Hypernatremic dehydration in children

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GRADUATE THESIS

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**Abbreviations**

AKI  Acute kidney injury
BF   Breastfeeding
HND  Hypernatremic dehydration
IVH  Intraventricular hemorrhage
MA   Metabolic acidosis
ORS  Oral rehydration solution
SP   Salt poisoning
SDS  Standard Deviation Score
TBS  Total body sodium
TBW  Total body weight
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1. Abstract

**Objective:** The objective of this paper was to provide a literature review on hypernatremic dehydration (HND) in children and the role of Charts, which are based on weight loss with Standard Deviation Score (SDS), in its management. Before this condition can be treated, it is important to assess the weight loss by the patient. Charts based on weight loss with SDS have been established by a case-control study to diagnose HND (1). The vast majority of children with already present or developing HND fell below the -1 SDS line at day 3, the -2 SDS line at day 4, and the -2.5 SDS line at day 5. Therefore, charts for weight loss can be helpful in early diagnosis of HND and in its prompt management plan.

**Methods:** The key terms of hypernatremic dehydration in children, management of hypernatremic dehydration and breast-feeding were searched on Pubmed and the literature available under these terms was reviewed and summarized in this paper, and a clinical case is presented to demonstrate HND due to problem in initiation of breastfeeding.

**Discussion:** The problem of HND is recurrent in the hospital setting but also represents a common issue in breastfed infants. It is normal over the 1st week of life for the neonate to lose as much as 7% of its birth weight through normal diuresis, but then should start to gain weight thereafter. Rapid weight loss > 7% of birth weight is the most obvious sign of neonatal dehydration due to HND with symptoms of decreased bowel movements (2,3), decreased urination, dry mucous membranes, sunken fontanel, poor skin turgor, and lethargy (2). Therefore, routine weight monitoring is universally proposed for the early detection and management of HND. The poor initiation of breastfeeding can lead to development of HND in infants, which besides the clinical assessment of the newborn also requires the educational preventive measures.

**Conclusion:** The monitoring of weight loss in children along with clinical presentation can help in diagnosis of HND, and subsequently proper management. To avoid serious morbidity and mortality, all breastfed infants should receive regular follow-up by health-care workers.

**Keywords:** Hypernatremic dehydration, metabolic acidosis, initiation of breastfeeding, management of hypernatremic dehydration, charts for weight loss monitoring
2. Sažetak

**Cilj:** Cilj ovog rada je prikazati pregled literature o hipernatrijemijskoj dehidraciji (HND) u djece, i ulogu Charts, koje su utemeljene na gubitku težine sa skorom standardne devijacije (SSD), u liječenju. Prije započinjanja liječenja bitno je procijeniti gubitak težine u pacijenta. Chart s utemeljene na gubitku težine sa SSD su potvrđene u case control studiji za dijagnozu HND (1). Velika većina djece sa već prisutnim ili razvijajućim HND pada ispod -1 SSD linije u trećem danu, -2 SSD linije u četvrtom i -2,5 SSD linije u petom danu. Stoga, charts za gubitak težine mogu biti od pomoći u ranom dijagnosticiranju HND te u brzom stvaranju plana postupanja.

**Metode:** Ključne riječi o HND u djece, menadžmentu HND i dojenju su pretražene na PubMed-u i literatura dostupna pod tim riječima je pregledana i sažeta u ovom radu. Dodatno, prezentiran je klinički slučaj za demonstraciju HND uslijed problema u započinjanju dojenja.

**Diskusija:** HND kao problem je ponavljajući u bolničkom okruženju, ali također predstavlja čest problem u dojenčadi. Za novorođenčadi je u prvom tjednu života normalno izgubiti do 7% porođajne težine kroz normalnu diurezu, međutim nakon toga ubrzo trebaju dobiti na težini. Brzi gubitak više od 7% porođajne težine je najočitiji znak novorođenačke dehidracije zbog HND-a sa simptomima smanjenog broja stolica (2,3) smanjenog mokrenja, suhih sluznica, uvučenih fontanela, smanjenog turgora kože i letargije (3). Stoga, rutinsko mjerenje težine se univerzalno predlaže za ranu detekciju i liječenje HND-a. Sporo započinjanje dojenja može voditi do razvoja HND-a u novorođenčadi, što pored kliničke procjene novorođenčadi također zahtijeva educacijske preventivne mjere.

