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Evaluation of Term Newborn Patients With Hypernatremic Dehydration

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What is already known on this topic?

- *Hypernatremic dehydration (HDH) is a medical emergency with high mortality and morbidity rate in the neonatal period.*
- *Malnutrition with breast milk is the primary etiology.*
- *In HDH, careful fluid and electrolyte therapy is required to prevent morbidity and mortality, although there is no consensus on this issue.*

What this study adds on this topic?

- *Impaired renal function, deep metabolic acidosis, and need for dialysis increase mortality.*
- *Convulsions are a presenting complaint, and can also be seen during treatment.*

ABSTRACT

Aim: We aimed to evaluate the demographic, clinical, and laboratory findings and the management of newborns with hypernatremic dehydration (HDH).

Materials and Methods: A total of 85 term newborns with serum sodium (Na) levels higher than 145 mEq/L who admitted to our hospital between January 2011 and December 2018 were included in this study.

Results: Among all cases, 54.1% were female infants with the mean birth weight, weight loss ratio, and median age at diagnosis of 3095 ± 540 g, 13.6 ± 10%, and 8 (2–24) days, respectively. The most common presenting complaints were breastfeeding difficulties (90.5%), fever (63.5%), decreased urination (43.5%), jaundice (22.3%), and convulsion (15.3%). The mean sodium and potassium, and median blood urea and creatinine levels on admission were 167.9 ± 13.4 mEq/L, 5.4 ± 2.8 mmol/L, 213 mg/dL (11–476 mg/dL), and 2.4 mg/dL (0.52–9.96 mg/dL), respectively. There was metabolic acidosis in 67% and acute renal failure in 74.4% of patients, while peritoneal dialysis was performed in 12 of them. There was a positive correlation between weight loss ratio and admission age, serum urea, and creatinine levels; there was a negative correlation between weight loss and blood pH. Eight patients died (9.4%).

Conclusions: In our study, serum urea, creatinine, potassium, metabolic acidosis levels, convulsion, and dialysis requirements at the time of admission of the newborns with HDH were found to be higher in those who died compared to those who survived. Convulsion was a presenting complaint, and it was also observed during the treatment.

Keywords: Hypernatremia, mortality, newborn

INTRODUCTION

Dehydration due to hyperbilirubinemia and nutritional problems with the early discharge strategies of newborns are the most common reasons for re-admissions to neonatal services.[1] In these babies, if sufficient fluid support cannot be provided in the postnatal period, HDH may develop, leading to severe neurological problems, permanent sequelae, and death.[2]

Serum sodium (Na) level between 146 and 149 mEq/L is considered mild, while Na level ≥ 150 mEq/L is considered severe hypernatremia. In the literature, the frequency of mild hypernatremia is reported as 0.9%, and the frequency of severe hypernatremia is reported as 0.6%.[2,3,4] Serious clinical and neurological effects in these patients are usually observed when the serum Na level exceeds 160 mEq/L.[5,6]

Hypernatremia in newborns develops as a result of a disturbance in water balance rather than sodium balance. Total body Na in these patients may be increased, decreased, or normal. As a preventive mechanism for hypernatremia, urine is concentrated and a strong thirst

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is created. However, hypernatremia may develop more easily in newborn babies due to the insufficient capacity to concentrate urine and their inability to express thirst. Insufficient water intake, increased water loss, and Na-rich fluid are reported as the 3 main causes of hypernatremia.[7]

In newborns with normal urination, 7% weight loss in the first week of life is considered normal. At the end of the first week, they start to gain weight again and reach birth weight on the 10th day. Rapid weight loss or weight loss of more than 7% in the first week of life should be evaluated by the pediatrician.[8] In this period, the limit value of weight loss in babies who are fed only breast milk is accepted as 10%.[2,9]

Insufficient breast milk is common in newborn babies in the first days of life. In the first few postnatal days, mothers' low amount of breast milk, especially if primipara, and the mothers' lack of knowledge and skills about breastfeeding, may cause breast milk deficiency. Among the other causes of breast milk deficiency, cesarean delivery, mothers with low education level, breastfeeding incompatibility between mother and baby, insufficient breastfeeding, wrong breastfeeding techniques, and rarely, nipple problems, have been reported.[2,9,10]

