

**Original article:**

**CLINICO-INVESTIGATIVE PROFILE & OUTCOME OF SEVERE HYPERNATREMIA - IN NEONATES IN A RURAL TEACHING HOSPITAL**

**<sup>1</sup>Dr.K.Navanitha Reddy, <sup>2</sup>Dr.(Col.)D.Y.Shrikhande\*, <sup>3</sup>Dr.Subhash S.Poyekar**

<sup>1</sup>PG Resident, <sup>2</sup>Professor & Head of Dept,of Pediatrics , <sup>3</sup>Associate Professor  
Department of Pediatrics, Rural Medical College, PIMS (DU) Loni, Maharashtra  
Corresponding author\*

**Abstract**

**Introduction:** Early diagnosis of neonatal hypernatremia is necessary for better prognosis. It's a medical emergency and it should not be overlooked. A detailed knowledge regarding the diagnosis, complications, etiology & treatment can help to prevent it. With this view present study was planned.

**Methodology:** Our investigation included 12 'out-born' cases of "lab-confirmed" Hypernatremia from NICU with Na<sup>+</sup> range 170-210 mEq/L and Acute Kidney Injury. Full neonatal & maternal history was taken.Details were obtained regarding pregnancy & the patterns of feeding.Clinical examinations were carried out from head to foot to evaluate for signs of hypernatremia, dehydration and further investigations were done.Analysis of the fluid correction therapy was undertaken.

**Results:** Majority of the neonates affected were born to primi mothers. Breast-feeding was in-frequent i.e. less than 7 times in 66% of the cases and 3 were formula-fed and 1 with top-fed (60% had undergone C-section).Delayed and inadequate feeding could have been major factors.

**Conclusions:** Key takeaway to note is that - complications such as cerebral edema & seizures should be watched out for. Early detection and proper management of the disorder are primarily required. All our cases responded well to the standardized protocol with high rate of survival (only 2 deaths out of 12 pts)

**Introduction:**

Early diagnosis of neonatal hypernatremia is necessary for better prognosis. It's a medical emergency and it should not be overlooked. A detailed knowledge regarding the diagnosis, complications, etiology & treatment can help to prevent it. Intraoperative changes including hemodynamic changes, aortic clamp time, and inflammatory response to cardiopulmonary bypass influence postoperative AKI. The most important factors that influence development of postoperative AKI are cardiac performance and sepsis .<sup>1</sup>

Prerenal azotemia (sometimes referred to as pre-kidney failure, occurs in response to decreased renal blood flow (RBF).Causes of pre renal azotemia in neonates include loss of effective circulating blood volume (perinatal blood loss, hemorrhage), dehydration (diarrhea, trans epidermal free water losses, poor intake, gastric or chest tube losses) capillary leak (hydrops fetalis, infection, or hypo albuminemia), increased abdominal pressure (necrotizing enterocolitis, ascites, repair or reduction of the gastroschisis, omphalocele, or congenital diaphragmatic hernia), and decreased cardiac output (cardiac surgery, heart failure, or the use of extracorporeal membrane oxygenation, which

results in the lack of pulsatile flow) Prolonged or severe hypo perfusion is the most common cause of intrinsic AKI. Other causes of intrinsic AKI include nephrotoxic medications and sepsis, which can cause AKI in both hypodynamic and hyperdynamic blood flow. Approximately 6% to 8% of the newborns admitted to the NICUs have intrinsic AKI, with severe perinatal asphyxia being the most common cause (Stapleton et al, 1987). Other rare causes of AKI include renal vein thrombosis, renal artery thrombosis, haemoglobinuria, and myoglobinuria. Laboratory markers of pre renal azotemia include low urinary sodium excretion, low fractional excretion of Na<sup>+</sup>, low renal failure index and high BUN: S Cr ratio.<sup>2,3</sup>

**Methodology:**

The present study was carried out in Department of Pediatrics for two months duration. Sample size was estimated.

Our investigation included 12 ‘out-born’ cases of “lab-confirmed” Hyponatremia from NICU with Na<sup>+</sup> range 170-210 mEq/L and Acute Kidney Injury.