**Zaključak:** Praćenje gubitka težine u djece zajedno sa kliničkom prezentacijom može pomoći u ranoj detekciji HND-a, a shodno tome i adekvatnom liječenju. Da bi se izbjegli ozbiljan morbiditet i mortalitet, sva dojenčad trebaju biti adekvatno i redovno praćena od strane zdravstvenih radnika.

**Ključne riječi:** Hipernatrijemijska dehidracija, metabolička acidoza, započinjanje dojenja, liječenje hipernatrijemijske dehidracije, charts za praćenje gubitka težine
3. Introduction

Hypernatremic dehydration (HND) is a common and potentially life-threatening condition in children. It is defined by a serum level of sodium greater than or equal to 145 mmol/L. We can see the abnormal ranges of serum sodium in the Table 1 below (4). The mild hypernatremia will present in the range between 146 mmol/L and 149 mmol/L, moderate between 150 mmol/L and 169 mmol/L, and severe over 170 mmol/L.

<table>
<thead>
<tr>
<th>Definition</th>
<th>Serum (Na⁺), mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyponatremia</td>
<td>&lt;135</td>
</tr>
<tr>
<td>Normal</td>
<td>135-145</td>
</tr>
<tr>
<td>Mild hypernatremia</td>
<td>146-149</td>
</tr>
<tr>
<td>Moderate hypernatremia</td>
<td>150-169</td>
</tr>
<tr>
<td>Severe hypernatremia</td>
<td>&gt;170</td>
</tr>
</tbody>
</table>

Table 1. Classification of serum sodium concentration abnormalities.

HND is a particular form of acute dehydration and constitutes a medical emergency requiring a rapid diagnosis for adequate and quick management. It is characterized by a deficit of total body water (TBW) relative to total body sodium (TBS) levels due to either loss of free water, or excessive administration of hypertonic sodium solutions. It is common in infants. Net water loss accounts for most cases of hypernatremia. Hypertonic sodium gain usually results from clinical interventions at the hospital settings or accidental sodium loading.

In the pediatric population, newborns and infants are most affected because of their dependence on caretakers for water and on mothers for breastfeeding (BF). BF is the most complete and balanced nutrition, it contains antibodies, enzymes, hormones and all the necessary nutrients in ideal proportions (5). Adequate breast milk intake depends on several interrelated stages such as normal mammary development (mammogenesis), unimpeded initiation of lactation (lactogenesis), sustained ongoing milk synthesis (galactopoiesis), and effective milk removal. Milk removal depends on effective maternal and infant BF techniques, combined with an intact milk-ejection reflex, and total daily milk intake depends on the frequency and duration of feeds (6). Successful and exclusive BF has important benefits, to mothers, infants and society as a whole, both in the developed and developing countries. However, in some cases, successful initiation BF seems to fail due to inadequate latching, milk production or intake (7). This may cause HND in the newborn.
Neonates born prematurely or with critical illness such as those in coma are also at a great risk. A possible association of intraventricular hemorrhage (IVH) with hypernatremia and/or high sodium intake has been suggested in preterm infants. IVH is the most important cause of mortality and long-term developmental impairment in preterm infants (8). In developing countries where gastroenteritis is a recurrent problem at the population scale, HND must always and at any time be closely considered. Childhood diarrhea caused by rotavirus infection often presents with watery stools, which is associated with the body’s conservation of sodium loss via stools (9).

4. Pathophysiology and etiology

HND represents a deficit of water in relation to the body’s sodium stores, which can result from a net water loss or a hypernatremic sodium gain. Water loss exceeding sodium loss as seen in diarrhea is one of the mechanisms leading to this condition. It is common in developing countries where gastroenteritis is a common problem. In patients with infective diarrhoea, undigested proteins and carbohydrates reach the colon, where bacteria metabolize them to small and osmotically active molecules that draw water, but not sodium, from the plasma into the colon; this makes the plasma hypernatremic (10). This also develops because the child does not have access to water or cannot drink properly due to neurologic impairment, or emesis. This mechanism also includes the problem of initiation or effectiveness of BF seen in young primiparous mothers. The Table 2 (1) below shows a study that has established a correlation between cases of hypernatremia in breastfed infants and high level of breast milk sodium as a cause.

