cortical sulci (e.g., frontal, parietal, occipital, temporal, unilateral/bilateral).

Final diagnosis: The definitive etiology of cSAH as determined based on integrated clinical and radiological findings.

Outcome: Status at discharge and mortality.

Statistical analysis

IBM SPSS Statistics was used for statistical analysis. The qualitative variables were displayed as frequencies and percentages, while the quantitative data were represented as means and standard deviations. The collected data, particularly regarding demographics, clinical features, and etiological spectrum, were compared with findings from several well-established international cohorts published in the literature.

Ethics

All patients gave written informed consent before participating in the study. The ethics committee of the institute approved the study protocol.

RESULTS

Demographic profile and associated risk factors

The study cohort comprised 17 patients. The mean age at presentation was 46 years (range: 23-76 years), reflecting a relatively younger patient population. Females constituted 47% (n=8) of the cohort. A spectrum of risk factors was identified among the patients: alcohol use was present in 47% (n=8), diabetes mellitus in 35% (n=6), hypertension in 24% (n=4), and smoking in 18% (n=3). Notably, postpartum status was identified as a significant risk factor in 24% (n=4) of the female patients.

Other less common but relevant risk factors included dyslipidemia, hypothyroid states, deep vein thrombosis, hyperhomocysteinemia, vitamin B12 deficiency, systemic lupus erythematosus with lupus nephritis, and gestational diabetes. The detailed demographic and clinical data of this cohort including their medical history are shown in Table 1.

Clinical Presentation

Headache was the cardinal symptom, reported by 88% (n=15) of patients. Of these, 35% (n=6) specifically experienced a thunderclap headache.

Table 1: Demographic and medical history of 17 patients with convexal subarachnoid haemorrhage

Variable	No. (%)
Age, years	46±14(23–76)*
Female	8 (47)
Medical history	
Alcohol	8 (47)
Diabetes mellitus	6 (35)
Postpartum	4 (24)
Hypertension	4 (24)
Dyslipidemia	4 (24)
Smoking	3 (18)
Hypothyroid	3 (18)
Deep vein thrombosis	1 (6)
Hyperhomocysteinemia	1 (6)
Vitamin B12 deficiency	1 (6)
Systemic lupus erythematosus	1 (6)

SD indicates standard deviation.
Values indicate no. (%), except for *mean ± 1 SD (range)

Seizures, encompassing both generalized tonic-clonic and focal types, were a prominent feature, affecting 41% (n=7) of the cohort. Focal sensory and/or motor deficits were present in 35% (n=6) of cases. Altered sensorium was observed in 29% (n=5) of patients, and visual symptoms were reported by 12% (n=2). Other less frequent presentations included transient ischemic attack-like episodes and diplopia.

Neuroimaging findings

All patients underwent MRI, while 2 patients had an initially normal non-contrast CT scan, highlighting the superior sensitivity of MRI for subtle cSAH detection. The cSAH was predominantly localized to the parietal lobes (71%, n=12) and frontal lobes (65%, n=11). Other affected regions included the occipital lobe (35%, n=6) and temporal lobe (12%, n=2). Bilateral hemorrhage was a common finding, observed in 35% (n=6) of the cases. Imaging findings specifically identified cerebral venous sinus or cortical vein thrombosis in 12 cases, an arteriovenous malformation in one case, and features suggestive of posterior reversible encephalopathy syndrome (PRES) or RCVS in others, guiding the final etiological diagnoses. The Figure shows representative images of cSAH.

Etiological spectrum

Cerebral venous thrombosis (CVT) was the leading identified etiology, accounting for 71% (n=12) of the cSAH cases, including two instances of postpartum CVT. PRES was diagnosed in 12% (n=2) of patients, one of which was postpartum-related and another associated with systemic lupus erythematosus. RCVS was identified in 6% (n=1) of cases (postpartum RCVS). An AVM was the underlying cause in 6% (n=1) of patients. The etiology remained indeterminate in 6% (n=1) of cases despite extensive workup.

Outcomes

One patient (6%) experienced mortality within the cohort. This fatality was associated with a diagnosis of AVM. The majority of patients experienced favourable neurological outcomes, often with successful resolution of symptoms

