

RESEARCH PAPER

International Journal of Health Research and Medico-Legal Practice

Copyright © 2022 Baruah SK, et al. This is an open-access article distributed under the Creative Commons Attribution License permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited



Profile of complications of spontaneous subarachnoid haemorrhage in a tertiary care hospital

Baruah SK¹ Medhi J² Islam M³

Address for Correspondence: ¹ Professor Mobile: +919435015668 Email: drskbaruah1@gmail.com ² Registrar (Corresponding author) Mobile : +919476521392 Email: jayasrimedhi123@gamil.com ³Senior Resident, Department of Medicine Gauhati Medical College and Hospital, Assam Article received: 04-08-2021 **Received** (revised): 01-06-2022 Editorial approval: 23-06-2022 Checked for plagiarism: Yes **Peer-reviewed**: Double-blinded Peer review comments: Three Editor who approved: Prof (Dr.) Putul Mahanta

Background and objectives: Subarachnoid haemorrhage (SAH) refers to bleeding within the subarachnoid space. The different short-term outcomes could be signs of delayed ischemic deficit, deterioration of the conscious level, motor deficit, aphasia, hydrocephalus and re-bleeding. When cardiac dysfunction is severe enough in some people, it can substantially lower the ejection fraction and result in heart failure. This paper aims to study the short-term outcome of spontaneous subarachnoid haemorrhage in a tertiary care hospital. Material and methods: This was an institution-based observational study on 73 patients admitted to the Department of Medicine and Neurology, Gauhati medical college and hospital, Guwahati, Assam. All patients underwent a CT scan brain, CT Angiography along with MR Angiography of head and neck vessels and DSA (Digital subtraction angiography), if necessary. Based on CT scan brain findings, the patients were categorized according to Fisher's scale. Results: The age of the SAH cases ranged from 29 to 78 years, with a male: female ratio of 1:1.6. Out of the 73 patients, 86.3% of patients have developed various complications. Signs of delayed ischemic deficit were observed in 42.46% of cases, out of which deterioration of conscious level (24.75%) was the most common, followed by a motor deficit (12.32%) and aphasia (8.22%). Hydrocephalus (21.91%), re-bleeding (6.84%) and electrolyte abnormalities like hyponatremia (38.35%) were also observed. Diastolic dysfunction (56.94%) was the most reported cardiac complication. **Conclusion:** Neurological complications like signs of delayed cerebral ischemia, cardiac complications, mainly diastolic dysfunction and low than normal electrolyte levels like Hyponatremia and Hypokalemia were the most common complications of SAH.

Keywords: Subarachnoid haemorrhage; aneurysms; hypertension; diastolic dysfunction.

Cite this article: Baruah SK, Medhi J, Islam M. Profile of complications of spontaneous subarachnoid haemorrhage in a tertiary care hospital. Int J Health Res Medico Leg Prae 2022 Jan-June;8(1):14-18. Doi: 10.31741/ijhrmlp.v8.i1.2022.4

INTRODUCTION

Spontaneous subarachnoid haemorrhage (SAH) is considered a global emergency. The presence of blood in the subarachnoid space between the arachnoid-mater and piamater is termed SAH and is always pathological. SAH accounts for almost 5% of all the causes of stroke worldwide¹ and 3-4.8% of strokes in India.² Almost 85% of cases occur due to rupture of the aneurysm.³ The most significant risk factors of aneurysmal SAH are smoking and hypertension. Hypertension increases the risk of SAH by 2.5 times.^3

SAH is clinically characterized by sudden, intense headaches, vomiting, and neck rigidity.⁴ One-third of the patients have focal neurological abnormalities, and half arrive unconscious. Seizures occur in up to 20% of patients, while 1 to 2% of patients get an acute confusional state, and in most patients, a history of sudden headaches is lacking.⁴