<table>
<thead>
<tr>
<th>Case number</th>
<th>Breast milk sodium level (mmol/L)</th>
<th>Infant serum sodium level (mmol/L)</th>
<th>Neonate presenting on day of life</th>
<th>Maternal information</th>
<th>Gestational age</th>
<th>Clinical manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>33</td>
<td>163</td>
<td>4</td>
<td>20</td>
<td>Prime</td>
<td>Term</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>152</td>
<td>5</td>
<td>22</td>
<td>Prime</td>
<td>Term</td>
</tr>
<tr>
<td>3</td>
<td>56</td>
<td>158</td>
<td>4</td>
<td>32</td>
<td>Second gravid</td>
<td>Term</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>169</td>
<td>6</td>
<td>29</td>
<td>Prime</td>
<td>Term</td>
</tr>
<tr>
<td>5</td>
<td>88</td>
<td>186</td>
<td>7</td>
<td>24</td>
<td>Prime</td>
<td>Term</td>
</tr>
<tr>
<td>6</td>
<td>17</td>
<td>156</td>
<td>8</td>
<td>23</td>
<td>Prime</td>
<td>Term</td>
</tr>
<tr>
<td>7</td>
<td>26</td>
<td>160</td>
<td>4</td>
<td>22</td>
<td>Prime</td>
<td>Term</td>
</tr>
<tr>
<td>8</td>
<td>43</td>
<td>170</td>
<td>5</td>
<td>25</td>
<td>Prime</td>
<td>Term</td>
</tr>
</tbody>
</table>

Table 2. Hypernatremia in infants breastfed with high sodium-containing breast milk and their clinical manifestations.
The second possible mechanism is related to pure water depletion that we can see in diabetes insipidus. Those children have inappropriately diluted urine. Renal dysplasia and obstructive uropathy are also taken into consideration during management. Nephrogenic diabetes insipidus is a serious condition with large water losses in the urine and high risk of HND.

The third main mechanism is simply the consequence of salt poisoning that is frequently seen in a hospital setting resulting from correction of metabolic acidosis (MA) with sodium bicarbonate. Improperly prepared oral rehydration salt solution (ORS), such as preparation with incorrect proportions (11) often leads to this condition with a certain degree of severity related to inadequately high frequency of intake. Children become excessively thirsty, which often pushes the caregivers to administer more and more ORS to quench thirst and ultimately leads to more severe and extreme hypernatremia (12). Acute systemic salt poisoning (SP) produces CNS damage when brain cells become dehydrated after the acute osmotic shift of intracellular fluids to the extracellular space. Brain cell damage may also occur after idiogenic osmoles have been established and vigorous therapeutic hydration leads to cerebral edema (13). Fatal SP has occurred in children following the inadvertent substitution of table salt for sugar in infant formulas. Symptoms are generally seen within hours of oral ingestion and include seizures, obtundation, coma, and cardiorespiratory arrest. Serum sodium levels may be greater than 180 mEq/L. Such high levels of sodium create a large osmotic gradient between the extracellular fluid (ECF) and intracellular fluid (ICF), leading to extreme cellular dehydration and brain injury. However, to maintain blood pressure in hypernatremia, water shifts from the intracellular to extracellular space. Therefore, this mechanism not only makes the infants with hypernatremia less symptomatic, but also causes more dehydration before suitable medical intervention is sought (14). Autopsy studies of children dying from salt poisoning have shown capillary and venous congestion of the central nervous system, subarachnoid and parenchymal hemorrhage, and dural sinus occlusion (15).