When HDH is not treated appropriately and promptly, it can lead to severe consequences such as jaundice, nutritional deficiency, insufficient weight gain, cerebral edema, convulsions, venous thrombosis, intracranial hemorrhage, disseminated intravascular coagulation, acute renal failure, brain damage, and death.[11] Globally, there is limited data on the treatment, management, and results of HDH patients. There is no consensus on fluid therapy applications in these patients.[6,12] However, in the literature, many experts report that the correction should be made in more than 48 hours and the reduction rate of serum Na should not exceed 0.5-1 mEq/L/h.[6,12,13]

In our study, demographic characteristics, laboratory findings, and treatment results of 85 newborns with HDH followed in our clinic were analyzed in light of the literature.

METHODS

In this study, patients who were hospitalized in the neonatal clinic of our hospital between 2011 and 2018, who were term born, and who were diagnosed with HDH were evaluated retrospectively. The demographic characteristics, clinical and laboratory findings, follow-up results, and complications of 93 term newborn patients with HDH were evaluated. Factors that can reduce mortality, morbidity, and dehydration frequency were reviewed. Ethics committee approval for the study was obtained from the ethics committee of the University of Health Sciences Gazi Yaşargil Training and Research Hospital (April 28, 2020/457).

The mode of delivery, gender, birth weight, gestational week, weight at presentation, age at presentation, physical examination findings, amount of weight loss, serum Na values, renal function tests, cranial, renal, and cardiac imaging results, discharge times, and treatments were recorded from the file records. Those diagnosed with a congenital malformation, metabolic disease, birth trauma, perinatal asphyxia, intrauterine infection, and diabetes insipidus were excluded from the study. The study continued with 85 patients who met the criteria.

The treatment of the patients was individually adjusted according to clinical and laboratory data. In the physical and laboratory examinations, patients with indications of dehydration such as tachycardia, low blood pressure, decrease in peripheral pulse fullness, change in skin color, cold skin, prolonged capillary refill time (>3 seconds), in addition to changes in consciousness, oliguria, and metabolic acidosis, received loading therapy according to the serum Na level. The patients with serum Na levels of < 175 mEq/L received 0.9% NaCl solution as loading therapy, and patients with a serum Na level of > 175 mEq/L were treated with a dose of 20 mL/kg by adjusting a liquid containing 15 mEq/L less NaCl than the patient's serum Na level. After the loading therapy (including those who did not require loading therapy), body free water deficit after hospitalization (BFWD (L) = [(Patient's Na value/145 mEq/L) − 1] × Body fluid ratio × kg) and replacement volume (Replacement Volume (L) = Body free water deficit (L)) ×{1 ÷ [(1 − Replacement fluid Na mEq/L ÷ 154 mEq/L)]} were calculated. The maintenance fluid appropriate for the patient's age was given with the replacement volume in a way that would reduce the Na level by a maximum of 12 mEq in 24 hours. The calculated amount of fluid was given orally to babies who were fed completely orally. The lack of fluid was compensated intravenously in babies who could not be fed completely orally and completely intravenously in babies who could not be fed orally. Patients were followed up with weight and electrolyte control at 6- to 24-hour intervals according to serum Na levels.

As the criteria for renal failure, creatinine level should be at least 1.5 times the upper limit appropriate for the age and urine amount < 0.5 mL/kg/h despite the 6-12 hours of fluid support.[14]

A pH value of blood gas below the 7.35 was considered as metabolic acidosis. Peritoneal dialysis was performed in patients who presented with very high serum Na levels and did not respond to fluid loading for 6-12 hours, such as anuria, renal failure, resistant metabolic acidosis, and resistant hyperpotassemia (> 6.5 mmol/L despite medical therapy).

