International Journal of Medical Research Professionals
P-ISSN: 2454-6356; E-ISSN: 2454-6364
DOI: 10.21276/ijmrp

Original Research Article.

Evaluation of Incidence of Hyponatraemia in Post-Neurosurgical Patients At a Tertiary Care Centre

G. Suresh Kumar[1], Yerukala Murali Krishna[2*]

[1]Associate Professor, Department of Nephrology,
Deccan College of Medical Sciences, Hyderabad, Telangana, India.
[2]Associate Professor, Department of Neurosurgery,
Mamata Medical College, Khammam, Telangana, India.

ABSTRACT

Background: Hyponatremia is a common finding in both the neurosurgical patient population and the inpatient population in general. This study was conducted to evaluate incidence of hyponatremia in post-neurosurgical patients.

Materials and Methods: A total of 100 patients scheduled to undergo neurosurgical procedures were enrolled. Complete demographic and clinical details of all the patients was obtained. A Performa was made and complete medical findings of all the patients was recorded. Routine biochemical and haematological examination of all the patients was done. Periodic cardiac monitoring was done. All the patients were shifted to ICU after neurosurgical procedures. Blood samples were obtained, and serum sodium profile was evaluated. Incidence and etiological profile of hyponatremia was evaluated. All the results were recorded in Microsoft excel sheet.

Results: A total of 100 patients with mean age of 45.8 years were evaluated. Among them, 66 percent were males while the remaining were females. Hyponatremia was seen in 15 percent of the patients. Out of these 15 patients with hyponatremia, 5 patients were symptomatic while the remaining 10 patients were asymptomatic. Syndrome of Inappropriate Antidiuretic Hormone Secretion, Cerebral salt wasting, Hypothyroidism and Adrenal insufficiency was seen in 46.67 percent, 33.33 percent, 13.33 percent and 6.67 percent of the patients respectively.

Conclusion: Hyponatremia represents a significant comorbidity in patients undergoing neurosurgery, necessitating careful consideration due to the variability in its treatment based on underlying causes. Furthermore, the implications of hyponatremia can substantially influence neurological outcomes.

Keywords: Hyponatremia, Neurosurgical.

***Correspondence to:**

Dr. Yerukala Murali Krishna,
Associate Professor,
Department of Neurosurgery,
Mamata Medical College,
Khammam, Telangana, India.

Article History:

Received: 08-10-2018, **Revised:** 03-11-2018, **Accepted:** 22-11-2018

Access this article online	
Website: www.ijmrp.com	Quick Response code
DOI: 10.21276/ijmrp.2018.4.6.090	

INTRODUCTION

Hyponatremia is a common finding in both the neurosurgical patient population and the inpatient population in general.[1,2] Subarachnoid hemorrhage, traumatic brain injury, spinal cord injury, meningitis, neurosurgical procedures and a multitude of other CNS-related pathologies have been shown to predispose neurosurgical patients to hyponatremia.[3]

Hyponatremia occurs when the kidneys are unable to effectively excrete an excess volume of water or when there is an overconsumption of water. The regulation of water intake is primarily governed by the thirst mechanism, which is activated by an increase in osmolality. Osmoreceptors situated in the hypothalamus detect this change and trigger the secretion of antidiuretic hormone (vasopressin) from the posterior pituitary gland. This hormone targets V2 receptors located on the basolateral side of the collecting duct cells, resulting in enhanced expression of aquaporins on the luminal surface of these cells. Consequently, this process facilitates increased water reabsorption and diminishes the sensation of thirst. Hypernatremia related to diabetes insipidus (DI) is perhaps the most anticipated electrolyte abnormality to occur after transsphenoidal surgery, but hyponatremia has in fact been shown to occur with greater frequency and result in a greater degree of patient morbidity in some series.[4,5]

Indeed, hyponatremia in general, whether adequately treated or not, is associated with a considerable degree of morbidity, mortality and healthcare-related economic burden.[6-10] This study was conducted to evaluate incidence of hyponatremia in post-neurosurgical patients.

MATERIALS AND METHODS

The study was conducted to evaluate incidence of hyponatremia in post-neurosurgical patients. A total of 100 patients scheduled to undergo neurosurgical procedures were enrolled. Complete demographic and clinical details of all the patients was obtained. A Performa was made and complete medical findings of all the patients was recorded. Routine biochemical and haematological examination of all the patients was done. Periodic cardiac monitoring was done. All the patients were shifted to ICU after neurosurgical procedures. Blood samples were obtained and serum sodium profile was evaluated. Incidence and etiological profile of hyponatremia was evaluated. All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software.

Table 1: Incidence of hyponatremia in post-neurosurgical patients

Hyponatremia	Number	Percentage
Present	15	15
Absent	85	85
Total	100	100

Table 2: Aetiological profile of hyponatremia

Aetiology	Number	Percentage
Syndrome of Inappropriate Antidiuretic Hormone Secretion	7	46.67
Cerebral salt wasting	5	33.33
Hypothyroidism	2	13.33
Adrenal insufficiency	1	6.67
Total	15	100

RESULTS

A total of 100 patients with mean age of 45.8 years were evaluated. Among them, 66 percent were males while the remaining were females. Hyponatremia was seen in 15 percent of the patients. Out of these 15 patients with hyponatremia, 5 patients were symptomatic while the remaining 10 patients were asymptomatic. Syndrome of Inappropriate Antidiuretic Hormone Secretion, Cerebral salt wasting, Hypothyroidism and Adrenal insufficiency was seen in 46.67 percent, 33.33 percent, 13.33 percent and 6.67 percent of the patients respectively.