As a result of increased extracellular sodium concentration, plasma tonicity increases. This increase in tonicity induces the movement of water across cell membranes out of the cells, causing cellular dehydration. Brain cells are especially vulnerable to complications resulting from cell contraction. Severe HND induces brain shrinkage, which can tear cerebral blood vessels, leading to cerebral hemorrhage, seizures, and encephalopathy.
5. Clinical manifestations
Most children with hypernatremia are dehydrated and have the typical signs and symptoms of dehydration, which are weight loss, decreased skin turgor, pale skin color, and dry mucous membranes. The first signs of neonatal dehydration include the failure to have bowel movements or the presence of urate crystals. Children with HND tend to have better preservation of intravascular volume owing to the shift of the water from the intracellular space to the extracellular space. Hypernatremic infants potentially become more dehydrated before seeking medical attention. That’s because of intracellular water loss, the pinched abdominal skin of a dehydrated, hypernatremic infant has a doughy feel.

The heart rate will be increased and the blood pressure decreased in severe cases. The urine output can go from low to none. Hypernatremia, even without dehydration, causes central nervous system symptoms that tend to parallel the degree of sodium elevation and the acuity of the increase. Patients are irritable, restless weak, and lethargic. Some infants have a high-pitched cry and hyperpnea. Alert patients are very thirsty, although nausea may be present. Hypernatremia causes fever, although my patients have an underlying process that contributes to the fever.

HND can lead to neurological impairment because brain cells are especially vulnerable to cell dehydration and severe HND induces brain shrinkage, which can tear cerebral blood vessels, leading to brain hemorrhage. Cerebral hemorrhages are the most serious complications of HND. Those include subarachnoid, subdural, and parenchymal hemorrhages that can eventually lead to convulsions and even coma.

6. Differential diagnosis
Hypernatremia caused by salt poisoning or dehydration must be distinguished correctly, as the two situations need different legal and medical approaches. But first it is important to exclude other medical causes. When a child presents with hypernatremia, a defect in the system that controls urinary concentration must be excluded as a cause. Children with hypernatremia caused by such a defect usually have obvious polyuria and polydipsia that result from central or nephrogenic diabetes insipidus, chronic renal failure, or rare renal problems. Such children can easily develop negative water balance (when the volume of water ingested is less than the volume excreted in urine), which reduces solute excretion and induces HND (16).
In hypernatremia caused by water deficit, we can differentiate some conditions by assessing the urine osmolality. Low urine osmolality indicates diabetes insipidus, either hereditary nephrogenic, or centrally caused by ADH deficiency. High urine osmolality on the other hand shows either an increased insensible loss as seen in premature infants and infants under phototherapy, or an inadequate intake sometimes encountered during ineffective BF and child neglect. Dehydration results from a child having negative water balance. If the child is also losing more salt than he is receiving (negative sodium balance), the plasma concentrations of sodium may remain stable. For hypernatremia to develop, the water loss must exceed any loss of salt. The patient's history usually reveals causative factors. Typically, diarrhea causes water loss, and vomiting prevents intake of water.

In excessive sodium hypernatremia, salt intoxication is the common cause, and this can be a result of excess sodium bicarbonate used to correct metabolic acidosis at the hospital, an improperly mixed formula due to incorrect education on feeding with formulas, an ingestion of seawater or even an intentional salt poisoning. The child will present with confusion, hypernatremia, and weight gain but without fever, diarrhea, and/or vomiting.

By measuring the urine output, we are able to lead to different other causes. A low urine output indicates water losses from the gastric tract such as diarrhea and emesis or from the skin in burned patients and excessive sweating. A high urine output shows some renal conditions, diabetes mellitus or osmotic diuretics intake.

![Figure 1. Differential diagnosis of hypernatremia (14).](image)
7. Management and treatment

To assess the dehydration, with the help of others clinical signs, weight loss represents a reliable sign. A recent study (1) has developed charts for weight loss observed in healthy breast-fed newborns and cases with breastfeeding-associated with HND. The charts will be compared to investigate when and to what degree weight loss of healthy breast-fed newborns, born at normal term, differs from those with HND. Charts based on weight loss with Standard Deviation Score (SDS) were then constructed as follow (graph 2). Values that are below the -1 SDS line at day 3, the -2 SDS line at day 4, and the -2.5 SDS line at day 5 confirm the condition of HND. This study demonstrated that weight loss in the very early postnatal days already differs between healthy newborns and newborns with HND, and this difference increase with age. Therefore, with the help of these charts an early diagnosis of HND can be made.