Statistical Analysis

Statistical Package for the Social Sciences (SPSS) version 23.0 (IBM SPSS Corp.; Armonk, NY, USA) was used for statistical analysis. For descriptive data, homogeneous data with normal distribution were given as mean ± standard deviation and heterogeneous data with anormal distribution were given as median (lower limit-upper limit). The Mann–Whitney U-test was used to evaluate the relationship between measurable variables that were not compatible with normal distribution, and Fisher's exact test was used for examinations of categorical variables. Median values were taken into account. Spearman's correlation test was used for the analysis of continuous variables. $P < .05$ was considered significant.

RESULTS

A total of 85 term newborns, 46 of whom were girls (54.1%), who were born at a median of 39 weeks (37-42), and who

Table 1. Demographic Characteristics and Laboratory Findings of the Cases

Gender (Female/male)	**46/39**
Gestational age† (weeks)	39 (37-42)
Mode of Delivery, (CS/NSVD) (*n*)	8/77
Birth weight (g)*	3095 ± 540
Nutrition	BM: 62, BM+formula: 19, only formula:4
Admission weight (g)*	2657 ± 473
Admission age (day)†	8 (1-24)
Weight loss (%)*	13.6 ± 10
Hemoglobin (g/dL)*	17.8 ± 2.8
CRP (mg/L)†	0.8 (0-99)
Glucose (mg/dL)†	70 (22-494)
Sodium (mEq/L)*	167.9 ± 13.4
Potassium (mmol/L)*	5.4 ± 2.8
Urea (mg/dL)†	213 (11-476)
Creatinine (mg/dL)†	2.4 (0.52-9.96)
0-24 hours sodium drop (mEq/L)*	9.5 ± 5.1
Mean of sodium drop (mEq/L)*	8.3 ± 23.1
Duration of treatment (day)†	3 (1-6)
Length of stay (day)†	8 (1-97)
Mortality (lost/total)	8/85

†Data are given as median (lower-upper limit); *Data are given as mean ± standard deviation.
BM, breast milk.

were hospitalized with the diagnosis of HDH were included in the study. It was determined that 8 (9.5%) patients were born by cesarean section (C/S) and 77 (90.5%) patients were born by normal spontaneous vaginal delivery (NSVD). A total of 62 (72.9%) of the cases were fed only breast milk, 19 (22.4%) with a formula supplement to breast milk, and 4 (4.7%) with formula only. The birth weight of the cases was calculated as 3095 ± 540 g, and the weight at presentation was calculated as 2657 ± 473 g. Mean serum Na values were found as 167.9 ± 13.4 mEq/L (146-200) in the laboratory examinations of the patients on admission. The demographic characteristics and laboratory findings of the patients are given in Table 1.

When the patients were evaluated according to the month of admission, it was found that 53 cases (62.4%) were admitted between May and October, when the temperatures were high, and 32 cases (37.6%) were admitted in the remaining months. In the analyses performed, no correlation was found between the season in which the patient was born and the levels of serum Na, urea, and creatinine on admission, mortality, convulsion, admission day, and weight loss ($P > .05$).

The average percentage of weight loss of the patients at presentation was found to be 13.6 ± 10%. There was a positive correlation between weight loss and admission day serum urea and creatinine levels, and a negative correlation with blood pH level (r and P values respectively, $r = 0.46$ and $P = .001$, $r = 0.41$ and $P = .001$, $r = 0.32$, and $P = .011$, $r = -0.27$ and $P = .019$).

The most common complaints at presentation were decreased sucking in 77 (90.5%), fever in 54 (63.5%), inability to urinate in 37 (43.5%), and jaundice in 19 (22.3%). Other admission complaints were seizure in 13 (15.3%) patients, vomiting in 4 (4.7%) patients, bruising in 3 (3.5%), and diarrhea and restlessness in 1 (1.1%) patient each.

In the follow-up, urinary tract infection was detected in 4 patients, sepsis in 2 patients, hydronephrosis in 3 patients, moderate ventricular septal defect in 1 patient, atrial septal defect in 2 patients, grade I-II intraventricular bleeding in 2 patients, adrenal hemorrhage in 1 patient, and thrombosis of the right tibial artery in 1 patient. Venous sinus thrombosis in 1 patient, hypothyroidism in 1 patient, and ABO incompatibility in 1 patient accompanied HDH.