Subarachnoid haemorrhage is a medical emergency that is also very prone to complications. Different arrays of neurological and medical complications are seen in subarachnoid haemorrhages with very high incidence rates. The various neurological complications of SAH are delayed ischemic deficit and cerebral vasospasm, deterioration of the conscious level, motor deficit, aphasia,⁴ re-bleeding,⁵ hydrocephalus, intraventricular and intracerebral haemorrhage etc. Medical complications are also common after SAH and contribute substantially to morbidity and mortality. Electrolyte disturbances, cardiac arrhythmias,6 etc., are the most reported medical complications. Severe cardiac dysfunction may significantly decrease the ejection fraction and cause heart failure in some patients.⁷ Deaths attribut-able to medical complications equalled those caused by neurological complications, with about a fifth of patients dving of vasospasm, re-bleeding, and direct effects of SAH, and medical com-plications.8

The objective of the present study is to evaluate the shortterm outcome of spontaneous subarachnoid haemorrhage in a tertiary care hospital.

MATERIALS AND METHODS

The study is an institution-based observational study including patients with spontaneous subarachnoid haemorrhage presenting in the emergency and outpatient department (OPD) of Medicine and Neurology, Gauhati medical college and hospital (GMCH) from 1st July 2018 to 30th June 2019.

Inclusion Criteria: All patients older than 12 years presenting to Medicine OPD, Neurology OPD and Emergency Department with the diagnosis of spontaneous subarachnoid haemorrhage on CT scan brain were included.

Exclusion Criteria: Patients with a history of head injury and associated findings in CT scan brain haemorrhage from other sites were excluded. Patients under 12 years of age and those who refused to consent were also excluded from the study.

All study participants underwent a physical examination and neurological assessment on admission, a CT scan of the brain, CT Angiography of head and neck vessels, MR Angiography of head and neck vessels and DSA (Digital subtraction angiography), if necessary, were performed. The grading of patients was done according to Fisher's scale⁷ based on CT scan brain findings (**Table 1**).

Table1	Grades	of	CT	scan	findings	according	to	the
			Fi	isher	scale			

Grade	Fisher Scale ⁷
1	No SAH or intraventricular haemorrhage
2	Diffuse deposition of a thin layer with all vertical layers of blood (interhemispheric fissure, insular cistern, ambient cistern) <1 mm thick
3	Vertical layers of blood >1 mm thick or localized clots (clots defined as $>3 \times 5$ mm)
4	Diffuse or no subarachnoid blood, but with intracerebral or intraventricular clots

RESULTS

A total of 73 patients with SAH, fulfilling the inclusionexclusion criteria, were included in the study. The age of the patients ranged from 29-78 years, with a mean age of 53.16 years. Out of the 73 patients, the majority (61.64%) were females with a male: female ratio of 1:1.6.

Out of the total 73 patients, 86.3% of patients have developed various complications. There was no mortality in the present study (**Figure 1**).

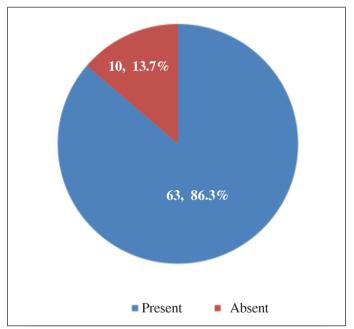


Figure 1 Complications of Subarachnoid haemorrhage

The majority, 42 out of 73(57.53%) patients, had Fisher's grade 2, and 20(27.39%) had grade 3 on CT brain. Intracerebral or intraventricular clots (Fisher scale Grade 4) were observed in 11(15.06%) patients.

In this present study, 63 patients have developed various complications. The delayed ischemic deficit was the most common neurological complication observed in 31(42.46%) cases. Hydrocephalus (21.91%) and re-bleeding (6.84%)

were the other neurological complications observed in the cases. Hyponatremia was observed in 38.35% of cases and Hypokalemia in 15.06% of cases. Diastolic dysfunction was the most reported cardiac complication found in 41(56.94%) cases, followed by ventricular wall motion abnormality (22.22%), as shown in **Table 2**.

