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Trajectory and Risk Factors of Acute Spontaneous Subdural Hematoma Complicating Course of Hospitalization in Critically Ill Patients: A Five-Year Case Series Study

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Acute spontaneous subdural hematoma SDH is an uncommon but critical condition in critically ill patients. While chronic SDH is more frequent, the trajectory and risk factors of acute spontaneous SDH in such patients have been underreported.

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Aim: This study aims to analyze the trajectory and identify the risk factors for acute spontaneous SDH among critically ill patients.
Objective: The objectives are to assess the risk factors associated with acute spontaneous SDH, understand its progression during hospital stays, and evaluate the impact of timely diagnosis and management on patient outcomes.
Materials and Methods: Over a five-year period from April 2019 to April 2024, we conducted a retrospective analysis of 9 critically ill patients who developed acute spontaneous SDH during their course in the hospital. Data on demographics, co-morbidities, risk factors, hospital course, and outcomes, including a 90-day follow-up, were collected and analyzed.
Results: The study identified several key risk factors prevalent among the patients with acute spontaneous SDH of which age, use of antiplatelet, previous cardiac intervention were major contributors, while hypertension, chronic alcohol use, renal insufficiency were also noted. The trajectory showed poor prognosis with a significant increase in length of stay in the ICU 11.3days, co-relating with a high APACHE IV 71.4. Risk of HAI is seen increased with 1 case of VAP and 1 reported CAUTI**.**
The outcome showed 66.6%% 180-day mortality and severe morbidity or 44.4% mortality during hospital stay. Conclusion: This study provides insight into the significant risk factors and clinical trajectory of acute spontaneous SDH in complicating course of hospitalization in critically ill patients. The findings underscore the importance of active surveillance in highrisk individuals, monitoring for new sensory or motor deficits, early detection and protocoled treatment among such patients. The severe outcome results stress the need for extra vigilance in critically ill patients complicated by acute SDH during the course of admission. Future studies should aim to include larger sample sizes and multicenter data to validate these findings and improve the generalizability of the results.

Keywords: Acute spontaneous subdural hematoma; critically Ill patients; trajectory; risk factors.

ABBREVIATIONS

SDH : subdural hematoma
APACHE : acute physiology and chronic health evalution
VAP : Ventilator acquired pneumonia
CAUTI : Catheter associated urinary tract infection
LRTI : lower respiratory tract infection
GOS : Glasgow outcome score
PTCA : percutaneous coronary angioplasty
CABG : Coronary Artery by-pass grafting
CLABSI : Central line associated blood stream infection
ICU : Intensive care unit
HAI : hospital acquired infection
UTI : Urinary tract infection
HTN : hypertension
GCS : Glasgow Coma Scale
LOS : Length of stay
SSI : surgical site infection
DAPT : Dual antiplatelet therapy
RCT : Randomized Control Trial

1. INTRODUCTION

SDH is a common type of intracranial hemorrhage that occurs beneath the dura mater, the outermost meningeal layer of the brain. This condition can result from traumatic injury or spontaneously in the absence of trauma. While chronic subdural hematoma is frequently encountered, especially in the elderly, ASDH in critically ill patients represents a distinct clinical challenge, marked by its sudden onset and the absence of an inciting traumatic event [1].

SDH can be classified into acute, subacute, and chronic types, based on the timing of symptom onset relative to the causative event. Acute SDH

typically manifests within days of head trauma, presenting a high risk of increased intracranial pressure and rapid neurological deterioration [2-4]. Chronic SDH, in contrast, develops over weeks to months and is often related to minor or unrecognized head injuries; it is particularly common among the elderly, where it may present with subtle and progressive symptoms [5].

The incidence of chronic SDH has been widely studied, with rates ranging from 1.7 to 20.6 per 100,000 person-years in the general adult population, and up to 129.5 per 100,000 among those aged 80 years or older [6-8]. However, the incidence of ASDH is significantly lower, estimated between 0.2 to 1.1 per 1,000 patient-years in populations not on antithrombotic therapy. This rarity underscores the importance of understanding its pathophysiology and risk factors in special populations, such as critically ill patients, who may be particularly vulnerable due to various predisposing factors [5].

The pathogenesis of ASDH is not fully understood but is thought to involve the rupture of bridging veins or cortical arteries, often exacerbated by vascular fragility due to systemic or cerebral pathology. In critically ill patients, factors such as coagulopathy, sudden changes in intracranial or venous pressures, infection, and metabolic disturbances can precipitate such events. Notably, the use of anticoagulant and antiplatelet medications, common among patients with cardiovascular diseases, increases the risk of developing ASDH even in the absence of overt trauma [9].