Full neonatal & maternal history was taken. Details were obtained regarding pregnancy & the patterns of feeding. Clinical examinations were carried out from head to foot to evaluate for signs of hyponatremia, dehydration and further investigations were done. Analysis of the fluid correction therapy was undertaken.

Patients were revealed as having hyponatremia, AKI with S Cr & Urea levels elevated

Na <sup>+</sup> (mEq/L)	No. of Babies
>170	4
>185	6
>200	2

Table 1) Na<sup>+</sup> (mEq/L) level representation

Stage of Kidney Injury	Serum Creatinine SCr Levels(mg/dl )	Urine Output /24 Hours (ml/kg/per hr)	No.of Babies
0	No change in SCr OR SCr rise<0.3 mg/dL	>1.0	-
1	SCr rise >=0.3 rise from Baseline SCr OR SCr rise >=1.5-1.9 X Baseline SCr	> 0.5 and <=1	-
2	SCr rise >= 2 to 2.9 X Baseline SCr	>0.3 and <=0.5	2
3	SCr rise >= 3 X Baseline SCr OR SCr >=2.5 <= 0.3	<= 0.3	10

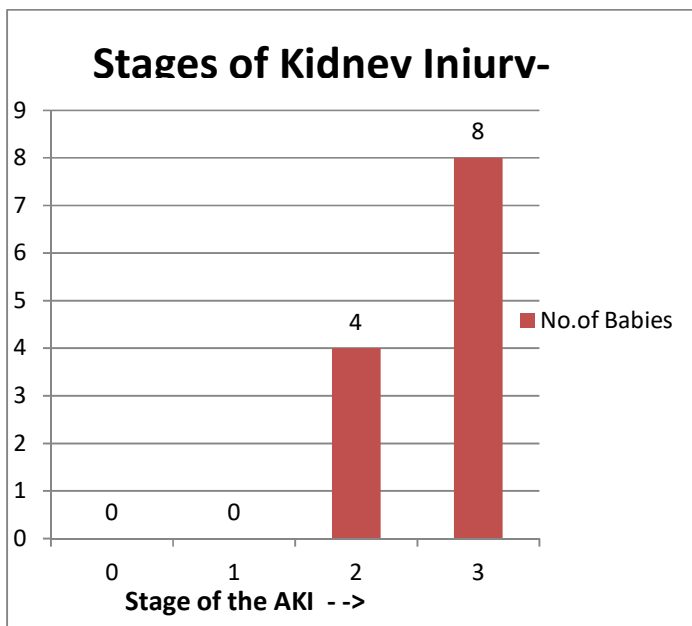
**Table 2) Stage of Kidney Injury**

NS bolus (10ml/Kg) --> did not pass urine -->Injection Lasix (1mg/Kg) repeated after 12 hours-->Then when no urine output the patients were placed on Oliguric Correction i.e Oliguric correction =  $500 \times \text{Body surface area (Dextrose 5\%)} + \text{Urine output (Normal saline)}$  -->When urine output is  $>1\text{ml/kg/hour}$  -->the patient was placed on N/2 saline (gradually decreasing the sodium levels by 12 mEq/L/day with rate of 0.5 mEq/L/hour). Some babies had seizures which were controlled by 3% saline (4 ml/Kg).Calcium was corrected by giving calcium gluconate 10% (8ml/kg/day).Potassium was corrected by administering calcium gluconate,salbutamol nebulization.