Table 2	Distribution	of	Complications	of	SAH
---------	--------------	----	---------------	----	-----

Neurological complications:						
Signs of delayed cerebral ischemia	31	42.46%				
Hydrocephalus	16	21.91%				
Rebleeding	5	6.84%				
Electrolyte imbalance						
Hyponatremia	28	38.35%				
Hypokalemia	11	15.06%				
Hypernatremia	5	6.84%				
Hyperkalemia	4	5.48%				
Cardiac complications						
Diastolic Dysfunction	41	56.94%				
Wall motion abnormality	16	22.22%				
U wave abnormality	14	19.44%				
ST Elevation	13	18.05%				
T wave abnormality	13	18.05%				
QT prolongation	10	13.89%				
LVH	9	12.5%				
ST Depression	6	8.33%				

Out of the 31 cases with signs of delayed ischemic deficit, deterioration of the conscious level is the most common complication observed in 18(24.65%) cases, followed by a motor deficit (15.06%) and aphasia (10.95%). There are four patients with both aphasias as well as a motor deficit. The median time of onset of delayed cerebral ischemia is the 8th day in the present study (**Table 3**).

Table	3	Signs	of	delayed	cerebral	ischemic	deficit
-------	---	-------	----	---------	----------	----------	---------

Deficit	Number of patients	Percen- tage	Median time of onset
Deterioration of consciousness	18	24.75%	7 th day
Motor deficit	11	15.06%	10 th day
Aphasia	8	10.95%	8 th day
Total no of patients with			
delayed neurological deficit	31	42.46%	8 th day

DISCUSSION

The incidence of various complications in SAH is found in 86.30% of cases in the present study. This corresponds to the study of Hijdra et al.,⁹ Roos et al.,¹⁰ and Wurm et al.^{11,} who had found the incidence of various complications of

SAH to be 80%, 91% and 84.5%, respectively. However, Krammer et al.¹² reported the incidence of complications in 70% of SAH cases. This discrepancy may be due to the lesser severity of the subarachnoid bleed in the cited study.

It has been found that 57.53% of cases are in Fisher's grade 2 and 27.39% in grade 3, and 15.06% in grade 4. However, Fisher⁷ found 24% of patients in grade I, 15% in Grade II, 51% in grade III and 10% in grade IV. Wurm et al.,¹¹ found 4.5% of Fishers' grade I cases, 25.4% in Grade II, 18.7% in Grade III and 51.5% in grade IV. The differences in Fisher's grade in the various studies indicate the difference in the severity of the Subarachnoid bleed in different cases.

Signs of delayed cerebral ischemia were seen in 31(42.46%)cases, with deterioration of the consciousness level being the most common complication. The observations agree with Wurm et al.,11 who found 42.86% of cases with delayed cerebral ischemia. Hijdra et al.,9 have found the incidence of delayed ischemia in 38.5% of cases which also correlates with the present study. However, Roos et al.,¹⁰ Krammer et al.,12 and Juvela et al.,13 have reported 17%, 26% and 22.08% delayed ischemia in their studies. The low incidence of delayed ischemia may be due to the lower severity of the bleed and vasospasm, as seen by the more significant number of Fisher's grade II and III patients in the abovementioned studies. The mean time of onset of delayed cerebral ischemia in the current study is the 8th day, which corresponds to the study done by Weidauer et al.,¹⁴ who reported it occurring between the 7th -14th day.

Re-bleeding is one of the most dangerous complications of SAH, seen in 5(6.84%) cases in the present study, which corresponds well to the 7% of re-bleeding cases reported by Solenski et al.¹⁵ It is found to occur at a rate of 16% and 21.6% in Roos et al.,¹⁰ and Juvela et al.,¹³ respectively. The higher incidence of re-bleeding in Roos et al.,¹⁰ and Juvela et al.,¹⁰ and Juvela et al.,¹³ may be due to various factors predisposing to re-bleeding, like the location of the aneurysm and the more significant number of Fisher's grades 3 and 4 patients in those studies.

Acute hydrocephalus was present in the study's 16(21.91%) cases. It is reported to occur in 20% of cases by Behrouz et al.,¹⁶ which corresponds to the present study. But Wurm et al.,¹¹ found a higher incidence (38%) of acute hydrocephalus in their study, which may be because he has got almost 24% of cases with IVH and ICH, which predisposes to the development of hydrocephalus.