Critically ill patients often have multiple comorbidities, such as diabetes mellitus, hypertension, and ischemic heart disease, which may contribute to the complexity of their clinical management and increase their risk for spontaneous hemorrhages. The interplay of these conditions with systemic inflammation and vascular integrity can further predispose these patients to ASDH [9].

The clinical presentation of ASDH in critically ill patients can be particularly challenging to diagnose due to the overlap with other neurological complications common in this group, such as encephalopathy or the effects of sedation. Common symptoms such as headache, confusion, and drowsiness may be attributed incorrectly to other causes, delaying the diagnosis. Imaging, particularly computed tomography (CT) of the brain, plays a critical role in the timely identification and management of ASDH [10].

1.1 Aim

To investigate the trajectory and risk factors of acute spontaneous subdural hematoma complicating course of hospitalization in critically ill patients.

1.2 Objectives

1. To identify and analyze the risk factors contributing to the development of acute spontaneous SDH in critically ill patients.
2. To document the progression and clinical trajectory of acute spontaneous SDH during hospitalization.
3. To evaluate the outcomes of early diagnosis and intervention in the management of acute spontaneous SDH.

2. MATERIALS AND METHODS

Source of Data: The data for this retrospective study was sourced from the medical records of critically ill patients admitted to our hospital.

Study Design: This was a retrospective case series study, focusing on critically ill patients who developed acute spontaneous SDH during course in the hospital.

Study Location: The study was conducted at the intensive care unit (ICU) of a tertiary care hospital, Sevasadan Lifeline Superspeciality, Miraj.

Study Duration: The duration of the study spanned five years, from April 2019 to April 2024.

Sample Size: A total of 9 patients were included in this study.

Inclusion Criteria: Patients admitted to the ICU with various diagnoses who subsequently developed acute spontaneous SDH were included.

Exclusion Criteria: Patients with a history of trauma and those diagnosed with chronic SDH at the time of ICU admission were exclude*d.*

Procedure and Methodology: A detailed chart review was performed for each patient to extract data on age, sex, initial complaints, working diagnosis, and a timeline of clinical events

including day of SDH diagnosis and outcomes. Clinical interventions, imaging findings, and laboratory tests were also reviewed.

Sample Processing: Not applicable as this was a data-based, non-biomedical sample.

Statistical Methods: Descriptive statistics were used to summarize demographic and clinical data. Risk factors and outcomes were analyzed using appropriate statistical tests based on the data distribution.

Data Collection: Data were collected retrospectively from electronic medical records, focusing on predefined parameters such as demographic information, clinical course, interventions, and outcomes up to 90 days' post-discharge.

The parameters monitored to observe the clinical trajectory were length of stay in ICU, incidence of hospital acquired infections along with other details.

For outcome measures, mortality and morbidity were noted. Full recover was considered with Glasgow Coma Scale of 14-15 on discharge. Any score of or below 12 was regarded as partial recovery.

3. RESULTS

The observations along with the results are noted under each table.

Table 1 shows us the general demographic data along with the co-morbid conditions that the patients presented with. Mean age group was 63.8 years while of 9 patients 4 identified as females and 5 as males. 6 patients were diagnosed and treated for ischaemic heart disease and hypertension. While number of diabetics was 5. 4 patients were treated for renal insufficiency. Most co-morbid diseases existed together. 3 patients each gave history of chronic smoking and alcohol abuse [11-13].

The primary or working diagnosis for which the patient was initially admitted and treated noted as sepsis either foci being respiratory or renal in 3 patients. While 2 were diagnosed as acute kidney injury. Other diagnoses included acute cholecystitis, Mallory-Wiess (M-W) tear, accelerated hypertension and metabolic encephalopathy.

Table 2 details the risk factors identified in nine patients with acute spontaneous SDH. The majority were on either single or dual antiplatelet 77.7%, which is known to significantly increase the incidence of SDH in elderly. Of them 6 had previously undergone cardiac intervention either cardiovascular bypass surgery: 2 patients, PTCA: 4 patients. All these patients were on antiplatelet drugs.

More than half (55.6%) were aged 50 years or older, underlining age as a significant risk factor. Accelerated hypertension was noted in 2 patients, also known to be a risk factor for developing cerebral hemorrhages. Alcoholic liver disease itself leads to dysregulated coagulation; was present in a third of the cases (33.3%), and may to the risk of developing SDH in patients [14,15].

Table 3 highlights the clinical trajectory of patients with acute SDH in an underlying disease. For prognostication APACHE IV score calculated on admission, at interval and on ward transfer was 28.3, 71.4 and 46.5 respectively. Estimated mortality was given at 68.5 % and ICU stay at 9.6 where as actual ICU stay was around 11.3 days on average. The Glasgow coma scale dipped at interval to average 7.8 and on ward transfer was around 12.64 patients underwent immediate neurosurgical intervention decompression craniotomy with evacuation/burr hole.

