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HISTORY OF ALCOHOL TO PREDICT OUTCOME IN PATIENTS OF ANEURYSMAL SUBARACHNOID HEMORRHAGE

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ABSTRACT Blood between the arachnoid membrane and the pia membrane is known as subarachnoid hemorrhage (SAH). Aneurysmal SAH occurs due to cerebral artery ruptures. Other than hypertension, there can be pre-ictal factors that can affect the outcome in addition to these independent risk factors that are linked to the extent of the bleeding. Whether smoking, drinking alcohol, or using nonsteroidal anti-inflammatory medicines affects the outcome is unknown. Drinking alcohol, particularly excessively, has been linked to an increased risk of stroke in all its forms. Finding their correlation in aneurysmal SAH could aid in ICU (intensive care unit) planning and treatment decision-making. **Summary:** Among 30 patients, 11 patients were alcoholic and 19 patients were non-alcoholic, among alcoholic 4 patients died (36.36%) their follow up noted using Glasgow outcome score from 1st to 3rd month and change in modified Rankin score from 1st to 3rd month.

KEYWORDS : Subarachnoid hemorrhage, Cerebral artery ruptures, History of Alcohol,

INTRODUCTION:

Blood that accumulated between the arachnoid and pia membranes is referred to as a subarachnoid hemorrhage (SAH). Aneurysmal SAH is caused by cerebral artery ruptures.1 Possible signs of aneurysmal SAH include nausea, changed sensation, a severe headache that appears suddenly, and possibly convulsions. Neck discomfort or stiffness is another prevalent problem.2 Aneurysmal rupture causes a global acute ischemic injury within 0–2 days, while vasospasm causes a delayed ischemic injury within 3–21 days. These two elements are the primary determinants of the natural course of aneurysmal subarachnoid hemorrhage. Aneurysmal subarachnoid hemorrhage is associated with risk factors such as smoking, high blood pressure, alcoholism, cocaine use, and family history.[1]

Finland and Japan have an incidence of about 20 cases of aneurysmal SAH per lac population, while other populations have an incidence of 6-7 cases per lac population per year (after age-adjusted rates). Every seven to eight years, a general practitioner with 2,000 patients will see one patient who has a subarachnoid hemorrhage. Even though the risk of subarachnoid hemorrhage rises with age, half of patients are under 55 when the bleeding occurs.85% of cases are caused by ruptured aneurysms; 10% are related to a very benign disorder called non-aneurysmal peri mesencephalic hemorrhage; and the other 5% are brought on by a variety of strange causes.[3]

Localized cerebral blood vessel dilatations, or intracranial aneurysms, are caused by vessel wall weakening. Although they are usually saccular in shape, they can occasionally have a fusiform shape. An out-pouched, tapering fundus connects saccular aneurysms to their proximal necks for support. There may be involvement of nearby branching arteries in the event of fundus lobulation. Intracerebral aneurysms are thought to be a chronic, acquired, degenerative disorder of the cerebral arteries, while the precise etiology is unknown. In most cases, cerebral aneurysms are seen where the circle of Willis's main arteries diverge. [4-6]

The effects of alcohol on platelet function, coagulation pathways, and vascular health may increase the risk of complications such delayed cerebral ischemia, vasospasm, and rebleeding, all of which have a major negative influence on patient life and recovery. Furthermore, alcohol misuse may make comorbid conditions including liver disease, hypertension, and cognitive impairment and worsen making recovery for people with aSAH even more challenging.

For the most part, patients with aneurysmal SAH cannot have their prognosis predicted by clinical or radiographic evaluations. While there have been a few studies on alcohol history to predict the outcome, they are extremely small in number and were conducted independently to evaluate the long-term result. In order to better understand alcohol history in patients experiencing aneurysmal subarachnoid hemorrhage the current study is designed to investigate outcome.

OBJECTIVE OF STUDY WAS; To evaluate the effect of alcohol in aneurysmal subarachnoid hemorrhage patient's outcome.

