Scholars Journal of Applied Medical Sciences (SJAMS)
Abbreviated Key Title: Sch. J. App. Med. Sci.
ISSN 2320-6691 (Online)
ISSN 2347-954X (Print)

A Unit of Scholars Academic and Scientific Society, India
www.saspublishers.com

Medicine

Study of Effects and Complications of Hypertonic Saline in Cases of Hyponatremia

Dr. Harishchandra*, Chaudhari R
Assistant Professor, Department of Medicine, Dr. DY Patil Medical College and research center, Maharashtra, India

Original Research Article

***Corresponding author**
Dr. Harishchandra

Article History
Received: 02.04.2018
Accepted: 12.04.2018
Published: 30.04.2018

DOI:
10.36347/sjams.2018.v06i04.036

Abstract: In case of hyponatremia when amount of sodium in fluids outside cells drops, water moves into cells to balance the levels. This causes swelling of cells. Although most cells can handle this swelling, brain cells cannot, because skull bones confine them. Brain swelling causes most of the symptoms of hyponatremia**.** The sample was collected from, patients admitted in NTU/ICU/CCU and various medical wards of ruby hall Clinic, Pune. The sample size was determined with the help of expert. Diagnosis wise 16 CCF patients, 13 cirrhosis patients, 11 patients of vomiting/diarrhea,9 patients with trauma/blood loss, 2 patients with sepsis and 1 patient each with Hypothyroidism, Pancreatitis and CKD are having urinary sodium ≤ 20 meq/L. In case of urinary sodium >20 meq/L, 34 patients are of SIADH, 17 are of CSW, 13 of Low intake, 12 of CKD, 10 of Diuretic excess, 7 of ARF, 2 of Hypothyroidism and 1 of Vomiting and diarrhea. Shrinkage of the brain triggers demyelination of pontine and extrapontine neurons that can cause neurological dysfunction, including quadriplegia, pseudobulbar palsy,seizures, coma, and even death. Hepatic failure, potassium depletion, and malnutrition increase the risk of this complication.

Keywords: hyponatremia, Brain swelling, pseudobulbar palsy,seizures, coma.

INTRODUCTION

In case of hyponatremia when amount of sodium in fluids outside cells drops, water moves into cells to balance the levels. This causes swelling of cells. Although most cells can handle this swelling, brain cells can not, because skull bones confine them.Brain swelling causes most of the symptoms of hyponatremia [1].

Clinically significant hyponatremia is relatively uncommon and is nonspecific in its presentation; therefore, the physician must consider the diagnosis in patients presenting with vague constitutional symptoms or with altered level of consciousness. Irreparable harm can befall the patient when abnormal serum sodium levels are corrected too quickly or too slowly. The physician must have a thorough understanding of the pathophysiology of hyponatremia to initiate safe and effective corrective therapy. The patient's fluid status must be accurately assessed upon presentation, as it guides the approach to correction [1].

Though clearly not indicative of the overall prevalence internationally, hyponatremia has been observed in as high as 42.6% of patients in a large acute care hospital in Singapore and in 30% of patients hospitalized in an acute care setting in Rotterdam[3-5]. So, in our study we tried to study effects and complications of hypertonic saline in cases of hyponatremia [3].

MATERIALS AND METHODS

The sample was collected from, patients admitted in NTU/ICU/CCU and various medical wards of ruby hall Clinic, Pune. The sample size was determined with the help of expert.

Inclusion criteria

All cases of hyponatremia who was admitted with hyponatremia and who developed it during first 4 days of admission to hospital

Hyponatremia will be considerd with serum sodium level < 130 mmol/lit
Normal sodium level in this hospital is 130-150 mmol/lit
These cases will be divided into groups like
1.130-120 mmol/lit -mild
2.120-110 mmol/lit-moderate
3.110-100mmol/lit -severe
4.<100 mmol/lit -critical
Patients plasma osmolality will be calculated by formula-

2 Na^++BSL/18+ BUL/2.8
(mg/dl) (mg/dl)

Affording patients measured osmolality will be recorded and then difference between measured and calculated osmolality willbe noted and cause for same will be found. Patient's clinical picture will be correlated with level of hyponatremia and clinical picture after correction will be studied and also other factors affecting clinical picture will be noted.

Exclusion criteria

All cases that develop hyponatremia after 4 days of hospital admission will be excluded

Definitions

1-Hyponatremia- serum sodium level <130 meq/L
2- Organic neurological disease: proved by CT scan, MRI and CSF studies
Cases meeting the above inclusion criteria have been selected randomly and studied.
Once selected in study following information about the patient has been collected:
The detail history was collected.

The data was processed using the statistical tools like Microsoft Office Excel 2010, SPSS 17 software, t test, coefficient of relation (r value),Wilcoxon Z value, Mean , Standard deviation etc.

OBSERVATIONS AND RESULTS

Table-1: Comparison of before and after treatment Sodium level in study group-

Parameter	Before Rx (n=150)		After Rx (n=150)		t Value	P Value
	Mean	SD	Mean	SD		
Na (mmol/lit)	118.52	6.01	132.67	4.36	34.51	<0.0001

In this study Mean sodium level before treatment is 118.52 with standard deviation of 6.01.

