Incidence of Hyponatremia in the Neurotoxic Snake Bite

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ABSTRACT

Introduction: Common krait bite is a serious public health problem in India causing neurological features like ptosis, ophthalmoplegia and paralysis of respiratory muscles. Krait bite also causes hyponatremia, The exact mechanism of action is not still fully understood, it may be due to cerebral salt wasting syndrome. So, aim of the study was to determine the serum sodium levels in patients with krait bite to check the effect of neurotoxin on it and the occurrence of hyponatremia among these patients.

Material and Methods: This cross-sectional observational study was carried out over a period of six months which included 39 established cases of common krait bite patients admitted in Deben Mahata Government Medical College & hospital, Purulia. The blood and urine samples were collected from the 39 subjects and stored and the serum and urinary sodium levels, hematocrit and serum albumin levels were estimated and analyzed statistically.

Results: Out of these 39 patients, on the first day only 9 patients had hyponatremia, on second day total 18 patients had the same and on the third day a total of 23 patients developed hyponatremia. The mean sodium level on first day was 137.14 ± 44.21 mEq/L, on the second day was 129.14 ± 08.99 mEq/Land that on third day was123.35 ± 24.08 mEq/L. On 4th day of admission, only 5 patients were hyponatremic and the mean sodium level was 136.67 ± 27.81 mEq/L.The mean hematocrit, serum albumin and 24 Hrs urinary sodium excretion levels were also in increasing pattern till third day after which they became tend to be normal.

Conclusion: So, common krait bite can develop hyponatremia due to cerebral salt wasting syndrome which requires further studies to prove or disprove.

Keywords: Common Krait, Hyponatremia, Cerebral Salt Wasting Syndrome, Hematocrit, Serum Albumin, Urinary Sodium Excretion.

INTRODUCTION

Common krait i.e. *Bungarus caeruleus* is found throughout South Asia including India, in which, the presynaptic neurotoxins release acetylcholine at neuromuscular junction, preventing further release of transmitter leading to ptosis, ophthalmoplegia and paralysis of respiratory muscles.[1,2] Common krait bites typically occur at night and are also painless. Hence patients are typically caught unaware being still fast asleep.The neuromuscular paralysis in krait bite is characterized by progressive descending paralysis caused by β-bungarotoxins found in krait venom and this presynaptic neurotoxins with phospholipase A_2 activity is the major cause of paralysis.[5] This pre-synaptic action of the venom is irreversible causing resistance even with antivenom once paralysis developed.[6]

Krait bite also causes hyponatremia, the exact mechanism of action is not still fully understood.[7,8,9] It is thought that neurotoxins induced damage of the brain releases some natriuretic protein that act on kidney to excrete sodium leading to hyponatremia due to cerebral salt wasting syndrome.[10] So, aim of the study was to determine the serum sodium levels in patients with krait bite to check the effect of neurotoxin on it and the occurrence of hyponatremia among these patients.

MATERIAL AND METHODS

This cross-sectional observational study was conducted in Deben Mahata Government Medical College & hospital, Purulia. These tests were carried out at the Department of Biochemistry, Deben Mahata Government Medical College & hospital. This study was conducted from May, 2019 to October, 2019.

Study subject: 39 subjects with established krait bite were selected from age group 5 to 65 years.

a) Inclusion criteria

Both male and female patients according to convenience

b) Exclusion criteria

1) Patients with serious co-morbidities such as pulmonary or endocrine disease, hepatic or renal diseases.
2) Patients who use medicines causing hyponatremia before bite
3) Patients with diarrhea and vomiting.
4) Patients with active or chronic infection,
5) Patients with any kind of malignancies.

The subjects were screened and physically examined and information regarding demography, nature of illness were collected. Informed consent was taken from each of the participant. The study design was approved by institutional ethical committee.

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Laboratory investigations: The blood and urine samples were collected from the 39 subjects and stored at -20ºC before analyzing. The serum and urinary sodium levels, hematocrit and serum albumin levels were estimated by SFRI ISE 3000, CellTech 380 and ErbaChem 5x clinical chemistry semiautoanalyzer respectively.

STATISTICAL ANALYSIS

The data were compiled in MS excel and analyzed by different statistical methods. Data display was done by charts and tables. Data were described by proportion, mean, SD, range etc.

RESULTS

The average age of the participants was 37.64 ± 3.93 (mean ± SD) years with a range of 60 years. Out of 39 participants 11 were female and rest were male (Figure 1).