Graph1: Standard deviation score (SDS) lines of the reference chart for relative weight change in healthy breast-fed newborns (logarithmic-scale axes). The dots represent the weight change of the newborns.
The first priority in managing a child with HND is to stop the ongoing water loss by treating the underlying cause. The next step is to restore the intravascular volume with isotonic fluid. Dehydration can be treated with oral, nasogastric, or intravenous fluids. The child is given a fluid bolus, usually 20 mL/kg of the isotonic solution, over about 20 to 30 minutes. More severe dehydration needs repeated boluses at a faster rate. After the fluid bolus is given, the signs of dehydration should be reassessed in order to confirm a complete rehydration. The serum values of electrolyte concentrations, and acid-base balance should be evaluated clinically and via periodic laboratory determinations during prolonged therapy with sodium chloride and in patients whose condition warrants such evaluation (17). The Table 3 below (18) guides us in management according to three phases encountered during HND.

Graph 2: SDS lines of the reference chart for relative weight change (left y-axis) of newborns with breastfeeding-associated HND (logarithmic-scale x-axis). The dashed line shows the increase in serum sodium concentration (right y-axis) by day when median weight loss occurs. The dots present the weight loss of the cases.
Oral rehydration solution (ORS) is a simple and cost-effective method to correct mild to moderate dehydration. It acts on the coupled transport of sodium and glucose in the intestine, hence it contains glucose and electrolytes. However, IV therapy is required in more severe and complicated situations, such as in lethargic patients or in coma, in patients with uncontrolled emesis, and cases with bowel distention. Small lethargic infants with signs of severe dehydration are managed according to the below algorithm in the Table 4 (4).

<table>
<thead>
<tr>
<th>First 8 h</th>
<th>Next 18 h</th>
<th>Next 24 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free water deficit</td>
<td>Replace half of the deficit in first 24 hours</td>
<td>Replace remaining half over next 24 hours</td>
</tr>
<tr>
<td>SFD</td>
<td>Replace ½ of the deficit divided evenly over 8 hours</td>
<td>Replace remaining ½ of the calculated deficit divided evenly over 16 hours</td>
</tr>
<tr>
<td>Solute sodium deficit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Solute potassium deficit</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The hourly maintenance fluid was calculated in addition to the above-calculated deficiency. SFD: Solute fluid deficit, FWD: Free water deficit

Table 4: Algorithm of management in severe case of dehydration presenting with lethargy.

Fluid loss should not be corrected rapidly. Cerebral oedema as well as convulsions are serious risks during rapid rehydration, so correction of deficit should be achieved slowly and
gradually over 48 hours and should not be decreased to less than 12 mEq/L. To prevent cerebral edema and convulsion, individuals with hypernatremia should be managed in such a way that the reduction rate of serum sodium occurs at approximately 10 to 12 mmol/L/24 hr (19). Cerebral edema and seizures can be consequences of rapid correction of serum sodium level in these patients in whom the rate of fluid and sodium administration are inappropriate (20). In one study, the relationship between cerebral edema occurrence and rapid rate of fluid administration were detected (21). Monitoring is essential in management of such patients. However, for individuals who develop hypernatremia because of acute SP, rapid correction (1.0mmol/L/hr) within a few hours is also recommended for them to extrude the sodium from the brain to prevent convulsion (22). Severe hypernatremia seen after SP is hard to correct by administering more volume because it can worsen it to a volume overload, so a loop diuretic or even a dialysis might be required. It is then very important to promote judicious use of ORS in managing HND in the absence of an indication for IV correction, such as in patients with severe dehydration or shock, and simultaneously to educate caregivers about the consequences of excess intake and inappropriate preparation of ORS.

This solution should be hypotonic in case of water loss due to diabetes insipidus. ADH analog is added if the diabetes insipidus is central, to prevent further water loss. A nephrogenic diabetes insipidus patient requires a urine replacement solution.

8. Prognosis and complications
Full recovery usually follows a prompt treatment intervention but patients with recurrent HND can develop neurologic impairment including intellectual deficits and mental retardation during development, recurrent seizures recurrences. Acute hypernatremia presents with neurologic sequelae in 15% of the patients and a mortality rate of up 20%. The mortality rate is lower in children with chronic hypernatremia. Other complications concern the central nervous system, as it is suspected. Intracranial hemorrhage, cerebral edema and cerebral infarction are the main concerns in those patients.