After treatment Mean sodium level is 132.67 with standard deviation of 4.36. t value is 34.51 and p value is <0.0001. This indicates that after treatment serum sodium value increased significantly.

Table-2: Comparison of before and after treatment GCS score in study group-

Parameter	Before Rx (n=147)		After Rx (n=147)		Wilcoxon Z Value	P Value
	Mean	SD	Mean	SD		
GCS	14.13	1.29	14.96	0.23	6.94	<0.0001

Table-3: Correlation between GCS on admission and before treatment Na in study group-

Correlation between GCS on adm.&	r Value	P Value
Before treatment Na	0.86	<0.0001

For 'GCS on admission' and 'before treatment sodium level' r value is 0.86 and p value is < 0.0001. This indicates there is positive correlation between these two variables. As serum sodium increases GCS value also increases.

Table-4: Correlation between GCS on discharge and after treatment Na in study group-

Correlation between GCS on discharge &	r Value	P Value
After treatment Na	0.16	<0.05

For 'GCS on discharge' and 'after treatment sodium level' r value is 0.16 and p value is <0.05. This indicates that there is positive correlation between these two variables. As serum sodium increases GCS value also increases (Table-4).

In this study 54 patients i.e. 36% are having urinary sodium ≤20 meq/L and remaining 96 patients i.e.64 % are having urinary sodium > 20 meq/L (Table-5).

Table-5: Urine sodium level wise distribution of cases in study group-

Urine Sodium level (mmol/lit)	**No of cases**	**Percentage**
≤20	54	36
>20	96	64
Total	150	100

Table-6: Urine sodium level and diagnosis in study group

Diagnosis	Urine Na≤20mmol/lit	Urine Na>20mmol/lit	Total
SIADH	0	34	34
CSW	0	17	17
CCF	16	0	16
Cirrohosis	13	0	13
Low intake	0	13	13
CKD	1	12	13
Omiting / Diarrhoea	11	1	12
Diuretic excess	0	10	10
Trauma/ Blood loss	9	0	9
ARF	0	7	7
Hypothyroidism	1	2	3
Sepsis	2	0	2
Pancreatitis	1	0	1
Total	54	96	150

Diagnosis wise 16 CCF patients , 13 cirrhosis patients, 11 patients of vomiting/diarrhea,9 patients with trauma/blood loss, 2 patients with sepsis and 1 patient each with Hypothyroidism, Pancreatitis and CKD are having urinary sodium ≤ 20 meq/L.

In case of urinary sodium >20 meq/L, 34 patients are of SIADH, 17 are of CSW, 13 of Low intake, 12 of CKD, 10 of Diuretic excess, 7 of ARF, 2 of Hypothyroidism and 1 of Vomiting and diarrhea.

Table-7: Treatment received wise distribution of cases in study group-

Treatment received	No of cases	Percentage (n=150)
3% Normal saline	17	11.3
Fluid restriction	71	47.3
0.9% Normal saline + salt	99	66

In this study 17 patients i.e.11.3% received 3% Normal saline, 71 patients i.e. 47.3% patients received Fluid restriction and 99 patients i.e. 66% received 0.9% NS +Salt.

Table-8: Study of patients who received 3% NS as treatment-

Parameters	Patient who received 3% NS (n=17)	
	Before treatment	After treatment
	Mean ± SD	Mean ± SD
Sr Na	107.06 ± 5.97	129.29 ± 5.53
GCS	11.82 ± 1.18	14.73 ± 0.59
Sr. Osmolality	229.32 ± 10.68	
Urine Na	98.06 ± 35.08	
CPM	3	

Out of 150 patients 17 patients received 3 % NS. 3 % NS is given mainly for patients with CSW i.e.14 patients. 2 patients with SIADH and 1 patient with trauma/blood loss received 3% NS. For them Mean sodium level before treatment is 107.06 with SD of 5.97, and after treatment is129.29 with SD of 5.53. Mean GCS before treatment is 11.82 with SD of 1.18, and after treatment is14.73 with SD of 0.59. Mean serum osmolality is 229.32 with SD of 10.68, Mean urinary sodium is 98.06 with SD of 35.08.

In this study most common symptom found to be Fatigue, nausea, vomiting, diarrhea in 77 patients. Out of 77 patients,

38 are having serum sodium level between 121 to 130 meq/L, 38 are having serum sodium level between 111 to 120 meq/L and 1 patient is having serum sodium level between 101 to 110 meq/L.

36 patients found to be asymptomatic and all of them are having serum sodium level between 121 to 130 meq/L. 16 patients are having headache as a

prominent symptom and all of them are having serum sodium level between 111 to 120 meq/L.

18 patients developed drowsiness, out of 18 patients, 14 are having serum sodium level between 101 to 110 meq/L, 2 are having serum sodium level between 111 to 120 meq/L and remaining 2 are having serum sodium level ≤100 meq/ L.