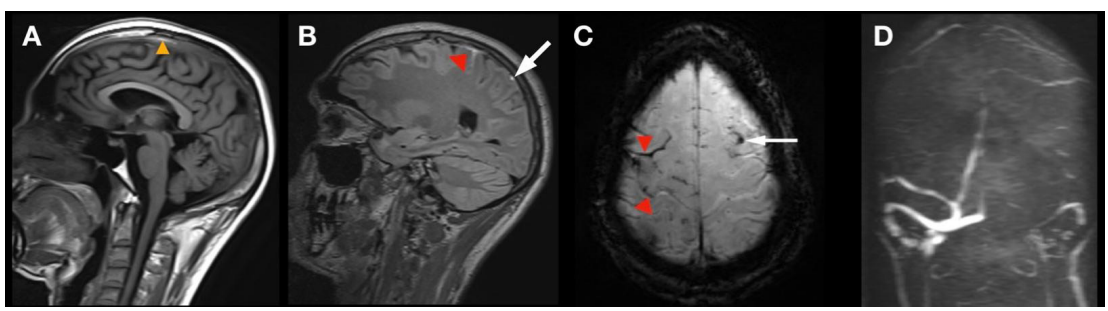

Figure 1. Representative images of cSAH. MRI Brain of a 36-year-old man diagnosed with CVT. A: T1W image showing hyperintensity in superior sagittal sinus (yellow arrowhead). B, C: T2 FLAIR and SWI images showing fronto-parietal cortical surface hyperintensity extending into the sulcus with gradient blooming suggestive of cSAH (red arrowheads), dot-like hyperintensity with gradient blooming suggestive of thrombosed cortical veins (white arrow). D: MRV showing absence of flow related hyperintensity in superior sagittal sinus, left transverse sinus and cortical veins.

Table 2: Clinical characteristics, hemorrhage location, etiology and mortality of cSAH in our cohort vs. published series

Variable	Our cohort	Forman *et al.*[10]	Chertcoff *et al.*[17]	Kumar *et al.*[11]	Geraldes *et al.*[12]	Zhao *et al.*[13]
No. of subjects	17	94	20	29	15	14
Age (years)*	46	58	53	58	64	62
Presenting symptom						
Headache	15 (88%)	50 (53%)	13 (65%)	18 (62%)	4 (27%)	8 (57%)
Thunderclap headache	6 (35%)	NA	5 (25%)	10 (34%)	NA	NA
Sensory and/or motor symptoms	6 (35%)	30 (32%)	10 (50%)	8 (27%)	10 (66%)	8 (57%)
Seizures	7 (41%)	16 (17%)	7 (35%)	0 (0%)	5 (33%)	1 (7%)
cSAH location						
Frontal	11 (65%)	NA	15 (75%)	15 (51%)	10 (67%)	9 (64%)
Parietal	12 (71%)	NA	6 (30%)	6 (20%)	3 (20%)	10 (71%)
Occipital	6 (35%)	NA	6 (30%)	1 (3%)	2 (13%)	0 (0%)
Temporal	2 (12%)	NA	3 (15%)	1 (3%)	2 (13%)	1 (7%)
Bilateral	6 (35%)	NA	10 (50%)	5 (18%)	1 (7%)	2 (14%)
Diagnosis						
CVT	12 (71%)	10 (11%)	4 (20%)	0 (0%)	1 (7%)	1 (7%)
PRES	2 (12%)	16 (17%)†	2 (10%)	1 (3%)	0 (0%)	0 (0%)
RCVS	1 (6%)	17 (18%)†	4 (20%)	11 (37%)	2 (13%)	0 (0%)
CAA	0 (0%)	15 (16%)	4 (20%)	10 (35%)	2 (13%)	1 (7%)
Large vessel stenosis	0 (0%)	7 (7%)	0 (0%)	0 (0%)	5 (33%)	7 (50%)
Others	1 (6%)	33 (35%)	6 (30%)	7 (24%)	5 (33%)	5 (36%)
Mortality	1 (6%)	7 (7%)	2 (10%)	3 (10%)	0 (0%)	0 (0%)

*values indicate mean. †five overlap RCVS/PRES patients. NA: not available, cSAH: convexal subarachnoid hemorrhage, CVT: cerebral venous thrombosis, PRES: posterior reversible encephalopathy syndrome RCVS: reversible cerebral vasoconstriction syndrome, CAA: cerebral amyloid angiopathy.

following appropriate treatment, such as anticoagulation for CVT or supportive care for PRES/RCVS.

DISCUSSION

Our study provides valuable insights into the clinical and etiological profile of nontraumatic convexal subarachnoid hemorrhage (cSAH) within our patient population, highlighting several key differences compared to established international cohorts, as summarized in Table 2.

Younger age and predominance of cerebral venous thrombosis

The mean age of our cohort was 46 years, which is notably younger than that reported in several other series.[10-15] This demographic difference likely contributes to the strikingly high prevalence of CVT (71%) as the underlying etiology in our study. CVT is generally more common in younger adults and has a recognized association with female sex and prothrombotic states.[16] The significant proportion of postpartum-associated cSAH cases in our cohort further supports this, as the puerperium is a well-established hypercoagulable state predisposing to CVT. In contrast, cohorts with older mean ages, such as those from Western populations, often report a higher incidence of reversible cerebral vasoconstriction syndrome and cerebral amyloid angiopathy (CAA) as leading causes of cSAH.[10,11,14]

