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Research Paper

Pattern of Electrolyte Imbalances in the Subacute Phase of Spontaneous Subarachnoid Hemorrhage-: A Single Tertiary Health Institutional Experience in Imo State Nigeria

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Abstract

Introduction

Subarachnoid hemorrhage triggers off metabolic changes that are centrally mediated leading to the syndrome of inappropriate ADH secretion and the secretion of atrial natriuretic factor. These substances can lead to alteration in electrolyte balances within the body system resulting to a heightened secondary injury mechanism. This study aims to study the pattern of these changes.

Objectives

To know how subarachnoid hemorrhage affects the electrolyte balance and the impact of electrolyte imbalances on patient survival.

Materials and Methods

This is a prospective study of patients with subarachnoid hemorrhage managed at a tertiary hospital from 2017 till 2023. Fifty patients were studied. The patients included those with intracerebral and intraventricular hemorrhage. The study excluded patients with traumatic brain injury and patients without brain imaging. The management of these patients were uniform. The GCS on admission were checked. The electrolytes were checked on admission - in the acute phase. The GCS and the electrolytes levels were correlated with the outcome. The outcomes were survival or mortality.

Results

Total number of patients studied was 50, male 24 (48%), females 26 (52%), Table 1. The age range was 27 to 86, . Mean age was 56. The minimum age was 27, the maximum age was 86. The median age of presentation was 53 years while the mode was 49 years. The median GCS was 9.

Those with normal Na^+ on admission were 31(62%), high 1(2%), low 18(36%), Table 5. Those with normal K^+ were 31(62%), high 2(4%), low 17(34%) Table 6. Normal Cl^- 36(72%) high 1(2%) and low 13(26%), Table 7. On the outcome, 27(54%) survived and 23(46%) died, Table 8. Death occurred more in the older age group 60 and above.

The relationship between normonatremia and outcome was p = 0.262, not statistically significant. The relationship between hyponatremia and outcome was p=0.624 not statistically significant.

The relationship between hypernatremia and outcome was 0.120, not statistically significant. The relationship between Na^+ and outcome 0.995 which is not statistically significant.

The relationship between hyperkamia and outcome was 0.187, not statistically significant. The relationship between hypokalemia and outcome was 0.330 which is more than 0.05, so not statistically significant. The relationship between normochloremia and outcome was 0.144 which is greater than 0.05, so not statistically significant. The relationship between hypochloremia and outcome was 0.835 which is not statistically significant.

Discussion

Fifty patients were prospectively enrolled in this study. Hyponatremia was the most common electrolyte imbalance. It did not worsen the outcome. Although less common, hypernatremia was associated with poor outcome. This is in total agreement with the work done by Maysam Alimohamadi et al in which fifty-three patients were prospectively enrolled. Hyponatremia was the most common electrolyte imbalance among the patients but did not worsen the outcome. Although less common, hypernatremia in the subacute phase was significantly

associated with poor outcome. [4]
In the study by Qureshi et al it was shown that hyponatremia is more common that hypernatremia (30% vs. 19%) after aneurysmal SAH. None of them was associated with symptomatic vasospasm. Hypernatremia was significantly associated with poor outcome, and a positive correlation was detected between higher serum sodium levels and poor GOS at 3 months after the ictus. Chandy et al found that hyponatremia was associated with increased risk of cerebral vasospasm after aneurysmal SAH.[5]
Conclusion
This study shows that majority of the patients with normal electrolyte levels in subarachnoid hemorrhage survived. Low electrolyte levels did not correlate with poor outcome. The GCS on admission was the only factor that correlated with poor outome. The major predictors of poor outcome were size and location of the hemorrhage, presence of co-morbidities. Efforts should be intensified to tackle these factors before and during the hemorrhage to improve outcome.

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I. Introduction

Subarachnoid hemorrhage triggers off metabolic changes that are centrally mediated leading to syndrome of inappropriate ADH secretion (SIADH) and the secretion of atrial natriuretic factor. These substances can in turn lead to alteration in electrolyte balances within the body system resulting to a heightened secondary injury mechanism..

