"STUDY OF HYPONATREMIA IN CRITICALLY ILL CHILDREN ADMITTED TO PAEDIATRIC INTENSIVE CARE UNIT . "

By

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ABSTRACT

BACKGROUND :

Hyponatremia is the most commonly encountered electrolyte disturbance in hospitalized paediatric patients with an incidence ranging from 15 to 30%.

Most cases of hyponatremia are mild and asymptomatic but this problem is now increasingly recognized as a significant cause of morbidity and mortality. It frequently develop or frequently exacerbated during hospitalization and are associated with increased length of stay and mortality. Clinical manifestations may range from absent to life threatening complications.

OBJECTIVES OF THE STUDY

1. To find the incidence of hyponatremia in critically ill children admitted in pediatric intensive care unit.

2. To evaluate the effect of hyponatremia on morbidity and mortality.

- (Morbidity is taken as ICU stay more than 5 days and the complications due to hyponatremia.

MATERIAL AND METHODS :

This was a prospective study on the incidence and outcome of hyponatremia in patients aged 1 month to 14 years old admitted in the paediatric intensive care unit in R L Jalappa hospital, kolar . Consent was taken from the parents of all the 152 children enrolled in the study to detect the serum sodium levels at the time of admission to the paediatric intensive care unit along with other necessary tests. Serum

sodium samples were analyzed using VITROS , a machine which utilizes indirect ion-specific electrode potentiometry for sodium determination.

MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data. EPI Info (CDC Atlanta), Open Epi, Med calc and Medley's desktop were used to estimate sample size, odds ratio and reference management in the study.

RESULTS :

In the study out of 152 cases, 51 (33.5%) had hyponatremia and 101 (66.5%) had normonatremia. Among subjects with Hyponatremia and Normonatremia majority of the subjects had Bronchopneumonia (41.3 % and 50.5 % respectively). Second most common diagnosis in both hyponatremia and Normonatremia was acute encephalitis (24% and 22.8% respectively). There was significant association between diagnosis and hyponatremia among subjects. The morbidity, as determined by the PICU stay was significantly higher in patients with hyponatremia when compared to those with normonatremia. Incidence of seizure and altered sensorium among hyponatremic group was 23.5% and 39.2% respectively. These complications are seen only in the moderate and severe hyponatremia group. Incidence of mortality in hyponatremic group was 11.7 % but hyponatremia cannot be attributed to mortality as the confounding factors are not excluded.

CONCLUSION :

From our study we infer that hyponatremia is common entity in varied clinical conditions in children. Giving hypotonic fluids to these patients put them at higher risk of developing hospital-acquired hyponatremia which poses greater risk for severe metabolic complications, and higher rates of morbidity and mortality.

ABBREVATIONS

TBW	: Total body water.
ICF	: Intracellular fluid.
ECF	: Extra cellular fluid.
CSF	: Cerebrospinal fluid.
Wt	: Weight in kilogram.
S.Na+	: Serum Sodium.
GFR	: Glomerular filtration rate.
Nacl	: Sodium Chloride.
AVP	: Arginine vasopressin.
ADH	: Antidiuretic hormone.
mEq/l	: Milli equivalent per liter.
%	: percentage.
<	: Lesser than equal to
>	: Greater than equal to
Ml	: Milliliter.
mg/dl	: Milligram per deciliter.
rpm.	: Rotation per minute.
S	: Significant.
NS	: Not Significant.
M	: Males.
F	: Females.
T	: Total
R.L.	: Ringer's lactate.

N.S : Normal Saline

Cl^- : Chloride ion

K^+ : Potassium ion

Na^+ : Sodium ion

PICU : Paediatric intensive care unit

Na^+ - K^+ - ATPase pump : Sodium – Potassium –activated Adenosine Triphosphatase pump

SIADH : Syndrome of Inappropriate Antidiuretic Hormone

ATP : Adenosine Triphosphate

HMP : Hexose Monophosphate

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INTRODUCTION

Electrolyte abnormalities are common in children admitted to intensive care unit. They are disorders of relative water loss or excess, resulting in alteration in effective plasma osmolality, leading to transcellular shift of water, there by altering cell volume. They have a significant contribution to the morbidity and mortality, irrespective of the primary problem. The consequences may be dire if abnormalities of serum electrolyte concentration remain undetected and untreated. The therapeutic objective in the treatment of fluid and electrolyte imbalances in infants and children is to restore normal physiologic homeostasis. To achieve this goal, timely recognition, a high index of suspicion and a thorough knowledge of the common electrolyte abnormalities are necessary.