3 patients developed convulsions and all of them are having serum sodium level ≤ 100 meq/L.

So for convulsions Mean sodium level is 98.67 with SD of 1.16 and Mean GCS 10.67 is with SD of 1.16.

For drowsiness Mean sodium level is 108.28 with SD of 3.91 and Mean GCS 11.78 is with SD of 1.44.

For headache Mean sodium level is 114.44 with SD of 1.31 and Mean GCS 13.31 is with SD of 0.87.

For Fatigue, nausea, vomiting and diarrhea Mean sodium level is 120.04 with SD of 2.62 and Mean GCS is 14.46 with SD of 0.86.

For Asymptomatic patients Mean sodium level is 123.86 with SD of 0.80 and Mean GCS is 15 with SD of 0.

DISCUSSION

In patients of intracranial lesion with hyponatremia, signs of dehydration, decreased CVP, increased blood urea level, increased urine output, normal or increased uric acid level and marked increase in urine sodium go in favor of CSW. Those patients with no signs of dehydration, normal or increased CVP, decreased urine output and decreased uric acid levels, SIADH should be suspected.

Before treatment

In this study 5 patients (3.3%) are having sodium level below 100 meq/lit i.e. critical hyponatremia. 15 patients (10%) are having sodium level between 101 to 110 meq/L i.e. severe hyponatremia.56 patients(37.3%) are having sodium level between 111 to 120 meq/L i.e. moderate hyponatremia and remaining 74 patients(49.3%) are having sodium level between 121 to 130meq/L i.e. mild hyponatremia.

After treatment

47 patients out of 150 (31.3%) still were having sodium level between 121 to 130meq/L and remaining 103(68.7%) patients were having sodium level > 130meq/L. (table 10)

Mild and Moderate hyponatremia are more common than severe and critical hyponatremia. In this study Mean sodium level before treatment is 118.52 with standard deviation of 6.01.

After treatment Mean sodium level is 132.67 with standard deviation of 4.36. t value is 34.51 and p value is <0.0001. This indicates that after treatment serum sodium value increased significantly.

Wilcoxon Z value is 6.94 and p value is <0.0001. This indicates that after treatment GCS value increased significantly [5].

3 % NS is given mainly for patients with CSW i.e.14 patients. 2 patients with SIADH and 1 patient with trauma/blood loss received3% NS. Fluid restriction is given mainly for patients with CCF, CKD, ARF, Cirrhosis, Sepsis and some SIADH patients.

0.9% NS and Salt is given mainly for patients with CSW, Vomiting and Diarrhea, Diuretic excess, Trauma/blood loss, Pancreatitis, Low intake, Hypothyroidism and some patients with SIADH.

Cerebral salt wasting syndrome is characterized by extracellular fluid depletion. The treatment regimen should therefore consist of administration of water and salt. On the other hand, SIADH is associated with slight hypervolemia and should be treated by fluid restriction [6,7].

Out of 150 patients 17 patients received 3 % NS.3 % NS is given mainly for patients with CSW i.e.14 patients. 2 patients with SIADH and 1 patient with trauma/blood loss received 3% NS.

For them Mean sodium level before treatment is 107.06 with SD of 5.97, and after treatment is129.29 with SD of 5.53. Mean GCS before treatment is 11.82 with SD of 1.18, and after treatment is14.73 with SD of 0.59.

Mean serum osmolality is 229.32 with SD of 10.68, Mean urinary sodium is 98.06 with SD of 35.08. Out of these 17 patients 3 patients developed CPM (Central Pontine Myelinolysis). Overt manifestations of hyponatremia are treated with hypertonic saline, whereas symptomatic hypovolemia associated with hyponatremia without overt symptoms is usually treated with isotonic saline [8].

All 3 patients received 3% NS as a treatment outside and then referred to our hospital. All 3 were having increase in sodium >12 meq/L in 24 hours.

On discharge 2 patients were having GCS 14 and 1 patient was having GCS 13. Newer interventions should keep in mind. Such data should be collected at

large scale and by using data mining techniques, it should further validate [9,10].

Even in symptomatic patients, the sodium level sholud not be raised by more than 12mEq/L in first 24 hours and by more than 18mEq/L in the first 48 hours, in order to avoid osmotic demyelination syndrome(ODS)[6]. In all cases ,close and frequent monitoring of serum sodium and electrolytes is mandatory until sodium levels and symptoms subsides.[11]

Shrinkage of the brain triggers demyelination of pontine and extrapontine neurons that can cause neurological dysfunction, including quadriplegia, pseudobulbar palsy,seizures, coma, and even death. Hepatic failure, potassium depletion, and malnutrition increase the risk of this complication.

CONCLUSION

Shrinkage of the brain triggers demyelination of pontine and extrapontine neurons that can cause neurological dysfunction, including quadriplegia, pseudobulbar palsy,seizures, coma, and even death. Hepatic failure, potassium depletion, and malnutrition increase the risk of this complication.

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