HND is a well-recognized complication of unrecognized insufficiency of lactation in exclusive breastfed infants. Exclusively breast fed babies presenting with acute renal failure secondary to HND is a relatively less recognized entity and is seldom focused upon. Acute kidney injury (AKI) associated with HND, though not uncommon, is a relatively less recognized condition. Of particular concern is that neonatal AKI can have a complete
recovery or may have residual renal damage which may translate into sequelae in later life such as hypertension and chronic renal insufficiency (23). As exclusive breast feeding is the norm of infant feeding in first 6 months of life and incidence of HND is not uncommon, it is important to recognize AKI in these neonates and the potential for its long-term consequences in such setting. AKI in the setting of HND should be looked for and not be ignored. These babies may merit long term follow-up for possible sequelae. Therefore, there is an urgent need for follow-up studies of such babies into childhood and adulthood to look for unrecognized sequelae of such insult.

9. Case Report
This clinical case (24) illustrates well the problem of severe HND that has led to a circulatory shock with the issue of difficulties of BF as the cause. We emphasize here the importance of the role of using charts to help us in management of the ill newborn infant. It has been presented at the 13th Congress of Croatian Pediatric Society.

Female newborn, at the age of 10 days presented to the pediatric emergency department malnourished, adynamic, with impaired consciousness (GCS > 8), shallow breathing and bradypnea (30/min.), bradycardia (90/min.), filiform pulse, delayed capillary refill (> 4 s), hypotonicity, diminished spontaneous motoric movements, dry mucosa, absent skin turgor, sunken fontanelles, sunken eyes, and pointed chin. Generally the child was hypotrophic, with a body weight of 2640 g (23% less than in comparison to birth weight), with -2.25 SDS line and vitally endangered. The charts established by the study described above and illustrated by the 2 graphs have been used to monitor this case to rapidly consider a pathological weight loss. We strongly relied on them since the child presented in an emergent life-threatening condition.

Her family history is unremarkable and this was the mother’s first pregnancy without remarkable abnormalities. The child was delivered in the 41st week, without any remarkable complication, the birth weight was 3460 g (60c./0.25 SDS), the birth length was 51cm, the head circumference was 35 cm, with an Apgar score of 10/10. The child was exclusively breastfed.
Laboratory examination showed Na 167 mmol/L, Cl 126 mmol/L, pH 7.33 , aHCO3 22 mmol/L, BE 3, lactate 3.3 mmol/L, urea 34.2 mmol/L, creatinine 86 μmol/L, AST 564 U/L, ALT 271 U/L, GGT 353 U/L. A cranial ultrasound has demonstrated an hyperechogenic basal ganglia.
In the first hour, the treatment addressed the hypovolemic circulatory shock with IV fluid replenishment (2x10mL/kg 0,9% NaCl). Parenteral hydration was continued in the following 48h with glucose and electrolyte infusion according to the HD protocol, which resulted in a stepwise reduction of serum sodium (167-163-158-151-142 mmol/L/48h; 0,5 mmol/h). Later enteral feeding was instituted with a manufactured milk formula.

With clinical improvement, the laboratory results changed: Na 137 mmol/L, urea 1,4 mmol/L, creatinin 38 μmol/L, AST 141 U/L, ALT 154 U/L, GGT 209 U/L. At the age of three weeks, the child was released home with normal vital functions and in good general state. At hospital discharge, the measured body weight was 3550 g (15c./-1 SDS), and unremarkable 3T-MR of the brain.

This was a case report of a healthy newborn who in its first 10 days of life, developed a severe HND with circulatory shock and was vitally endangered due to inadequate BF. Such a condition is usually a consequence of enterocolitis or severe febrility in newborns on formula. However, this case demonstrates difficulties in BF as the culprit. Vigilant surveillance of the BF techniques, monitoring of body weight using the charts and education of pregnant women are steps and interventions, which can help prevention or early recognition of malnourishment or HND in newborns.