Hyponatremia is generally defined as serum sodium concentration below 135meq/Litre. This is the most commonly encountered electrolyte disturbance in hospitalized pediatric patients with an incidence ranging from 15 to 30% [1-3].

Most cases of hyponatremia are mild and asymptomatic but this problem is now increasingly recognized as a significant cause of morbidity and mortality. It frequently develop or frequently exacerbated during hospitalization and are associated with increased length of stay and mortality. Clinical manifestations may range from absent to life threatening complications.

A high frequency of hyponatremia has been implicated in respiratory diseases like pneumonia and bronchiolitis, acute diarrhoea, CNS infections and septicaemia [3,5].

Significant adverse events involve the central nervous system and are brought about by acute changes in serum sodium levels or by overzealous correction of hyponatremia. Hyponatremic encephalopathy, the most severe neurologic complication, has recently been largely attributed to the use of hypotonic maintenance intravenous fluids.[6] Studies have elucidated that at serum sodium levels less than 130meq/L, there is an increased risk of developing neurologic symptoms, especially if it occurs acutely (or less than 48 hours) [1].

Despite its deleterious effects, literature on pediatric hyponatremia is still lacking especially in the local setting. Available literatures also had small sample size, making it difficult to draw conclusions and recommend appropriate treatment. With its mortality rate reaching up to 20% in some studies[1], it is vital to identify diseases which confer high risk for hyponatremia. Early intervention will ultimately lessen the risk for developing adverse events from this metabolic emergency. In this study, the incidence and outcome of hyponatremia in the patients admitted in the paediatric emergency room of a tertiary institution were determined and diseases commonly associated with it were identified.

OBJECTIVES

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

BODY FLUID

The maintenance of a relatively constant volume and a stable composition of the body fluid are essential, for homeostasis. Some of the most important problems in clinical medicine arise because of abnormalities in the control systems that maintain this constancy of the body fluids. The total amount of body fluid volume and the total amounts of solutes as well as their concentrations are relatively constant during steady state conditions as required for homeostasis. This constancy is remarkable because there is continuous exchange of fluid and solutes with the external environment as well as within the different compartments of the body[8].

BODY FLUID COMPARTMENTS

The total body fluid is distributed between two major compartments:

The extracellular fluid and the intracellular fluid.

The extracellular fluid in turn divided into the interstitial fluid and the plasma. There is another small compartment of fluid called transcellular fluid. These fluids are synovial, peritoneal, pericardial, intraocular and cerebrospinal. All together they constitute about 1 to 2 litres [8].

WATER

Water is the most important solvent of the fluid composition of living system. Next to oxygen, water is the most essential element of life. Total body water (TBW) as a percentage of body weight changes with age, decreasing rapidly at early life. Prenatally, TBW may be around 90% it decreases as the gestation advances. At birth TBW is 78% of the body weight. In the first few months of life, TBW drops dramatically to approximate the adult level of 55-60% of body weight at 1 year of age. At puberty, a further change in TBW takes place. Because fat has a low water content, TBW as a percentage of body weight is lower in mature women, who have greater amounts of body fat, 55%, than men, 60%. In the nonobese child, a close linear relationship is maintained between TBW and body weight and TBW can be calculated using body weight alone [10].

TBW (L) = 0.61 x weight (kg) + 0.251

TABLE 1 : DISTRIBUTION OF TOTAL BODY WATER AS PERCENT OF BODY WEIGHT

FLUID COMPARTMENT	**Infants**	**Older children**
Intracellular fluid (ICF)	40%	35-40%
Extracellular fluid (ECF)	35-40%	20-25%
Interstitial		15%
Intravascular (Plasma)		5%
Transcellular		1-3%
Total body water	75-80%	60%

INTRACELLULAR FLUID COMPARTMENT (ICF)

It consists of fluid inside the cells of the body. ICF has been estimated as the difference between total body water and ECF compartment.