Fifty-eight (68.2%) patients had metabolic acidosis. Peritoneal dialysis was performed in 12 (14.1%) patients with resistant metabolic acidosis, renal failure, refractory hyperkalemia, and anuria. Convulsions were detected in 12 (14.1%) patients at admission and in 4 (4.7%) patients at follow-up. All of the convulsions in the follow-up were seen in the first 24 hours of treatment. The need for dialysis and the creatinine level at presentation were determined as the variables indicative of convulsion ($P < .05$).

Renal ultrasonography (USG) was performed in 71 of the patients. While the examination of 35 (47.2%) patients was normal, 26 patients had increased echogenicity of the kidney bilaterally, 3 patients had hydronephrosis, 3 patients had nephrocalcinosis, 2 patients had pelvicaliectasis, 1 patient had an adrenal hemorrhage, and 1 patient had a renal cyst.

Cranial USG was performed in 73 patients during follow-up. While no pathological findings were detected in 67 of the patients (91.7%), grade I-II intraventricular bleeding was present in 2 patients, periventricular leukomalacia in 1 patient, perinatal asphyxia sequela in 1 patient, ventriculomegaly in 1 patient, and venous sinus thrombosis in 1 patient.

Echocardiographic examination was performed in 44 patients, for various reasons. While ventricular septal defect was found in 1 of the patients, atrial septal defect was found in 2 patients. On echocardiography, 41 (93%) patients were evaluated as having normal or temporary neonatal circulation. In the follow-up, 15 (17.6%) patients had bradycardia. In the cardiological examination, all patients had sinus bradycardia and it was observed that the bradycardia improved in the follow-up.

As complications, 64 patients (75.2%) had acute renal failure, 4 had seizures during follow-up, 2 had intraventricular (grade I-II) bleeding, 1 patient had an adrenal hemorrhage, 1 patient had thrombosis of the right tibial artery and 1 patient had venous sinus thrombosis.

We lost a total of 8 (9.4%) patients, 4 of whom were within the first 24 hours. All of our patients, except 1, had acute renal failure and resistant metabolic acidosis. Peritoneal dialysis was performed in 7 of them. There was sepsis in 1 of the patients who died, and grade I-II intracranial hemorrhage in another. In the analyses, while the need for dialysis and more convulsions were more commonly determined in patients who died, the K, urea, creatinine, and 24th hour K levels at the time of admission were found to be significantly higher than in patients

Table 2. Comparison of Data of Living and Deceased Patients

Parameter	Living	Deceased	*P*
Gender (female/male)	41/36	6/2	.28[a]
Type of delivery (C/S-NSVD)	8/69	0/8	.33[a]
Nutrition (BM/BM+formula)	56/21	6/2	.94[a]
Weight loss (%)*	13.6 ± 10.1	14.3 ± 12.1	.64[b]
Admission date†	8 (2-24)	8.5 (1-12)	.39[b]
Convulsion (yes/total)	11/77	5/8	.001[a]
Need for dialysis (yes/total)	5/77	7/8	.000[a]
Admission Na (mEq/L)*	166.6 ± 12.6	178.3 ± 20.3	.08[b]
Admission K (mmol/L)*	5.2 ± 1.2	6.7 ± 1.6	.001[b]
Admission Urea (mg/dL)*	218 ± 125	293 ± 199	.05[b]
Admission Creatinine (mg/dL)†	2.1 (0.52-9.96)	4.7 (0.59-8.1)	.042[b]
Admission Glucose (mg/dL)†	70 (22-494)	83 (8-187)	.26[b]
24th hour Na (mEq/L)*	158 ± 12.5	167.5 ± 17.6	.07[b]
24th hour K (mmol/L)*	4.6 ± 0.77	6.08 ± 2.2	.02[b]
24th hour Urea (mg/dL)*	182 ± 115	259 ± 191	.38[b]
24th hour Creatinine (mg/dL)†	1.5 (0.42-7.8)	4.5 (0.97-7.4)	.06[b]
0-24 hours Na drop (mEq/L)*	9.66 ± 5.2	7 ± 2.9	.27[b]
Mean of Na drop (mEq/L)*	8.3 ± 2.9	7.8 ± 5.4	.66[b]