This study looked at 50 patients with subarachnoid hemorrhage, their electrolytes and GCS on admission and their correlation with outcome . While the electrolyte pattern is the focus of this study, the GCS is considered since the GCS at presentation is known to have direct correlation with the possible outcome (mortality). The outcome in this study centers on survival or mortality.

Patients were recruited prospectively into the study. Patients with brain trauma were excluded from the study. The GCS and electrolyte levels were checked on admission.

The results showed normal levels of electrolytes in most of the survivors but the association was not significant. Hyponatremia was the commonest electrolyte imbalance, but this did not correlate with poor outcome. Other factors like age, size and location of the hemorrhage, rebleed, hypertension and other co-morbidities plays a prominent role in survival.

The finding that more patients with normal electrolytes survived buttresses the fact that such patients need regular electrolyte monitoring. Importantly, life-style modification and surgical means of reducing raised ICP, the size of the hemorrhage, cerebral vessel embolisation etc should be made to be routine.

Objectives:
To know how subarachnoid hemorrhage affects the electrolyte imbalances and the impact of electrolyte imbalances and presenting GCS on patient survival.

II. Materials and Methods

This is a prospective study of the patients with subarachnoid hemorrhage managed at a tertiary hospital from 2017 till 2023. Fifty patients were studied. The patients included those with intracerebral and intraventricular hemorrhage. The study excluded patients with traumatic brain injury and patients without brain imaging. Statistical analysis was with Statistical Package for the Social Sciences, SPSS. The imaging techniques were brain CT scan and brain MRI. The management of the patients were uniform. They were managed in the ICU and had intravenous fluids, nasogastric feeding when needed, antihypertensives, diuretics, broadspectrum antibiotics, intranasal O2 when necessary, DVT prophylaxis. The GCS on admission were checked. The electrolytes were checked on admission - in the acute phase. The abnormalities were promptly corrected and followed up. The electrolytes studied were sodium Na+, potassium K+, and chloride. The GCS and the electrolytes levels were correlated with the outcome. The outcomes were survival or mortality.

III. Results

Total number of patients studied was 50, male 24 (48%), females 26 (52%), Table 1. The age range was 27 to 86, Table 1. Mean age was 56. The minimum age was 27, the maximum age was 86. The median age of presentation was 53 years while the mode was 49 years. The median GCS was 9.Table 1. Eclampsia accounted for the subarachnoid hemorrhage in the younger age group.

The GCS ranged from 3-15. The mean GCS was 9. The minimum was 3 and maximum 15. GCS of 7, 9 and 15 accounted for the highest frequency, 12% each,Table 4, Figure 3. Followed by 10 (10%), 6, 8, 14 each 8%. GCS of 3 and 12 were 6% each, 5 and 11 were 4% each and 13 was 2%. Table 2, Figure 3.

Those with normal Na^+ on admission was 31(62%), high 1(2%), low 18(36%), Table 5. Those with normal K^+ were 31(62%), high 2(4%), low 17(34%) Table 6. Normal Cl^- 36(72%) high 1(2%) and low 13(26%), Table 7. On the outcome, 27(54%) survived and 23(46%) died,

Electrolytes

20(40%) patients who had electrolyte imbalances on admission died. 15 (30%) patients who had normal electrolytes on admission survived. 10 (20%) patients with electrolyte imbalances on admission survived. 5 (10%) patients with normal electrolytes on admission died.

Females had lesser imbalance in Na+ but the association was not statistically significant, p= 0.826. Males had more K+ imbalance but the association was not statistically significant, p=0.832. Males had more Cl^- imbalance but the association was 0.200, not statistically significant. Males had better outcome p=0.096, but the association was not statistically significant.

The relationship between normonatremia and outcome was p = 0.262, not statistically significant. The relationship between hyponatremia and outcome was p=0.624 not statistically significant.