Table no 1 showed that the serum sodium levels were decreased from Day 1to Day 3 after that on Day 4 the serum sodium level became higher. This table no 1 also revealed that out of these 39 patients, on the first day only 9 patients had hyponatremia, on second day total 18 patients had the same and on the third day a total of 23 patients developed hyponatremia. Lastly on Day 4 only 5 patients remained as hyponatremic.

Table no 2 revealed that both serum albumin levels and hematocrit levels were increased from Day 1to Day 3 after that on Day 4 the serum sodium level became lower comparatively.

Table no 3 revealed that urinary Na^+ excretion levels also were increased from Day 1to Day 3 after that on Day 4 the serum sodium level became lower comparatively.

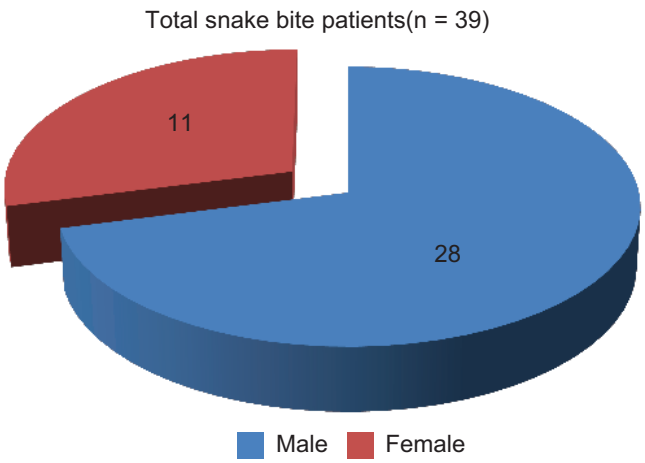

Figure-1: Distribution of snake bite patients according to sex

Day in hospital	Serum sodium level in mEq/L	Number of hyponatremic pateints
Admission day	137.14 ± 44.21	9
Day 2	129.14 ± 08.99	18
Day 3	123.35 ± 24.08	23
Day 4	136.67 ± 27.81	5

Table-1: Distribution of Sodium levels in different days in snake bite patients

Day in hospital	Serum albumin level in g/dL	Hematocrit level in %
Admission day	4.66 ± 0.76	53.76 ± 4.53
Day 2	5.43 ± 0.47	57.56 ± 7.05
Day 3	5.89 ± 0.36	62.86 ± 5.42
Day 4	5.47 ± 0.29	54.24 ± 8.43

Table-2: Distribution of serum albumin and hematocrit levels in different days in snake bite patients

Day in hospital	Urinary Na^+ excretion in mmol/24 hours
Admission day	234.65 ± 55.72
Day 2	297.78 ± 69.34
Day 3	314.87 ± 64.56
Day 4	229.17 ± 37.15

Table-3: Distribution of urinary Na+ excretion levels in different days in snake bite patients

DISCUSSION

Number of male snake bite patients was more than female counterpart, as they were more exposed in outside environment. Normal range of serum sodium is 135-155 mEq/L and the normal range of urinary sodium excretion is 41-227 mmol/24 hours.[9] Out of these 39 patients, on the admission day only 9 developed hyponatremia, on second day 18 developed the same and on the third day interestingly 23 developed hyponatremia. The mean sodium levels were in decreasing pattern since day 3 after it become higher. On 4th day of admission, only 5 patients were hyponatremic. This was may be due to removal of the neurotoxin from the body. The mean hematocrit, serum albumin and 24 Hrs urinary Na^+ excretion levels were also in increasing pattern till third day after which they became tend to be normal. The increasing levels of albumin, hematocrit and 24 Hrs urinary Na excretion along with hyponatremia suggestive of cerebral salt wasting syndrome, as the serum creatinine levels in all patients were within normal limits. The findings of our study were similar to Kumar Keyal N et al[10] as well as Hojer J et al.[8] The actual mechanism of development of hyponatremia in krait bite is unknown. It is thought that the neurotoxins damage of the brain by the neurotoxin ultimately releases natriuretic protein that can act on the kidney excreting excess sodium. To conclude, krait snakebite can develop hyponatremia and cerebral salt wasting which was evidence by raised in hematocrit and albumin levels.

CONCLUSION

So, krait snakebite can develop hyponatremia and cerebral salt wasting which was evidence by raised in hematocrit, albumin and 24 Hrs urinary sodium excretion levels. So, it should be evaluated and managed early as this can affect the patient outcome. The understanding of its pathophysiology, though, is still unclear and needs further investigations.

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