[ICF = TBW – ECF]

The barrier separating ICF and ECF is the cell membrane. The principal caution in the cells is potassium and the principal anions are phosphates and proteins [9].

EXTRACELLULAR FLUID COMPARTMENT

ECF consists of about one third of the total body water (20-25%) and is composed of plasma water (5%) and interstitial fluid water (15%) sodium is the principal caution in the ECF, and chloride and bicarbonate the principal anion [9].ECF in the fetus is larger than ICF, and the ECF decreases with age. The ECF drops precipitously after birth, in large part because of postnatal diuresis. The ECF is calculated by [10].

ECF (L) = 0.239 x Wt (kg) + 0.325

GIBBS – DONNAN EQUILIBRIUM

There is a small difference between electrolyte concentration of the plasma and that of interstitial fluid. This is attributed to the Gibbs- Donnan Equilibrium. Donnan and Gibbs showed that in the presence of non diffusible ion, the diffusible ions distribute themselves, so that the products of the concentration of the diffusible ions on one side equal that on the other

side. The major difference between the plasma and the interstitial fluids is that the protein concentration is greater in the plasma as the capillary wall is impermeable to the flow of

proteins. There is also a small difference in the distribution of diffusible ions as Na^+ is little higher in plasma and Cl^- is little higher in the interstitial fluid [11].

TRANSCELLULAR WATER COMPARTMENT

The transcellular fluids from a minor part of the ECF, representing several small volumes. These include cerebrospinal, pleural, peritoneal and synovial fluid and those in the salivary glands, the pancreas, the liver, biliary tree, the eyes and the intraluminal fluid of the gastrointestinal tract [11].

Slowly Exchangeable Fluid Compartment

This comprises 8-10% of the body weight is contained in bone, dense,connective tissue, and cartilage. Because of poorly exchangeable nature, this compartment is not accessible to the body fluid regulatory mechanism. However, the fluid infused into the bone compartment can enter the plasma volume, an important physiologic relationship in situation necessitating interosseous fluid resuscitation [10].

BLOOD VOLUME

Blood contains both extracellular fluid (fluid in plasma) and intracellular fluid (fluid in red blood cells). However, blood is considered to be a separate fluid compartment because it is contained in a chamber of its own, the circulatory system. The average blood volume of normal adult is about 7% of body weight or about 5 litres [8].

TABLE 2 :

OSMOLAR SUBSTANCE: EXTRACELLULAR AND INTRACELLULAR [8].

	Plasma (mOsm/L of H_2O)	Interstitial (mOsm/L of H_2O)	Intracellular (mOsm/L of H_2O)
Na^+	142	139	14
K^+	4.2	4.0	140
Ca^{2+}	1.3	1.2	0
Mg^{2+}	0.8	0.7	20
Cl^-	108	108	4
HCO^{3-}	24	28.3	10
HPO_4^-,$H_2PO_4^-$	2	2	11
SO_4^{2-}	0.5	0.5	1
Phosphocreatinine			45
Carnosine			14
Aminoacids	2	2	8
Creatinine	0.2	0.2	9
Lactate	1.2	1.2	1.5
ATP			5
HMP			3.7
Glucose	5.6	5.6	
Protein	1.2	0.2	4
Urea	4	4	4
Others	4.8	3.9	10
Total mOsm/L	301.8	300.8	301.2
Corrected osmolar activity(mOsm/L)	282	281	281
Total osmotic pressure at 37^0C (mm of Hg)	5443	5423	5423

OSMOTIC EQUILIBRIUM AND FLUID SHIFT BETWEEN INTRA AND EXTRACELLULAR FLUID

The distribution of fluid between intracellular and extracellular compartments is determined mainly by osmotic effect of the smaller solutes especially sodium chloride and other electrolytes acting across the cell membrane. The cell membranes are highly permeable to water but relatively impermeable to small ions such as sodium and chloride. Therefore water moves across the cell membrane rapidly so that the intracellular fluid remains isotonic with extracellular fluid [8].