The relationship between hypernatremia and outcome was 0.120, not statistically significant. The relationship between $Na^+$and outcome 0.995 which was not statistically significant. The relationship between hyperkamia and outcome was 0.187, not statistically significant.

The relationship between hypokalemia and outcome was 0.330 which is more than 0.05, so not statistically significant. The relationship between normochloremia and outcome was 0.144 which is greater than 0.05, so not statistically significant. The relationship between hypochloremia and outcome was 0.835 which is not statistically significant.

The electrolyte imbalances detected on admission were instantly corrected and followed up. This highlights the fact that other factors like age, size of the hemorrhage, location of the hemorrhage, unilateral or bilateral hemorrhage, repeat hemorrhage, co-morbidities like obesity, hypertension, asthma, immunosuppression, played a more significant role in the outcome.

GCS

The outcome relationship between the GCS and the electrolytes (Na^+, K^+, and $Cl^{-)}$ shows p-value of 0.01, thus indicative of statistical significance. This supports the finding in this study that GCS was the only variable considered which has significant impact on the outcome. Figure 1 shows Frequency of Age, Figure 2 shows Frequency of Sex, Figure 3 shows Frequency of GCS, Figure 4 shows Frequency of Na^+, Figure 5 shows Frequency of K^+, Figure 6 shows Frequency of Cl^{-}

Key to the histogram

Figure 1 Bar chart depicting GCS and frequency, Figure 2 Histogram depicting Na^+ and frequency, Figure 3 Histogram depicting K^+ and frequency, Figure 4 Histogram depicting Cl^-

		Age	Sex	GCS	Na	K	Cl	Outcome
N	Valid	50	50	50	50	50	50	50
	Missing	0		0	0	0	0	0
Mean		55.8200	1.5200	9.0800	135.4200	3.7860	100.3600	1.4600
Std. Error of Mean		2.28119	.07137	.52515	1.02543	.28105	.73997	.07120
Median		53.0000	2.0000	9.0000	136.5000	3.5000	100.0000	1.0000
Mode		49.00	2.00	7.00[a]	140.00	3.50[a]	100.00	1.00
Std. Deviation		16.13045	.50467	3.71341	7.25087	1.98731	5.23240	.50346
Minimum		27.00	1.00	3.00	109.00	2.40	82.00	1.00
Maximum		86.00	2.00	15.00	161.00	17.00	113.00	2.00

Table 1. Shows the statistics of the variables.

Na^+

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	31	62.0	62.0	62.0
	High	1	2.0	2.0	64.0
	Low	18	36.0	36.0	100.0
	Total	50	100.0	100.0	

Table 3. Shows the Sodium level on admission.

K^+

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	31	62.0	62.0	62.0
	High	2	4.0	4.0	66.0

	Low	17	34.0	34.0	100.0
	Total	50	100.0	100.0	

Table 4. Shows the potassium level on admission

Cl^-

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	36	72.0	72.0	72.0
	High	1	2.0	2.0	74.0
	Low	13	26.0	26.0	100.0
	Total	50	100.0	100.0	

Table 5. Shows the electrolyte levels on admission

Outcome

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Survived	27	54.0	54.0	54.0
	Died	23	46.0	46.0	100.0
	Total	50	100.0	100.0	

Table 6. Shows the patient outcome.

Charts and Histograms

page3image53221888.jpg ¬

Figure 1 Bar chart depicting GCS and frequency.