INCLUSION CRITERIA:

1. Those who are clinically and radiologically diagnosed with aneurysmal subarachnoid hemorrhage.
2. Age> 18 years
3. Underwent clipping of an aneurysm SAH

EXCLUSION CRITERIA

1. History of previously diagnosed neurological diseases.
2. History of any psychiatric medications.

MATERIALS AND METHODS:

The present study was conducted at the Department of Biochemistry in collaboration with the Department of Neurosurgery. N=30(Thirty) patients admitted within 48hrs of ictus and then underwent microsurgical clipping of aneurysm have been included in the study. Their clinical history, detailed alcohol history, World Federation of Neurological Surgeons (WFNS) grade, radiological findings, and modified fisher grade recorded. They were subjected to complete clinical examination, and routine blood investigations. Operative findings in terms of the time of surgery, time since ictus, brain bulge, operative time, temporary clipping time, and any other surgery other than clipping have been recorded. Postoperative clinical status and available radiological findings have been recorded in the immediate post-operation period, at the time of discharge from ICU, and at the time of discharge from the hospital. Follow-up outcome score is recorded in terms of Glasgow outcome score (GOS) and modified Rankin Score (mRS) at 1st and 3rd from discharge of the hospital.

Table 1— Glasgow outcome score (GOS) according to which outcome recorded at 1st month and 3rd month from discharge of the hospital

GOS rating	Definition
5. Good recovery	Resumption of normal life despite minor deficits
4. Moderate disability	Disable but independent but can work in sheltered setting
3. Severe disability	Conscious but disabled. Dependent on daily support
2. Persistent vegetative	Minimal responsiveness
1. Death	No survival

Table 2—modified Rankin scale according to which outcome recorded at 1st month and 3rd month from discharge of the hospital

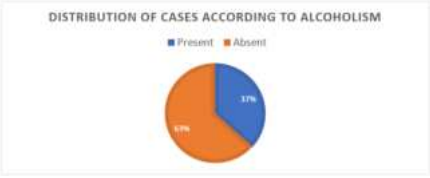
Modified Rankin scale	
0	No symptoms
1	No significant disability, Able to carry out all usual activities, despite some symptoms.
2	Slight disability. Able to look after own affairs without assistance, but unable to carry out all previous activities.
3	Moderate disability. Requires some help, but able to work unassisted.
4	Moderate severe disability, Unable to attend to own bodily needs without assistance, and unable to walk unassisted.
5	Severe disability. Requires constant nursing care and attention, bedridden, incontinent.
6	Dead

RESULTS AND DISCUSSION:

Table 3: Distribution of cases according to alcoholism. (N = 30)

H/o Alcoholism	Number of cases	Patients died
Present	11 (36.66%)	4 (36.36%)
Absent	19 (63.33%)	5 (26.31%)

Chart 1- Distribution of cases according to alcoholism



In the present study among cases, 11 patients had history alcohol consumption and 19 patients were non-alcoholic. Among alcoholic patient group 4 patients died (36.36%) within 3rd month of discharge from the hospital, followed up using Glasgow outcome score (GOS) and modified Rankin Score (mRS). Among Non-alcoholic group 5 patients died within 3rd month of discharge from hospital. Results are statistically insignificant but the patient dying among alcoholic group is more (36.36%) compared to patient with non-alcoholic group patient (26.31%).

The absence of statically insignificant data between alcohol history and aSAH outcomes does not necessarily mean that alcohol is irrelevant to the patient's recovery. It is possible that alcohol's impact on aSAH outcomes may be more indirect, interacting with other factors in ways that were not fully accounted for in this study. For example, alcohol's role in increasing the risk of hypertension, liver disease, or cognitive dysfunction may have a cumulative effect that is not fully reflected in the acute phase of recovery but may still influence long-term outcomes. Alcohol may have a more indirect effect on aSAH outcomes, combining with other factors in ways that this study did not fully account for. For instance, alcohol may have a cumulative effect that is not entirely evident during the acute phase of recovery but may still have an impact on long-term results due to its role in raising the risk of hypertension, liver disease, or cognitive dysfunction.

CONCLUSION:

Results are statistically insignificant but the patients dying among alcoholic group is more compared to patient with non-alcoholic group. The history of alcohol consumption is a significant component in the acute care and long-term recovery in aneurysmal SAH patients. In the end, more investigation is required to improve our comprehension of the specific processes by which alcohol influences aSAH outcomes and to create focused therapies meant to enhance recovery in this high-risk group.

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