**Results:**

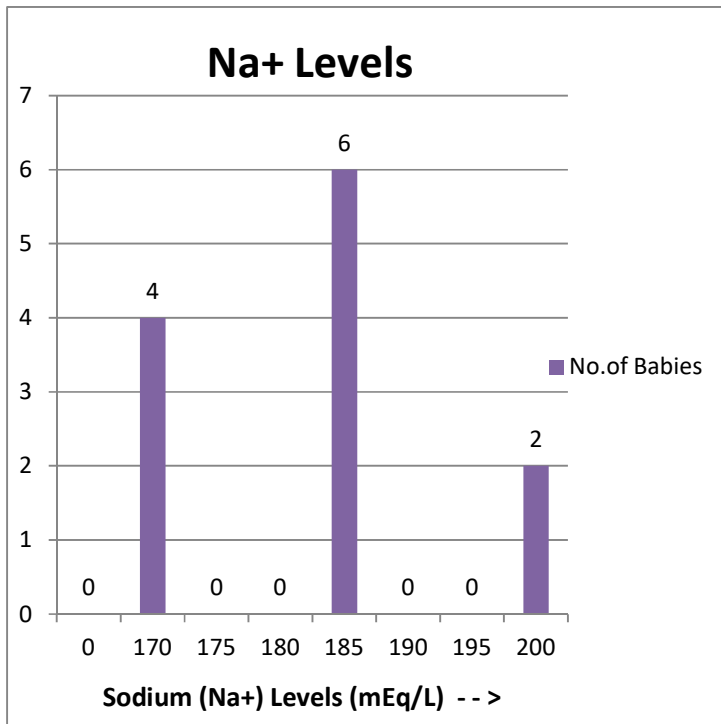
Majority of the neonates affected were born to primi mothers. Breast-feeding was in-frequent i.e. less than 7 times in 66% of the cases and 3 were formula-fed and 1 with top-fed (60% had undergone C-section).Delayed and inadequate feeding could have been major factors.

Of the neonates with Hyponatremia, about 75% of them - had  $>10\%$  weight loss due to dehydration. Age on admission for most was 9-11 days and all the cases responded to oliguric correction. Survival rate was high (only 2 deaths/12 cases).Hyperbilirubinemia was seen in 68% cases. Complications expected during management & recovery were “few” due to frequent monitoring and gradual lowering of Sr Na+. One patient died due to cerebral edema. Ventilated the 2nd patient before death(Note:2nd baby hadn't passed urine at all).Fever, poor feeding & irritability were present in most of the cases. Seizures & posturing were seen in 3 of the cases. Azotemia seen in 2 cases while sepsis was present in 7 cases.



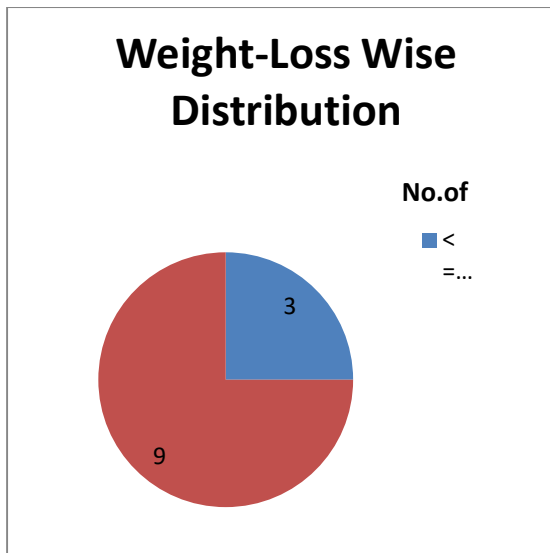
**Graph 1: Stage of Kidney Injury**

33% were found with Stage2 and the rest were with Stage3 of AKI.



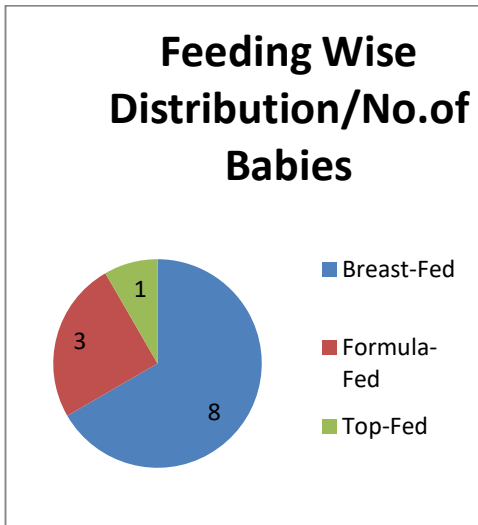
**Graph 2: Sodium Levels Distribution**

In this study, no. of babies with Na<sup>+</sup> levels above 170 mEq/L were 4 in number and above 185 mEq/L were 6 and those infants with above 200 mEq/L were 2 .