OSMOSIS

Osmosis is the net diffusion of water across a selectively permeable membrane from a region of high water concentration to one that has a lower water concentration.

OSMOTIC PRESSURE

Osmotic of water molecules across a selectively permeable membrane can be opposed by applying a pressure in the direction opposite that of the osmosis. The precise amount of pressure required to prevent the osmosis is called the osmotic pressure.

Osmotic pressure is the indirect measurement of water and solute concentration of a solution. The higher the osmotic pressure of a solution, the lower the water concentration but the higher the solute concentration of the solution [8].

OSMOLALITY

Osmolality is defined as the number of osmotically active particles per 1000gm of water in solution (mOsm/kg). The most important determinants of the osmolality or tonicity of the body fluids are the excretion or retention of water by the kidney thirst

and intake of water. The serum sodium concentration under most circumstances accurately, reflects the osmolality of the body fluids because the two fluid compartments (ECF and ICF) are always in equilibrium regarding tonicity. The normal osmolality of the body fluids from patient to patient varies between 280 and 295 mOsm per liter, but in any given person it is maintained within narrow limit (1 to 2 percent) because at the precise control of water balance. Osmolality can be measured directly with an osmometer (which measures freezing point depression) or can be estimated indirectly by determining concentrations of various solutes on the blood.

Plasma osmolality mOsm per litre [9]

$$= 2\,Na + \frac{\text{Blood urea nitrogen (mg per dl)}}{2.8} + \frac{\text{blood glucose (mg per dl)}}{18}$$

Because glucose and urea usually contribute about 5 mOsm per liter each to the osmolality, it is apparent that twice the sodium concentration plus 10 estimates the osmolality accurately under most circumstances. The osmolar gap can be calculated by a direct determination of osmolality (with an osmometer) and then calculating the osmolality indirectly from the preceding formula

Actual osmolality – calculated osmolality = Osmolar gap

Osmolar gap is quite small. A large osmolar gap suggests that another substance other than urea or glucose (e.g. a poison) is contributing to the osmolality of the plasma [9].

Osmolarity : Osmolal concentration of a solution when expressed as osmols per liter of solution.

RELATION BETWEEN OSMOTIC PRESSURE AND OSMOLARITY

As osmotic pressure depends on concentration of osmotically active particles, it is directly proportional to the osmolarity of the solution. (As osmolarity is a measure of the concentration of solute particles).[8]

Van't Hoffs kaw $\pi = CRT$

(π – Osmotic pressure, C – osmolal solute concentration)

R - Gas constant

T – Absolute Temperature

MAINTENANCE OF OSMOTIC EQUILIBRIUM BETWEEN INTRACELLULAR AND EXTRACELLULAR FLUID

Large osmotic pressures can develop across the cell membrane with relatively small changes in concentration of solutes in the extracellular fluid.

Isotonic Fluid

If a cell is placed in a solution 280-mOsm/liter, the cells will not shrink or swell because the water concentrations in intracellular and extracellular fluids are equal and the solutes cannot enter or leave the cells. Such a solution is said to be isotonic because it neither shrinks nor swells, the cells.(E.g. Normal saline (0.9% NaCI). 5% Dextrose solution)

Hypotonic Fluid

If a cell is placed into a solution that has a lower concentration of impermeable solutes (<280 mOsm/L) water will diffuse into the cells causing, it to swell; water will continue to diffuse into the cell, diluting the intracellular fluid while also

concentrating the extracellular fluid until both solutions have equal osmolarity .(E.g. Solution of sodium chloride with a concentration of less than 0.9%)

Hypertonic Fluid

If a cell is placed in a solution have a higher concentration of impermeable solutes, water will flow out of the cell into extracellular fluid, concentrating the intracellular fluid and diluting the extracellular fluid. In this case the cell will shrink until the two concentrations become equal. (E.g: sodium chloride >0.9%).