Risk factor profile

The risk factor profile in our Indian cohort shows a distinct pattern of prothrombotic conditions and high rates of chronic alcohol use, differing from cohorts reported elsewhere.[10-13] While alcohol use is also noted in few western studies, it is

often linked to RCVS or multifactorial triggers in older adults.[14,15] In our series, it was frequently associated with CVT and cortical venous thrombosis, suggesting a role in venous endothelial injury or dehydration-related thrombosis. In contrast to Western cohorts, where hypertension, dyslipidemia, and smoking contribute to CAA or ICA stenosis, our patients more often had postpartum CVT and secondary risk factors like vitamin B12 deficiency, hyperhomocysteinemia, and nutritional anemia.[10,12] In the Chinese cohort, traditional vascular risks such as hypertension and diabetes were prominent, reflecting high rates of intracranial atherosclerosis.[13]

High incidence of seizures

Our cohort exhibited a remarkably high incidence of seizures, considerably greater than that reported in previous studies.[11,13,14] This elevated seizure rate is highly congruent with the high burden of CVT in our series. CVT and associated venous congestion are known epileptogenic factors, and seizures are a frequently reported complication of CVT, often presenting as focal motor seizures that can secondarily generalize.[16] The specific localization of cSAH predominantly in the frontal and parietal lobes in our patients further supports this, as these regions have a lower seizure threshold. Headache was the most common presentation in our cohort, consistent with the previous studies.[10,11,14]

Distinct etiological spectrum

The etiological profile in our study, overwhelmingly dominated by CVT, differs from most of the international cohorts.[10-14] For instance, the Argentinian cohort by Chertcoff *et al*. reported a more balanced distribution, with CVT, RCVS, and CAA each accounting for 20% of cases.[17] The absence of CAA in our cohort is likely a reflection of our younger patient demographic. Similarly, RCVS was rare in our series compared to some Western cohorts.[11,14] This discrepancy highlights potential regional and ethnic variations in disease prevalence.

Imaging patterns and bilateral involvement

Consistent with previously published literature, our findings confirm that frontal and parietal lobes are the most commonly affected sites of cSAH.[11,13,14] The high incidence of bilateral hemorrhage in our cohort is comparable to a few of the previous studies.[14,17] Bilateral involvement can often be indicative of widespread or systemic etiologies, such as CVT affecting multiple venous territories or PRES.

Mortality and prognosis

Our observed mortality rate of 6% is lower than reported by Beitzke *et al*.[16] but falls within the range of other series.[10,12,13,14] The single fatality in our cohort was attributed to underlying ruptured AVM rather than the cSAH itself. This supports the general consensus that mortality in cSAH is predominantly determined by the underlying etiology and its systemic implications rather than the localized hemorrhage per se. The majority of our patients achieved favorable outcomes, underscoring the importance of timely and accurate etiological diagnosis to initiate specific treatments like anticoagulation for CVT.

Comparison with Chinese and Western cohorts

The comparative analysis further underscores the impact of ethnic and geographical factors on cSAH etiology. In a Chinese cohort by Zhao *et al*.[13], internal carotid artery (ICA) stenosis or occlusion was the leading cause of cSAH, a finding distinct from our cohort where CVT predominated. The Chinese cohort had a mean age of 62 years and a male predominance, with a high prevalence of vascular risk factors such as hypertension and diabetes mellitus. This contrasts sharply with our younger cohort (mean age: 46 years) and the higher prevalence of postpartum states and prothrombotic conditions. Furthermore, Western studies tend to report a higher frequency of etiologies like CAA in older adults and RCVS in younger adults.[10,11,14] The absence of CAA and rarity of RCVS in our younger cohort further exemplifies this variability.

Ethnic and geographic considerations

Our findings strongly advocate for an ethnically and regionally informed approach to evaluating cSAH. While Western populations often present with cSAH secondary to age-related pathologies like CAA, younger South Asian populations appear to have a higher burden of venous thrombosis and postpartum-related conditions.

These differences may be influenced by genetic factors affecting coagulation, varying access to advanced neuroimaging techniques, and differing clinical practices. A global understanding of cSAH must, therefore, integrate these diverse demographic, ethnic, and geographic contexts to optimize diagnostic pathways and therapeutic strategies.

This study is subject to several limitations. Its single-center design may limit the external validity and generalizability of the findings. The relatively small sample size (17 patients) further restricts the statistical power and may not fully capture the heterogeneity of etiologies and clinical presentations. The lack of standardized, long-term follow-up data for all patients also limits a comprehensive assessment of long-term functional outcomes.

In conclusion, CVT emerged as the leading cause of nontraumatic cSAH in this South Indian cohort, in contrast to Western and Chinese cohorts. These geographic and demographic differences highlight the need for region-specific diagnostic algorithms. Prompt neuroimaging is essential for early diagnosis and etiological workup.

DISCLOSURE

Financial support: None

Conflicts of interest: None

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