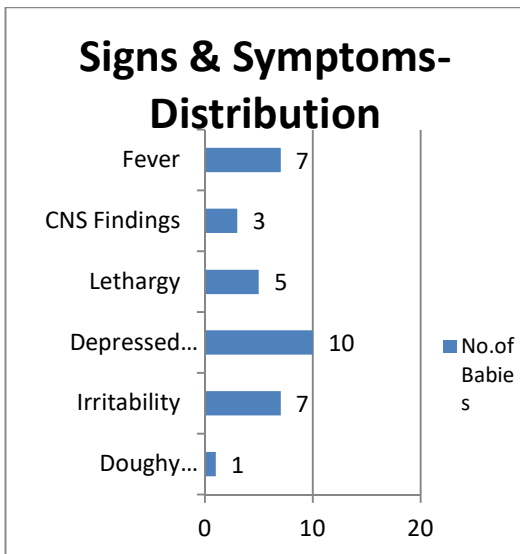
**Graph 3: Weight-Loss wise Distribution**

75% of patients had more than 10% weight-loss and 25% had less than 10% weight loss.



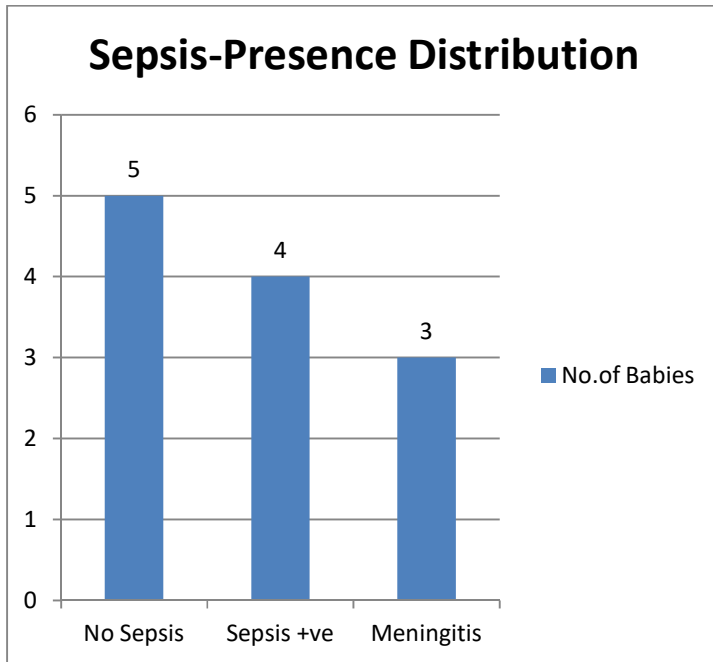
**Graph 4: Feeding-Wise Distribution**

In this study, breast-feeding (66%) was the most common feeding method, followed by formula-fed (25%) and then top-fed among the patients.



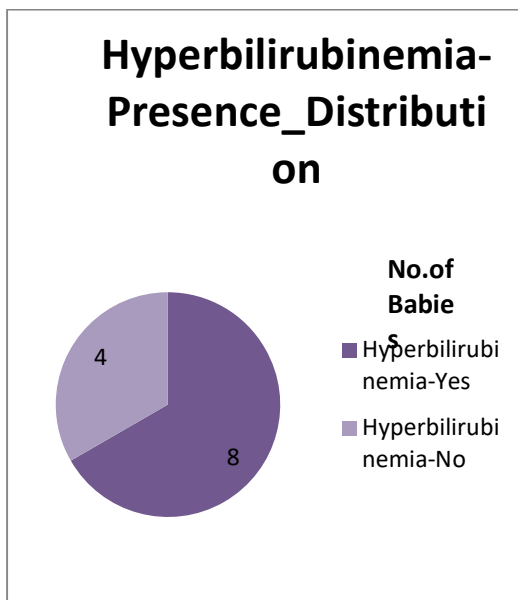
**Graph 5 : Signs and Symptoms on presentation**

In this current study as shown in above Graph5 : Fever was found in 7 cases (58%) , CNS findings were found in 3 patients(25%), lethargy was present in 5 cases (41%), depressed anterior fontanelle was seen in 10 cases(83%),irritability in 7 patients(58%) and doughy abdomen in 1 case(8%)