Hyponatremia was seen in 28(38.35%) cases and corresponds to the study reported by Wijdicks et al.,¹⁷ and Qureshi et al.,¹⁸ who found hyponatremia in 34% and 30%, respectively. 5(6.84%) cases of hypernatremia are present in the present study. Takaku et al.,¹⁹ found hypernatremia only in 2% of cases, whereas 19% of cases of hypernatremia were found in the study of Qureshi et al.¹⁸. This discrepancy may be due to other causes of hypernatremia apart from SAH, like fluid intake. Hypokalemia was found in 11(15.06%) cases and hyperkalemia in 4(5.48%) cases which corresponds to Cruickshank et al.,¹⁹ who found Hypokalemia in 19% of cases. Frontera et al.,²⁰ reported 28% cases of Hypokalemia in their study, which may be due to the larger sample size.

Diastolic dysfunction (56.94%) was the most observed cardiac complication reported in the present study. In contrast to our findings, studies by Kopelnik et al.,²¹ and Bilt et al.,⁸ reported diastolic dysfunction in 71% and 69% of cases with SAH. This higher incidence may be due to the larger sample size in their study group and the increased incidence of hypertensive cases and age. However, in the present study, 10 patients were found to have diastolic dysfunction on admission, possibly due to previous cardiac illness or hypertension. Various ECG abnormalities are found in 56(77%) cases in the current study, while Sakr et al.,²² found ECG changes in more than 60% of cases which may again be due to the larger sample size in their study.

CONCLUSION

In the present study, it was found that most of the patients developed complications after SAH. Among the short-term outcome, the signs of delayed ischemic deficit were the most frequently encountered complications, followed by hydrocephalus and re-bleeding. Out of delayed ischemic deficit, deterioration of the level of consciousness was the most common, followed by motor deficit and aphasia. Electrolyte abnormalities like hyponatremia and Hypokalemia were also common among SAH patients. Diastolic dysfunction was the most common cardiovascular complication encountered by the patients.

Both neurological and medical complications are common after SAH. Prompt assessment and management of the associated complications of SAH may help reduce the burden of morbidity and mortality in those cases.

Data availability: The data used to support the findings of this study are included in the article.

Conflicts of interest: None declared.

Funding statement: This research did not receive any funding.

Acknowledgements: We acknowledge the department's staff members who have helped us complete this project by providing the department's necessary infra-structures.

Supplementary materials: Nil.

Ethical clearance: Taken from the institutional ethics committee of Gauhati medical college and hospital, Guwahati, vide ref no: MC/190/2007/pt-1/1EC/91.

REFERENCES

- 1. Anderson C, Ni Mhurchu C, Scott D, Bennett D, Jamrozik K, Hankey G et al. Triggers of subarachnoid haemorrhage: role of physical exertion, smoking, and alcohol in the Australasian Cooperative Research on Subarachnoid Hemorrhage Study (ACROSS). Stroke 2003 Jul; 34(7):1771-6.
- 2. Banerjee TK, Mukherjee CS, Sarkhel A. Stroke in the urban population of Calcutta-an epidemiological study. Neuroepidemiology 2001 Aug;20(3):201-7.
- Banki NM, Kopelnik A, Dae MW, Miss J, Tung P, Lawton MT, et al. Acute neurocardiogenic injury after subarachnoid hemorrhage. Circulation 2005 Nov 22; 112(21):3314-9.
- Bederson JB, Levy AL, Ding WH, Kahn R, DiPerna CA, Jenkins AL 3rd, et al. Acute vasoconstriction after subarachnoid hemorrhage. Neurosurgery 1998 Feb; 42(2):352-62.
- Bederson JB, Connolly ES Jr, Batjer HH, Dacey RG, Dion JE, Diringer MN, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. Stroke 2009 Mar;40(3):994-1025.
- Behrouz R, Birnbaum LA, Jones PM, Topel CH, Misra V, Rabinstein AA. Focal Neurological Deficit at Onset of Aneurysmal Subarachnoid Hemorrhage: Frequency and Causes. J Stroke Cerebrovasc Dis 2016 Nov;25(11):2644-7.
- 7. Ropper AH, Samuels MA, Klein J. Adams and Victor's principles of neurology. 2014; 34(2):565-82.
- 8. Van der Bilt IA, Hasan D, Vandertop WP, Wilde AA, Algra A, Visser FC, et al. Impact of cardiac complications on outcome after aneurysmal subarachnoid hemorrhage: a meta-analysis. Neurology 2009 Feb 17;72(7):635-42.
- Hijdra A, Van Gijn J, Stefanko S, Van Dongen KJ, Vermeulen M, Van Crevel H. Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage: clinicoanatomic correlations. Neurology 1986 Mar; 36(3):329-33.
- Roos YB, de Haan RJ, Beenen LF, Groen RJ, Albrecht KW, Vermeulen M. Complications and outcome in patients with aneurysmal subarachnoid haemorrhage: a prospective hospital-based cohort study in the Netherlands. J Neurol Neurosurg Psychiatry 2000 Mar; 68(3):337-41.
- 11. Wurm G, Tomancok B, Nussbaumer K, Adelwöhrer C, Holl K. Reduction of ischemic sequelae following spontaneous subarachnoid hemorrhage: a double-blind,