CHANGES IN VOLUMES AND OSMOLALITES OF EXTRACELLULAR AND INTRACELLULAR FLUID IN ABNORMAL STATES

Abnormalities of the composition and volumes of body fluids are common and important clinical problems and are of concern for almost all seriously ill, hospitalized patients.

The factors that cause changes in volumes and osmolalities of extra and intracellular fluids are:-

a. Ingestion of water.

b. Dehydration.

c. Intravenous infusion of different types of solutions.

d. Loss of large amount of fluid from the gastrointestinal tract.

e. Loss of large amounts of fluid by sweating or through kidney.

SODIUM DISTRIBUTION

Body content and distribution of sodium

Sodium is the bulk caution of extracellular fluid. Sodium is the principal osmotically active solute responsible for the maintenance intravascular and interstitial volumes. Of the total quantity of sodium in the body more than 30% is non exchangeable.

Of the total body sodium[9].

• 11% - is the plasma sodium.

• 29% - is in interstitial lymph fluid.

• 2.5% - in intercellular fluid.

• About 43% of total body sodium is in bone, but only one third of the sodium in bone is exchangeable.

• Dense connective tissue and cartilage contain 12% of body sodium, of which about two thirds is exchangeable.

• Exchangeable sodium content of fetus is 85mEq/kg. Compared with the adults value of 40mEq/kg.

Sodium is predominantly distributed in the extracellular compartment.

Intracellular concentrations are maintained at level of approximately 10 mEq/L and extracellular concentrations at the level of approximately 140 mEq/L. This low intracellular concentration is maintained by active extrusion of sodium from the cells by sodium potassium activated ATPase pump.

The intracellular sodium content usually remains relatively constant and changes in total body sodium reflect mostly changes in extracellular sodium. Redistribution of intracellular and extracellular sodium may occur in the absence of significant changes

in total body sodium.Intracellular sodium may increase in severely ill patient (Sick cell syndrome**)**[10] .

Because of the Donnan distribution of anionic proteins, the concentration of sodium in the interstitial fluid is approximately 97% of theat of the serum sodium value; changes in concentration of sodium in the serum are reflected by proportional changes in the concentration of sodium in the interstitial fluid [10].

REGULATION OF SODIUM

Intake: Regulatory mechanism of sodium intake is poorly developed, but may respond to large changes in sodium. Salt craving may occur in salt –wasting syndrome. Infants generally have relatively high sodium intake, because of the high sodium content of cow's milk (21 mEq/L). Sodium content of many infant formulas is high compared with that of breast milk (7 mEq/L) [10] .

Absorption

Maximum absorption is in the jejunum and minimal absorption in the stomach. Absorption takes place by way of a sodium/potassium/activated adenosine triphosphatase system, which facilitates movement of sodium by a transport protein that couples sodium with glucose. Intestinal transport mechanism of sodium augmented by aldosterone or desoxycorticosterone acetate [10] .

Excretion: Sodium excretion occurs through urine, sweat and faeces. The kidney is the principal organ for facultative regulation of sodium output.

REGULATION OF SODIUM BALANCE

The sensing mechanisms that are responsible for maintenance of sodium balance are as follow [12,65].

Cardiac sensors in atria and ventricle carotid artery.

- Arterial baroreceptors in carotid artery and renal baroreceptors in juxtaglomerular apparatus.
- Hepatic vascular sensors.
- Hypothalamic sensors.

Efferent Mechanisms

Renal factors

• GFR

• Physical forces in proximal tubule.

• Distal nephron.

• Neurohumoral factors.

• Sympathetic nervous system output.

• Renin angiotensin-aldosterone axis.

• Prostaglandin.

• Atrial natriuretic peptide.

• Nitric oxide.

• Arginine vasopressin (AVP).

Afferent Mechanisms

These mechanisms are designed to detect alterations in the ECF volume. These include sensors within the central portion of the vasculature, arterial sensors on the

high-pressure side of the circulation, organ specific sensors, and vascular sensors on the low-pressure venous side of the circulation. Within the central circulation are sensors in the right and left atria.