[a]Fisher's exact test; [b]Mann–Whitney *U*-test; †Data are given as median (lower-upper limit); *Data are given as mean ± standard deviation.
C/S, cesarean section; NSVD: normal spontaneous vaginal delivery.

who survived. Although the Na levels of the patients at the time of admission were higher in the patients who were lost than in the surviving patients (178.3 ± 20.3, 166.6 ± 12.6 mEq/L, respectively), the difference was not statistically significant. The comparison of the data of living and deceased patients is presented in Table 2.

DISCUSSION

If HDH is not recognized in time and not treated correctly, it may lead to serious complications such as acute renal failure, cerebral edema, hydrocephalus, convulsion, disseminated intravascular coagulation, intracranial hemorrhage, peripheral and central venous thrombosis, and death.[10,15] In our study, the demographic, clinical, and laboratory characteristics of 85 term neonatal patients with HDH who were hospitalized in our clinic were reviewed in light of the literature.

HDH is a serious problem that occurs with a decrease in fluid intake as a result of poor nutrition in newborns who are fed exclusively with breast milk, and its frequency has gradually increased in recent years.[2,8] In the literature, its frequency in newborns fed with breast milk has been reported to be between 1.9 and 4.1%.[4,16,17] It has been reported that weight loss is over 10% in most of the cases. Weight loss of more than 7% in the first week of life is considered pathological weight loss.[2,8] Close clinical and laboratory follow-up is recommended for newborns with serum Na levels > 150 mEq/L.[7] The American Academy of Pediatrics reports that the sucking in the first 2-3 days of newborns discharged in the early period and the adequacy of breastfeeding in babies with more than 7% weight loss in the first 7 days should be evaluated.[2,18] In a retrospective study, it was reported that seasons are not a risk factor in HDH.[15] Similarly, in this study, although 62% of the cases applied between May and October, no seasonal relationship was found in the analyses.

Studies have reported that weight loss in neonatal HDH cases ranges between 8 and 30% generally due to insufficient fluid and calorie intake, and there is a positive relationship between weight loss and serum Na, urea, and creatinine levels. In our study group, similar to the reported studies, there was a positive correlation between weight loss and serum urea and creatinine levels on the day of admission, and a negative correlation with blood pH level.

It is known that feeding with breast milk in the early postnatal period is critical for successful breastfeeding.[18] In the literature, the reasons for insufficient breast milk have been reported as a mother with a low level of education, a primipara mother, insufficient breastfeeding, incompatibility between mother and baby, wrong breastfeeding techniques, and rarely, nipple problems.[17,19] Although it has been stated that C/S delivery delays breastfeeding and increases the incidence of dehydration, there are also studies arguing that there is no relationship between mode of delivery and serum Na level, dehydration, and weight.[2,17,20] Erdeve et al.[21] showed in their study that C/S delivery was a risk factor in the development of hypernatremia. Unlike our study, the C/S rate was very high. No relationship was found between delivery mode and weight loss and serum Na levels in our cases. Regardless of the mode of delivery, babies should be fed with breast milk immediately after birth. Although early discharge practices reduce hospital costs, it is responsible for problems arising from malnutrition and HDH, together with insufficient breastfeeding training for mothers.[1,11]

Patients with HDH may present with some complaints and symptoms or be asymptomatic.[13,22] In the early stages of hypernatremia, patients may be asymptomatic, because intracellular fluid reaches the extracellular region. However, symptoms occur when the patient is dehydrated.[22] In the literature, it has been observed that the cases show symptoms between 3-21 days.[2,11,13,15,23] In our study, the mean age at presentation was calculated as 8 days, in line with the literature.