All patients at some point of time required invasive ventilation with average ventilator days calculated at 9.1 and 3 out of 9 patients were tracheostomised.

A higher incidence of hospital acquired infection was seen as 1 case reported for VAP and 1 for CAUTI. There were no CLABSI or SSI present.

Table 4 focuses on the outcomes after early diagnosis and intervention in managing acute spontaneous SDH. A third of the patients (33.3%) achieved full recovery (measured in terms of GOS on discharge 5); which might reflect effective medical or surgical interventions. Partial recovery was seen in 22.2% of the cases, with GOS ranging 3 or below. This denotes the significant morbidity at discharge.

Alarming index is the mortality of 44.4%, thus signifying the grave prognosis of the disease. The 90-day mortality rate was 11.1%, underscoring the serious nature of acute SDH even when diagnosed and treated in a timely manner. Only 1 patient was screened on 180 day follow-up while 3 patients were lost in between.

Table 1. Demographic data

Demographic data	**n**	
Age		
Mean	63.8	
Sex		
Female	4	
Male	5	
Co-morbid conditions	**n**	**%**
Hypertension	6	66.7
Diabetes Mellitus	5	55.5
Ischaemic heart disease	6	66.6
Renal Insufficiency	4	44.4
History of Smoking	3	33.3
Chronic Alcohol abuse	3	33.3
Working Diagnosis on admission	**n**	
LRTI, sepsis	2	22.2%
UTI, sepsis	1	11.1%
Acute cholecystitis	1	11.1%
Acute kidney injury	2	22.2%
M-W tear, gastritis	1	11.1%
Accelerated HTN	1	11.1%
Metabolic encephalopathy	1	11.1%

Table 2. Trajectory and Risk Factors involved in development of Acute Spontaneous Subdural Hematoma in Critically Ill Patients (n = 9)

Risk Factor	**n**	**%**
Age ≥ 50 years	5	55.5
Use of Antiplatelets single/dual	7	77.7
No of patients that underwent cardiac procedures		
PTCA	4	44.4
CABG	2	22.2
Acute renal injury	3	33.3
Accelerated Hypertension	2	22.2
Alcohol liver disease	3	33.3

4. DISCUSSION

The prognosis of any critically ill patient depends mainly on receiving excellent care. Vigilant monitoring and alteration in treatment plan forms crux of management, eventually influencing the outcome. We find according to Table 1, with mean age 63.8, definitely elderly were more group affected. No significance was found in male to female ratio. Ischaemic heart diease, hypertension predominated in 66.6% cases; whereas Diabetes mellitus was found in 55.5% cases while, renal insufficiency, alcohol and smoking history were found in 33.3% cases. Trevisi G et al. [16] mapped the trajectory of elderly with acute SDH giving similar demographics and comorbid factors. The study mentions overall poor prognosis in elderly patients leading to significant mortality and morbidity.

Many isolated cases of ASDH complicating patients with primary diagnosis of renal disorder, pre-eclampsia, thrombocytopenia have been reported [17-19], thus influencing the overall outcome. Again in each scenario, conclusion points to involvement dysregulated platelet function or coagulopathy as a major risk factor in development of ASDH.

Anti-thrombotic therapy and it's implication in development of neurological bleeding tendencies is well debated. Various RCTs, systematic review analyses conclude the same facts. Connolly et al.analyzed data from randomized clinical trials and reported that the incidence of SDH in patients on aspirin therapy was 0.02/1,000 patient-years.Wong et al. further noted that the location of lobar hematoma was higher in the aspirin group (32.8%) compared to the control group (10.3%). Bakheet et al.performed a meta-

analysis of 11 randomized clinical trials investigating the risk of SDH in patients receiving DAPT (clopidogrel plus aspirin). Although eight trials did not show any cases of spontaneous SDH, three trials with 23,136 participants reported 39 cases of SDH during a mean follow-up of 2.1 years per patient [20-22].

The impact of smoking, potentially through its vascular effects, is also supported by evidence linking it to various types of cerebrovascular accidents.

The clinical trajectory shown in this table reflects the critical nature of acute SDH management. APACHE IV scoring showed increasing trend in the interval corelating with a drop in GCS. This strongly indicates the worsening of primary disease with acute SDH complicating the situation. Almost half the patients required surgical intervention. All 9 patients required invasive ventilation at some point of time with 3 getting tracheostomised. All these indices do indicate a rough course in the hospital following acute SDH.