Distention of either atrium activates neurons that are responsive to mechanical stretch or wall tension. There is increased release to a humoral factor atrial natriuretic peptide. Atrial natriuretic peptide is synthesized as a prohormone, processed to the active form and stored in granules in cardiac myocytes. The net result of atrial distention is a brisk natriuresis and diuresis.Volume sensors in the ventricles also contribute to the maintenance of ECF volume. Activation of baroreceptors in the carotid artery and the juxtaglomerular apparatus in the kidney stimulates renal sodium excretion to minimize increase in effective ECF volume. The hepatic vascular bed also contains volume responsive sensors that regulate renal sodium excretion. Finally there are central nervous system sensors that stimulate natriuresis following infusion of hypertonic saline. The hypothalamus may secrete low molecular weight non-peptide natriuretic substance that inhibits the sodium – potassium ATPase pump [12].

Efferent Mechanism

Glomerular Filteration of Sodium

Under normal conditions, changes in GFR do not affect sodium homeostasis. A constant fraction of the filtered load of the sodium is reabsorbed in the proximal tubules despite transient, variations in GFR. This balance is called “glomerular tubular balance”. The factors that affect the GFR and promote sodium reabsorption in response to a decrease ECF volume such as dehydration, or hemorrhage are activation of sympathetic renal nervous system and stimulation of the rennin angiotensin system.

When expansion of ECF volume occurs atrial natriuretic peptide is released into the circulation and cause increase in urinary loss of sodium; in part as a response to increase GFR [10].

Tubular reabsorption of sodium

First reabsorption of sodium in the proximal tubules and loop of Henle delivers a constant proportion of the filtered load of sodium to the distal nephron. Secondly reabsorption of sodium in the distal tubules and collecting ducts is the fine regulator of the final amount of sodium excreted, which closely matches the amount of sodium ingested.

Reabsorbed sodium is actively transported out of the cell across their Basolateral membranes, producing an osmotic gradient that causes the movement of an equivalent amount of water. Significant sodium reabsorption (20% of filtered sodium) occur in loop of Henle. Water absorption occurs in the descending limb of loop of Henle, and sodium reabsorption occurs in the ascending limb. Sodium transport at the thick ascending limb is active. When the load of sodium delivered to the loop of Henle is increased most of the excess load is reabsorbed in the loop. Thick ascending limb of the loop of Henle absorbs 30%, distal convoluted tubule 7.8% and collecting tubule 2% total of 40% and 60% absorbed by proximal tubule15. Fine regulation of sodium occurs in distal convoluted tubules and collecting ducts. If the load of sodium reaching the distal tubules increases significantly, reabsorption does not increase proportionately and added load is excreted in the urine. In healthy children less than 1% of filtered sodium is excreted in the urine [10].

FACTORS REGULATING SODIUM EXCRETION

Renin-angiotensin aldosterone axis

Renin is a proteolytic enzyme released from the juxtaglomerular apparatus, stimulation of release of rennin include decrease in renal perfusion pressure and decrease in sodium chloride concentration of delivery to the macula densa. Angiotensin I is formed by cleavage of the substrate angiotensinogen by rennin. Angiotensin I is converted to angiotensin II by a specific converting enzyme. Angiotension II, by inhibiting rennin secretion, act as a negative-feedback regulator of rennin release.

The renin – angiotensin system regulates tubular sodium reabsorption by the direct stimulation of sodium reabsorption in the proximal tubule by angiotensin II and by the stimulation of aldosterone secretion by angiotension II. Aldosterone secretion promotes sodium reabsorption in late distal convoluted tubule and collecting ducts. Aldosterone increases potassium secretion leads to loss of potassium in urine [10].

Atrial natriuretic peptide

Atrial natriuretic peptide released from cardiac myocytes, when the ECF volume increases and stretches atrial wall. ANP acts on kidney generally antagonize the sodium retaining mechanism of the rennin-angiotensin system and increase water and sodium excretion .[10]

Prostaglandins also modulate sodium excretion by influencing the glomerular microcirculation and via direct effects on the tubule epithelium. PGE2 inhibits NaCl reabsorption I the medullary thick ascending limb of Henle [12].