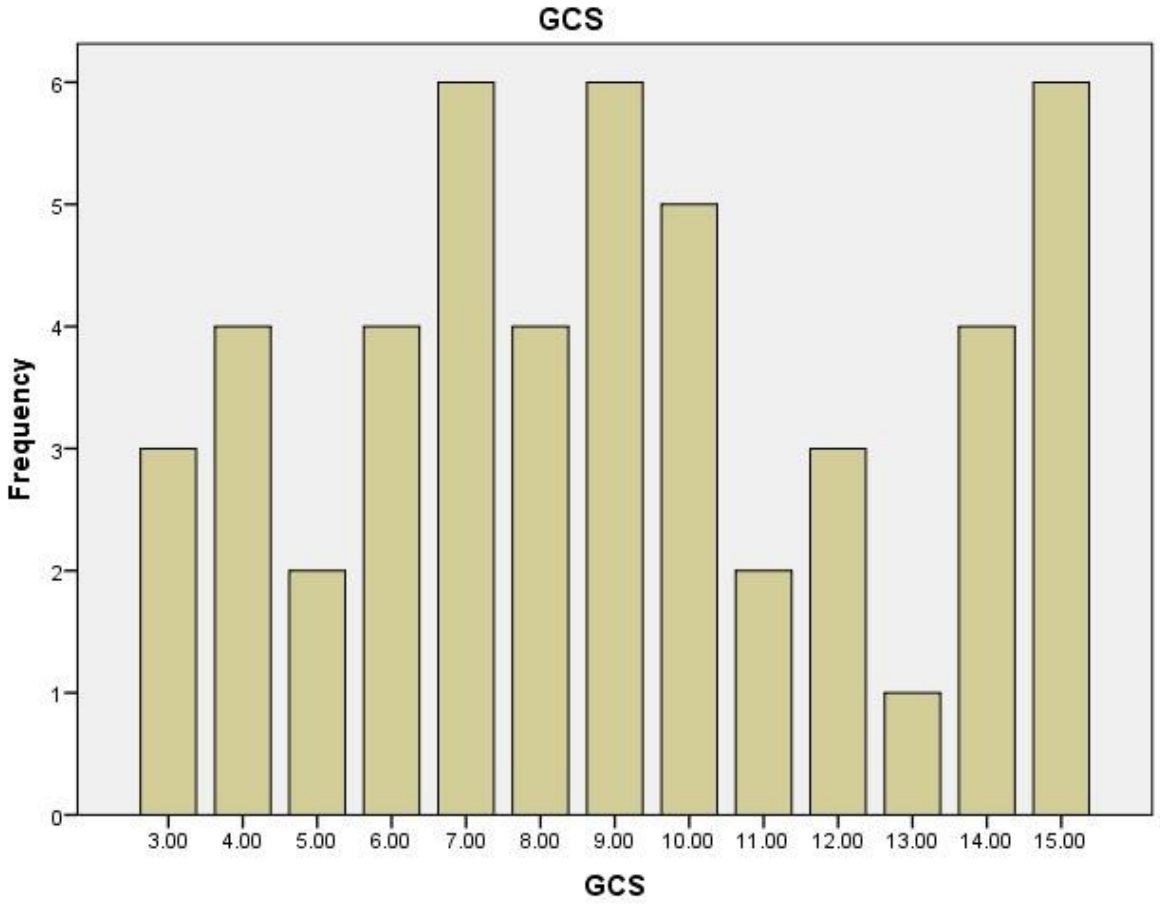

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Figure 2 Histogram depicting Na^+ and frequency