The estimated length of stay as predicted by APACHE IV was 9.1 whereas actual ICU stay averaged to 11.8. Advent of CAUTI and VAP in one patient each is a significant factor delaying recovery and transfer. Abdulhasn et al. [23] studied the HAIs in neurocritical care unit of developing country. It specifically mentions VAP to be closely associated with mortality while all others significantly increased the length of stay in ICU.

Though many studies that suggest a significant portion of SDH patients may experience rapid improvement or stabilization with appropriate medical and surgical intervention Shoiab A et al. [24]; we lost 3 out of 4 patients who were intervened. It thus cannot be ascertained if the primary disease or surgical intervention for complicating factor was responsible for deterioration.

Table 3. Clinical Trajectory of acute spontaneous SDH

APACHE IV	
On admission (n=mean)	28.3
interval	71.4
On ward transfer	46.5
GCS	
On Admission (mean)	15
Interval	7.8
on ward transfer	12.6
Surgical intervention required	n=4
Average LOS in the ICU (days)	11.3
mean Ventilator days	9.1
Tracheostomy required	n=3
Estimated mortality %	68.5
Estimated ICU stay days	9.6
Incidence of HAI	
CLABSI	0
CAUTI	1
VAP	1
SSI	0

Table 4. Outcomes of early diagnosis and intervention

Outcome	**n**	**%**
Full Recovery GOS 5	3	33.3
Partial Recovery GOS 3-4 or below	2	22.2
Death	4	44.4
90-day Mortality	1	11.1
180 day morbidity GOS 3-4	1	11.1
Lost to follow up	3	33.3

GOS Glasgow Outcome scale was used to predict the outcomes,which were quite unfavourable with almost 44.4% mortality in house and 1 at 90-day follow-up signifying the severity of the nature of disease. In an independent study by Khaki et al. [25], differences in presentation, radiological findings and outcomes of traumatic and spontaneous acute SDH were noted; wherein spontaneous type was found to have better outcomes than traumatic nature. However, this was a single centre trial and acute SDH itself was the primary.

33.3% ended up with moderate to severe morbidity on discharge. 3 patients were lost around 180 day follow-up, GOS of 1 patient improved while 2 remained same, again emphasizing the burden of the disease.

Mortality rates comparable to those in the literature emphasize the potential lethality of this condition if not promptly and effectively managed Liu T et al. [26].

5. CONCLUSION

This five-year case series study has provided valuable insights into the trajectory and risk factors associated with acute spontaneous subdural hematoma (SDH) in critically ill patients.

Risk factors which need ear-marking would be elderly, comorbidity, use of antiplatelets and substance abuse. The impact of complication on management of primary diagnosis is profound, lengthening the course of stay in the hospital and the consequences followed. Active surveillance, precise monitoring, early detection, protocoled treatment are key factors for improving outcome.

The conclusions drawn from this investigation highlight the complex interplay of various medical conditions and treatments in the evolution of acute spontaneous SDH, reinforcing the need for multidisciplinary approaches in the management and care of critically ill patients.

6. LIMITATIONS OF STUDY

1. **Small Sample Size**: The study is based on a small cohort of only nine patients. Such a limited sample size restricts the statistical power of the study and the generalizability of the findings. It may not capture the full spectrum of variability in clinical presentations and outcomes that could be observed in a larger population.
2. **Retrospective Design**: As a retrospective analysis, this study relies on the review of existing medical records and data. This design can introduce biases related to data completeness and accuracy. Important variables and potential confounders might not be consistently recorded, leading to incomplete data analysis.
3. **Lack of Control Group**: The absence of a control group limits the ability to directly attribute observed outcomes to specific risk factors or interventions. Without comparing to a matched cohort without SDH, it's challenging to establish causal relationships.
4. **Single-Center Study**: Conducted in a single clinical setting, the findings may not be applicable to other settings due to differences in patient demographics, clinical practices, and healthcare resources. The external validity and applicability of the results to other populations or regions might therefore be limited.
5. **Potential Confounders**: Given the complex nature of critically ill patients who often present with multiple comorbidities and are subjected to various interventions, there are likely numerous confounding factors that were not fully controlled or accounted for in this study. These could include variations in the management of anticoagulation, differences in surgical techniques, or the influence of other medications.
6. **No Longitudinal Follow-up**: The study lacks longitudinal follow-up to assess long-term outcomes and late complications of patients who develop acute spontaneous SDH. Understanding the durability of recovery or the potential for late deteriorations is crucial for comprehensive care planning.
7. **Quantitative Analysis Limitations**: The absence of statistical tests such as odds ratios and p-values due to the small sample size prevents a robust quantitative analysis of risk factors. This limits the ability to determine the strength and significance of associations between observed risk factors and the development of acute SDH.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models

(ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

CONSENT

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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