10. Discussion

HND represents a common issue in the pediatric population. Many etiologies are associated to the recurrent condition hence it requires rapid evaluation to determine the cause of the problem. Many etiologies are associated to the condition and these include excessive sodium as seen in infants feeding with inappropriately mixed formulas, in patients with metabolic acidosis incorrectly treated with sodium bicarbonate or in salt intoxicated children. Water deficit as another cause of HND includes the problem of diabetes insipidus, premature neonates, infants with excessive vomiting and diarrhea, or simply an ineffective BF. Those are the main causes encountered. The patient present with signs of dehydration such as weight loss, decreased skin turgor, pale skin color, and dry mucous membranes. Along with neurological signs like irritability or lethargy in severe cases, heart rate and blood pressure are vital signs to assess and can help in evaluating dehydration with its severity. However, to maintain blood pressure in hyponatremia, water shifts from the intracellular to extracellular space. Therefore, this mechanism not only makes the infants with hyponatremia less symptomatic, but also causes more dehydration before suitable medical intervention is sought. This puts therefore the assessment of weight loss in a central place when one assesses the
newborn infant. HND constitutes a medical emergency requiring an immediate diagnosis for adequate management. The pediatric literature proposes many forms of evaluation and monitoring of the patient and articles found online propose further and more accurate approaches to assess the problem. Their objective is to develop and establish a simple and quick but precise and reliable way that can be used to deal with the problem promptly. Charts for weight loss to detect HND have been developed as described above in the management and treatment part and represent a valuable tool to assess the neonates. A normal weight loss is expected during first 10 days as the newborn infant can lose up to 7 percent of his or her birth weight but then starts to regain weight at different proportions. Nonetheless this early physiological weight loss has to be followed with more attention and accuracy to avoid missing the problem of pathological dehydration and/or to prevent further damages to the patient. These charts for weight loss could be proposed as a routine monitoring in the hospital and maternity setting. The problem of HND represents a common issue in breastfed neonates and those infants have to be given a particular attention, as the development of HND can be relatively rapid with the serious consequences. The reliable charts are very helpful as it has been used in the clinical case of an infant in NICU described in the case report. The cause of HND in this case was a poor initiation of BF. HND always represented a recurrent problem in infants who are breastfed. When one faces a case of sick neonate, failure of galactopoiesis can occur due to infrequent breast stimulation and drainage. The issue is mostly related to inadequate prolactin surges and a buildup of local inhibitory factors in the milk. Ineffective milk removal was primarily associated with poor breastfeeding techniques such as an incorrect position and an inadequate latch. There can also be factors related to maternal and infant anatomical abnormalities that then interfere with suckling dynamics. It is important to emphasize the importance of an efficient BF with proper techniques, good volume of milk taken at the right frequency. Preventive measures through educational attitude toward young mothers especially primipare mothers must take a central place in the early postnatal days until they can be sent home safely. Table 5 (25) provides instructions to assess the efficacy of breastfeeding.
11. Conclusion

Monitoring the mother and baby in the 1st week of life for successful establishment of BF is essential. In countries with an early weighing policy and appropriate lactation support, the risk of HND is small (26,27). An overview of the baby should show alarming signs that will need prompt and further investigations. Those signs simply include how well the baby looks like, his level of reaction to his surroundings, the way he moves and cries, responds to normal stimulation from the mother and the team in the maternity. In an otherwise healthy baby, poor feeding and weight loss could be a reason to highly suspect hypernatremia. Neonatal HND due to inadequate BF or even underfeeding would appear to be a rather common problem. In order to avoid serious morbidity and mortality, all breastfed infants should receive regular follow-up by health-care worker. Furthermore, we advocate that all mothers should be taught the skills of BF, and warning signs of BF failure and hypernatremia, accompanied with the monitoring of the weight of infants until growth begins, in order to reduce the likelihood of this condition arising, especially for first-time mothers.

Table 5: Assessment of the efficacy of breastfeeding.
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13. References


14. Biography
Narindra Zoantsoa Ramanantseheno was born in France and spent his childhood in Madagascar where he finished high school. He moved back to France to start his studies and showed great interest in human science and biology. After his bachelor, he enrolled into School of medicine at the University of Zagreb. He currently does many clinical practices in Germany where he wants to continue his education after graduation.