The presenting complaints of the patients are mostly reported as fever, jaundice, decreased sucking, and less urination.[20] In a study conducted in our country, it was shown that 27.6% of patients with jaundice had dehydration and 14.6% had weight loss > 10%.[24] In the cases in our study, the most common complaint was a decrease in sucking. Other admission complaints were fever, inability to urinate, jaundice, seizure, vomiting, bruising, diarrhea, and restlessness. The coexistence of fever and hypernatremia is common in patients presenting with weight loss. It is thought that only fever, without any other finding, is associated with dehydration rather than infection in

newborns.[11,25] In our study, while the infection was detected in 1 of 35 babies who presented with fever, it was observed that their fever returned to normal when they were hydrated.

The main purpose of treatment is to replace the circulating volume depletion and to avoid a rapid reduction in serum Na. The osmolarity of brain cells is increased in HDH. Since the rapid correction of plasma osmolarity does not provide the necessary time to reduce osmolarity in the brain, it will lead to problems such as brain edema, brain damage, convulsion, and death. Therefore, the slow correction of high serum Na levels in the treatment will prevent such serious complications as it will decrease the osmolarity in central nervous system cells in balance with serum osmolarity. There is no consensus on the rate at which it is safe to lower serum Na in the treatment. However, a daily weight gain up to 5%, and a decrease in serum Na level of 0.5-1 mEql/L/h with a maximum of 10-15 mEq/L daily, are reported as safe.[20] The time taken to normalize the Na level in our patients and the rate of fall were calculated following the literature.

The major complications of HDH are acute renal failure, disseminated intravascular coagulation, vascular complications, intracranial hemorrhage, convulsion, brain damage, and death.[2,17] The most common complication in this study was found to be acute renal failure.

Brain damage associated with HDH occurs due to hyperosmolar state (decrease in brain volume, bleeding, thrombosis) and inappropriate rehydration therapy.[13,17] Although it has been reported that convulsions in these patients usually developed while correcting Na serum level with fluids containing low Na levels and due to rapid correction during treatment,[17] 12 of our patients presented with the complaint of seizure. Convulsions were detected in 4 of the treated patients within the first 24 hours. The need for dialysis and high levels of creatinine at presentation were determined as factors indicative of convulsions. The fact that the vast majority of our patients with seizures had a seizure at the time of admission suggests that our patients were admitted to the hospital in a clinically very delayed state.

The most tragic and important complication of HDH is undoubtedly death. While mortality was not reported in many studies in the literature,[2,9,11] it was reported as 1.2% in the study of Unal et al.[17] and 6.5% in the study of Akgün et al.[14] Again, in the study conducted by Chouchane et al.,[26] mortality was reported as 11.2% in patients with severe HDH, whose mean age was 6.5 months, while the 76.2% had acute renal failure and 97.2% had metabolic acidosis. In this study, 8 (9.4%) of our patients were lost. All of our patients, except one, had acute renal failure and resistant metabolic acidosis. Peritoneal dialysis was performed in 7 of these patients. Also, one of the patients who died had sepsis and 1 had grade I-II intraventricular bleeding. The presence of metabolic acidosis, the need for dialysis, having a convulsion, K, urea, creatinine at the time of admission, and 24th hour K levels were found to be significantly higher in patients who died. Although the Na levels of the patients at the time of admission were higher in the patients who were lost than in the surviving patients, the difference was not statistically significant. In the studies that did not report mortality in the literature, it is noteworthy that the cases were admitted to the hospital earlier and the clinical and laboratory conditions were better. Although our mortality was higher than the rates reported in the literature, it was observed that the patients presented with delay and very severe clinical and laboratory findings. Four of the 8 patients who died were lost in the first 24 hours after admission.

In conclusion, HDH is a serious and life-threatening condition in the neonatal period with a high incidence in developing countries. In our study, in HDH, serum urea, creatinine, K, metabolic acidosis levels, seizure, and need for dialysis at the time of admission were found to be higher in deceased patients than in surviving patients. Convulsion can be seen at the time of admission or during treatment .

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