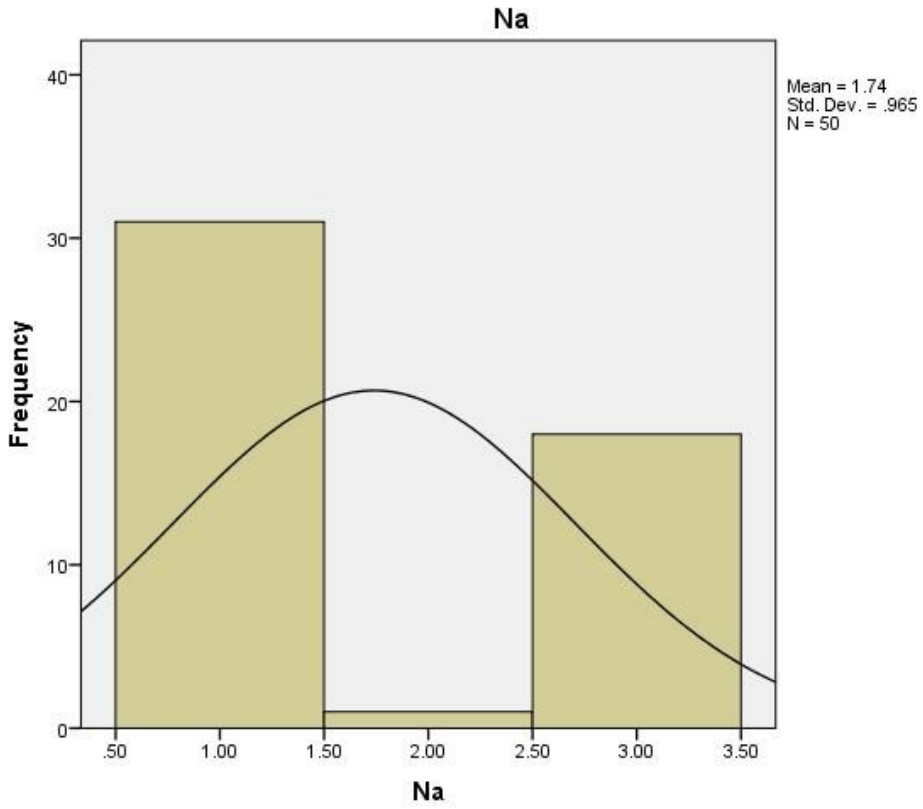

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Figure 3 Histogram depicting K^+ and frequency