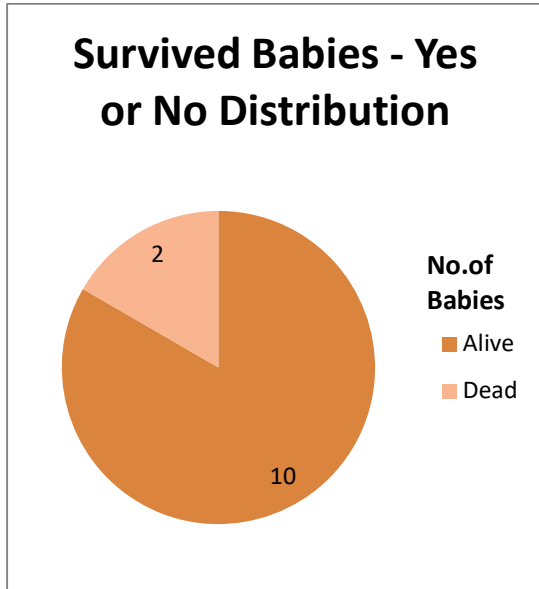
**Graph 6 – Sepsis presence-wise distribution**

In this study, more than 41% were not having Sepsis. Sepsis indicated by CRP positivity and/or blood culture positivity.



**Graph 7 – Hyperbilirubinemia presence-wise distribution**

66% of patients developed Hyperbilirubinemia along with Hyponatremia which is a significant association. Hyperbilirubinemia was assessed by NICE guidelines(2014)



Graph 8- Survived Babies- Number distribution

10 patients survived i.e 83% of the cases and mortality was seen in 2 patients.

#### Discussion:

Severe Hyponatremia (serum sodium concentration  $>170$  mEq/L) reflects a deficiency of water relative to the TBS (Total Body Sodium) and is most often a disorder of water rather than sodium homeostasis. Clinical manifestations are fever, hyper-tonicity and hyper-reflexia and neurological (due to an osmotic shift of water out of brain cells) including confusion, neuromuscular excitability, seizures & coma. Hyponatremia and its effect in neonates and its relation with breastfeeding is been studied for long. Few authors reported that hyponatremia is more common in bottle fed infants specially those using concentrated formula feeds and undiluted cow milk.<sup>4</sup>

However other authors reported that some normal healthy newborns developed neonatal hyponatremic dehydration, even if they are exclusively breast fed. Weight loss up to 5-7% of its birth weight during first week of infant's life is considered normal however in case of weight loss more than 10% of birth weight the neonate should be suspected for neonatal hyponatremic dehydration. Plasma hyper tonicity and subsequent intracellular water loss causes the brain cells to shrink, leading to rupture of bridging vessels with the hemorrhages in subarachnoid and brain parenchyma and thrombosis. If an attempt is made to correct high sodium concentration quickly, there is severe risk of osmotic changes in the brain which can exacerbate the cerebral edema, thus adding to potential brain damage.<sup>5</sup> Care should be taken to that the process of rehydration should be carried out slowly to minimize or prevent the damage to the brain. Hyponatremic dehydration is the most dangerous form of dehydration because of complication of the hyponatremia and of therapy.<sup>6,7,8</sup>

Fluid required for correction was calculated by considering BSA (body surface area) i.e. placing on Oliguric correction. The gradual reduction of Na<sup>+</sup> levels resulted in good recovery with minimal complications. Those patients who developed complications during correction therapy were observed and investigated. Out of 12 cases, 3 patients developed seizures and Azotemia was seen in 2 cases.<sup>9</sup>

All our cases responded well to the standardized protocol with high rate of survival (only 2 cases out of 12 died). All our cases were due to hypernatremia and acute kidney injury. Our findings showed an increased occurrence in the babies of primigravidas, and a high simultaneous association of neonatal hyperbilirubinemia due to “breast feeding jaundice”. The complications expected during our management and recovery were also few due to frequent observation and monitoring with a gradual, and steady lowering of the serum sodium with higher sodium content and higher fluid volumes. If the mothers are educated about the benefits of frequent breast feeding the occurrence of Hypernatremia could be greatly reduced.<sup>10,11</sup>

#### **Conclusions:**

Key takeaway to note is that - complications such as cerebral edema & seizures should be watched out for. Early detection and proper management of the disorder are primarily required. All our cases responded well to the standardized protocol with high rate of survival (only 2 deaths out of 12 pts)

#### **References :**

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