DISCUSSION

Disorders of sodium balance are common after transsphenoidal surgery.[11] The mechanism underlying this predisposition is not entirely clear, but some authors have suggested that it may be attributable to the aberrant release of hormones (e.g., ADH from the neurohypophysis, cortisol from the adenohypophysis) or abnormal sympathetic hypothalamic outflow occurring as a result of surgical manipulation of the gland itself, the stalk or the hypothalamus.[11] Fisher[12] and others[13] described a now classic, triphasic trend of serum sodium abnormalities seen after transection of the infundibular stalk in both animals and humans (in which DI is initially seen, followed by hyponatremia related to SIADH several days later and a final phase consisting of DI, which often persists). Indeed, much of the early literature suggests that post-transsphenoidal hyponatremia (particularly when arising in a stereotypically delayed fashion, 5–7 days after surgery) is predominantly due to SIADH.[14-17] The study was conducted to evaluate incidence of hyponatremia in post-neurosurgical patients. A total of 100 patients with mean age of 45.8 years were evaluated. Among them, 66 percent were males while the remaining were females. Hyponatremia was seen in 15 percent of the patients. Out of these 15 patients with hyponatremia, 5 patients were symptomatic while the remaining 10 patients were asymptomatic. Syndrome of Inappropriate Antidiuretic Hormone Secretion, Cerebral salt wasting, Hypothyroidism and Adrenal insufficiency was seen in 46.67 percent, 33.33 percent, 13.33 percent and 6.67 percent of the patients respectively. Sivakumar V et al[18] evolved a practical management protocol for neurosurgical patients with hyponatremia and natriuresis, based on their blood volume status and hematocrit. Twenty-one patients with hyponatremia and natriuresis and 3 control patients were studied. Patients with hyponatremia were categorized on the basis of their hematocrit, central venous pressure, and total blood volume. Group A consisted of patients with hypovolemia and anemia (16 patients); Group B patients had hypovolemia but no anemia (5 patients); Group C included those with hypervolemia (0 patients). Patients in Groups A and B received isotonic saline (> 50 ml/kg/d) and oral salt (12 g/d). Additionally, those in Group A were transfused with 500 ml of whole blood. The end points in the study were 72 hours after entry or two consecutive serum sodium values of > 130 mEq/L, whichever was earlier. Hyponatremia was corrected in all the patients within 72 hours. They conclude that most neurosurgical patients with hyponatremia and natriuresis have hypovolemia, with or without anemia. Fluid and salt replacement and a blood transfusion rather than fluid restriction often results in the correction of the hyponatremia. Their findings offer indirect evidence to support the hypothesis that in most of these patients, hyponatremia is caused by cerebral salt wasting syndrome, rather than the syndrome of inappropriate secretion of antidiuretic hormone. Rahman M et al[19] identified the determinants for the development of hyponatremia and which management strategies provided the best outcomes. A multidisciplinary panel in the areas of neurosurgery, nephrology, critical care medicine,

endocrinology, pharmacy, and nursing summarized and classified hyponatremia literature scientific studies published in English from 1950 through 2008. The panel's recommendations were used to create an evaluation and treatment protocol for hyponatremia in neurosurgical patients at the University of Florida. Hyponatremia should be further investigated and treated when the serum sodium level is less than 131 mmol/L (class II). Evaluation of hyponatremia should include a combination of physical examination findings, basic laboratory studies, and invasive monitoring when available (class III).[19] Arieff A.I et al observed development of severe hyponatremia after elective surgery in 15 previously healthy women who subsequently either died or had permanent brain damage. The preoperative serum sodium concentration was recorded at 138 mmol per liter. All patients successfully emerged from anesthesia; however, approximately 49 hours post-surgery, when the mean plasma sodium level had dropped to 108 mmol per liter, all 15 individuals experienced grand mal seizures, which were followed by respiratory failure necessitating intubation. At this juncture, the average urinary sodium concentration and osmolality were 68 mmol per liter and 501 mOsm per kilogram, respectively, indicating a likely inappropriate secretion of antidiuretic hormone. In 10 out of the 15 patients, an acute cerebrovascular event was suspected, resulting in a delay in therapeutic intervention and the performance of various diagnostic procedures, including CT scans, cerebral angiography, and open-brain biopsies. The total postoperative fluid retention was measured at 7.5 liters, and when efforts to correct the serum sodium levels commenced, the correction rate was observed to be less than 0.7 mmol per liter per hour. Histological examinations of the brain in five patients yielded no conclusive findings, and autopsy, brain biopsy, or CT scans did not reveal any signs of central pontine myelinolysis. Seven patients regained consciousness after their serum sodium levels were elevated to 131 mmol per liter; however, coma reoccurred within two to six days, ultimately resulting in either death or a persistent vegetative state.[20]

CONCLUSION

Hyponatremia represents a significant comorbidity in patients undergoing neurosurgery, necessitating careful consideration due to the variability in its treatment based on underlying causes. Furthermore, the implications of hyponatremia can substantially influence neurological outcomes.

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Source of Support: Nil. **Conflict of Interest:** None Declared.

Cite this article as: G. Suresh Kumar, Yerukala Murali Krishna. Evaluation of Incidence of Hyponatraemia in Post-Neurosurgical Patients At a Tertiary Care Centre. Int J Med Res Prof. 2018 Nov; 4(6): 376-78. DOI:10.21276/ijmrp.2018.4.6.090