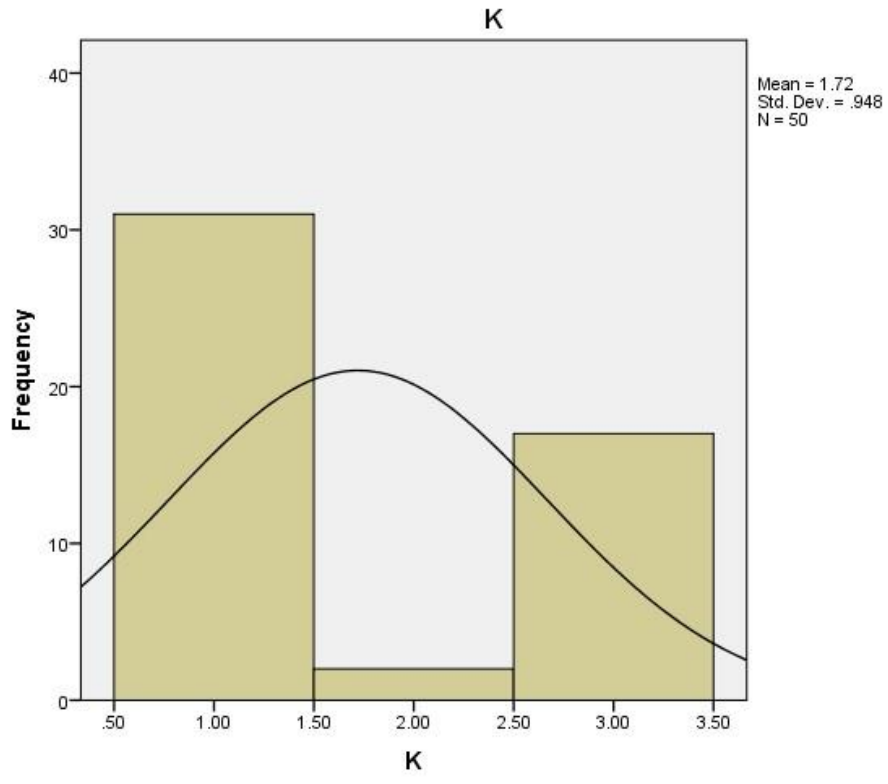

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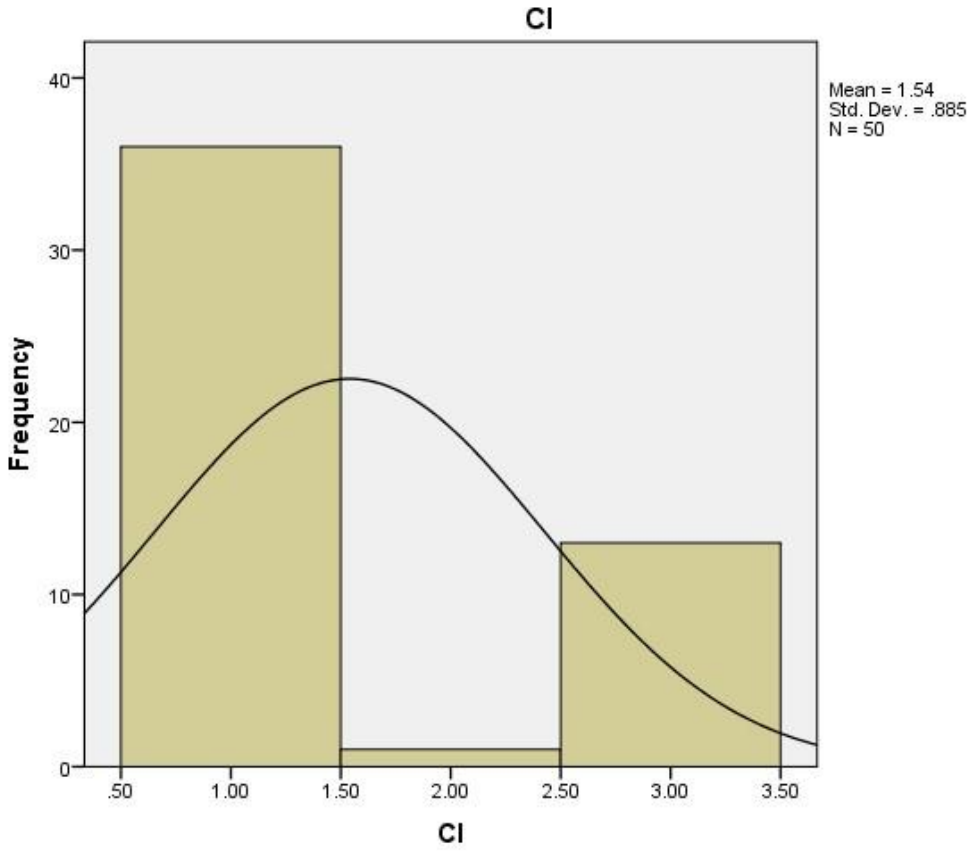
Figure 4, Histogram depicting Cl^-



IV. Discussion

In this study, normonatremia was seen in 62% of the patients and hypernatremia in 2%. Hyponatremia 36%. The level of hyponatremia seen in this study agrees with the normal level seen in spontaneous subarachnoid hemorrhage (SSAH), 20–40% of SAH patients. [1]

Although the main predictive factor of the outcome is the severity of neurologic morbidity. The studies on the pattern and importance of electrolyte disturbance in SAH patients, have reported conflicting results. Some of them have reported adverse effects of electrolyte abnormalities on the outcome of these patients, while some others did not find such relationship. [2]

Hyponatremia is usually caused by inappropriate secretion of antidiuretic hormone and free-water retention and/or excessive renal sodium excretion due to increased atrial natriuretic factor, so called 'cerebral salt wasting syndrome'. Intravascular volume depletion and sodium loss may increase the risk of DCI and infarction. Delayed cerebral infarction (DCI) is known to be present in about 30% of SSAH[3]. Thus the knowledge and early mitigation of its occurance or swift attenuation of this deleterous secondary injury mechanism in SSAH is helpful. In 124 WFNS grade IV and V patients, hyponatremia (serum sodium <135 mmol/l) developed in 63% of patients, caused by cerebral salt wasting syndrome in 55%. [4]