randomized comparison of enoxaparin versus placebo. Clin Neurol Neurosurg 2004 Mar;106(2):97-103.

- Kramer AH, Hehir M, Nathan B, Gress D, Dumont AS, Kassell NF, et al. A comparison of 3 radiographic scales for the prediction of delayed ischemia and prognosis following subarachnoid hemorrhage. J Neurosurg 2008 Aug;109(2):199-207.
- 13. Juvela S. Minor leak before rupture of an intracranial aneurysm and subarachnoid hemorrhage of unknown etiology. Neurosurgery 1992 Jan;30(1):7-11.
- 14. Weidauer S, Lanfermann H, Raabe A, Zanella F, Seifert V, Beck J. Impairment of cerebral perfusion and infarct patterns attributable to vasospasm after aneurysmal subarachnoid hemorrhage: a prospective MRI and DSA study. Stroke 2007 Jun;38(6):1831-6.
- 15. Solenski NJ, Haley EC Jr, Kassell NF, Kongable G, Germanson T, Truskowski L, et al. Medical complications of aneurysmal subarachnoid hemorrhage: a report of the multicenter, cooperative aneurysm study. Crit Care Med 1995 Jun;23(6):1007-17.
- Behrouz R, Birnbaum LA, Jones PM, Topel CH, Misra V, Rabinstein AA. Focal Neurological Deficit at Onset of Aneurysmal Subarachnoid Hemorrhage: Frequency and Causes. J Stroke Cerebrovasc Dis 2016 Nov;25(11):2644-2647.

- 17. Wijdicks EF, Vermeulen M, Hijdra A, van Gijn J. Hyponatremia and cerebral infarction in patients with ruptured intracranial aneurysms: is fluid restriction harmful? Ann Neurol 1985 Feb;17(2):137-40.
- Qureshi AI, Suri MF, Sung GY, Straw RN, Yahia AM, Saad M, et al. Prognostic significance of hypernatremia and hyponatremia among patients with aneurysmal subarachnoid hemorrhage. Neurosurgery 2002 Apr; 50(4):749-55; discussion 755-6.
- Cruickshank JM, Neil-Dwyer G, Brice J. Electrocardiographic changes and their prognostic significance in subarachnoid haemorrhage. J Neurol Neurosurg Psychiatry 1974 Jun;37(6):755–9.
- 20. Frontera JA, Parra A, Shimbo D, Fernandez A, Schmidt JM, Peter P, et al. Cardiac arrhythmias after subarachnoid hemorrhage: risk factors and impact on outcome. Cerebrovasc Dis 2008;26(1):71-8.
- 21. Kopelnik A, Fisher L, Miss JC, Banki N, Tung P, Lawton MT, et al. Prevalence and implications of diastolic dysfunction after subarachnoid hemorrhage. Neurocrit Care 2005; 3(2):132-8.
- 22. Sakr YL, Lim N, Amaral AC, Ghosn I, Carvalho FB, Renard M, Vincent JL. Relation of ECG changes to neurological outcome in patients with aneurysmal subarachnoid hemorrhage. Int J Cardiol 2004 Sep; 96(3):369-73.