Fifty patients were prospectively enrolled in this study. Hyponatremia was the most common electrolyte. It did not worsen the outcome. Although less common, hypernatremia was associated with poor outcome. This is in total agreement with the work done by Maysam Alimohamadi et al, in which fifty-three patients were prospectively enrolled. Hyponatremia was the most common electrolyte imbalance among the patients in this study but did not worsen the outcome. Although less common, hypernatremia in the subacute phase was significantly associated with poor outcome. [5]

In the study by Qureshi et al, it was shown that hyponatremia is more common than hypernatremia (30% vs. 19%) after aneurysmal SAH. None of them was associated with symptomatic vasospasm. Hypernatremia was significantly associated with poor outcome, and a positive correlation was detected between higher serum sodium levels and poor GOS at 3 months after the ictus. Chandy et al. found that hyponatremia was associated with increased risk of cerebral vasospasm after aneurysmal SAH[6]

Our findings are also in agreement with Sherlock et al. In a retrospective study on 316 patients with aneurysmal SAH they found that hyponatremia was a common electrolyte imbalance among their patients (56%

overall prevalence) that caused longer hospital stay but did not affect the mortality rate. In their study, there was not a correlation between serum electrolytes levels in the acute phase and patient outcome 3 months after SAH. In the subacute phase, hyponatremia was associated with better clinical grades, less radiographic severity, and more favorable outcome, but hypernatremia was associated with worse clinical grades, more radiographic severity and less favorable outcome[7]

The Mean age was 56 years. The median age of presentation was 53 years while the mode was 49 years. The mean age of patients was 49 (range of 36-64) in the study done by Maysam Alimohamadi et al.[8]

Hypernatremia and hyponatremia in the subacute phase were associated with worse and better outcomes, respectively. In our study, the electrolyte imbalances detected on admission were instantly corrected and followed up. This highlights the fact that other observed factors like age, size of the hemorrhage, location of the hemorrhage, unilateral or bilateral hemorrhage, repeat hemorrhage, co-morbidities like obesity, diabetes, hypertension, asthma, immunosuppression, played a more significant role in the outcome.

This is in agreement with the findings by Alimohamadi et al. According to them, the causes of mortality and morbidity after aneurysmal SAH could be divided into two groups: Neurologic and Non-neurologic. The most important neurologic causes are rebleeding, vasospasm and hydrocephalus. Electrolyte imbalance, aspiration pneumonia, and venous thromboembolism were the most important nonneurologic causes. The mortality and morbidity rate increased with advancing age and more than one-third of the survivors had major neurologic affectations [9]. Those with normal K^+ were 31(62%), high 2(4%), low 17(34%) Table 6. Males had more K+ imbalance but the association was not statistically significant, p=0.832. The relationship between hyperkalemia and outcome was 0.187, not statistically significant.

The study by Rashmi Rekhaphunka et al showed that SSAH patients were prone to develop both hypokalaemia and hyponatremia with occurrence of hypokalaemia (40%) being more as compared to hyponatremia (26.7%)[10]. The relationship between hyperkamia and outcome was 0.187, not statistically significant. The probable cause of hypokalemiat in SSAH is the release of catecholamines. The circulating catecholamines stimulates Na+ - K+ ATPase by activation of beta2 adrenergic receptors.

Normal Cl^- 36(72%) high 1(2%) and low 13(26%), Table 7. On the outcome, normochloremia and outcome was 0.144 which is not statistically significant. The relationship between hypochloremia and mortality outcome was 0.835 which is not statistically significant.

V. Conclusion

This study shows that majority of the patients with normal electrolyte levels in subarachnoid hemorrhage survived. Low electrolyte levels did not correlate with poor outcome. The GCS on admission was the only factor that correlated with poor outome or mortality. The major predictors of poor outcome were size, location of the hemorrhage, rebleed and presence of co-morbidities. Efforts should be intensified to tackle these factors before and during the hemorrhage to improve outcome.

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