Nitric oxide modulates the renal arteriolar resistance that regulates glomerular filtration rate, and it contributes to the activity of the tubular glomerular feed -back

mechanism. Nitric oxide directly inhibits sodium transport via apical channel in cultured collecting duct cells [12].

During severe hypovolemia, AVP is secreted to promote water reabsorption and restoration of the intravascular space. Volume sensitive AVP secretion overrides osmotic suppression of hormones release, that is, preservation of ECF compartment size takes precedence over normalization of serum osmolality [12] .

Starling forces in the intercellular space of the proximal tubule cells and the interstitial space between the tubular cells and the peritubular capillaries influence the movement of reabsorbed solute and water into the peritubular capillaries. Sum of starling force favors the movement of solute and water from the intercellular and interstitial space into the peritubular capillary. Rapid expansion at the extracellular fluid volume increases the interstitial hydrostatic pressure, preventing sodium reabsorption and producing increased urinary sodium excretion [10] .

CONTROL OF EXTRACELLULAR FLUID OSMOLARITY AND SODIUM CONCENTRATION

The regulation of extracellular fluid osmolarity and sodium concentration is closely linked. The factors involved are [13]:-

1. Osmoreceptor _ ADH system.
2. Thirst mechanism.
3. Role of angiotensin II and aldosterone.

1. Osmoreceptor – ADH feedback system

a. An increase in extracellular fluid osmolarity (that is increase in plasma sodium concentration) causes shrinkage of osmoreceptor cell located in anterior hypothalamus near the supra optic nuclei. Then nerve signals are sent to additional nerve cells in the supraoptic nuclei, which then relay these signals down the stalk of the pituitary gland to the posterior pituitary. This leads to stimulation of the release of ADH, which is stored in secretory granules or (vesicles), in never endings.

b. ADH increases the water permeability of the late distal tubules, collecting tubules increases water reabsorption and excretion of a small volume of concentrated urine. Thus water is conserved in body white sodium and other solutes continue to be excreted in urine. This causes dilution of solutes in extracellular fluid, thereby correcting initial excessively concentrated extracellular fluid. Opposite sequence of events occurs when extracellular fluid becomes too dilute .

TABLE 3 : Regulation of ADH secretion [13]

↑ ADH	↓ADH
↑Plasma osmolality	↓Plasma osmolality
↓Blood volume	↑Blood volume
↓Blood Pressure	↑Blood pressure

2. Thirst mechanism

Increase extracellular fluid osmolarity causes intracellular dehydration in thirst center, stimulating the sensation of thirst leading to dilution of extracellular fluid and return of osmolarity towards normal.

3. Role of angiotensin II and aldosterone

When sodium intake is low, increase level of these hormones stimulates sodium reabsorption by the kidney, therefore preventing large sodium losses and reverse occurs when sodium intake is high [13] .

HYPONATREMIA

Hyponatremia is defined as a sodium concentration of less than 135mEq./L [10,14] . Commonly encountered electrolyte disturbance occurring is about 1.5% of children hospitalized after the new born period[14] .

Definition of hyponatremia based on biochemical severity.[4]

Mild hyponatremia – 130 – 135 meq/L

Moderate hyponatremia – 125 – 129 meq/L

Profound or severe hyponatremia - < 125 meq/L

Anderson RJ et al (1986) found clinically significant hyponatremia (serum sodium less than 130mEq/L) was a frequent occurrence with incidence 1-2% of hospitalized patients with acute or chronic illness [15] .

Wattad A et al in (1992) observed that, out of 11,702 hospital admissions, 161 patients were hyponatremic (serum sodium less than 130 mEq/l), an overall frequency of 1.38% [16] .

Many patients do not have symptoms directly attributable to hyponatremia, because the level of serum sodium is not severely depressed. Serum sodium concentrations below 120mEq/L are more frequently associated with serious clinical symptoms. Low plasma sodium in seriously ill patients is a bad sign, whatever the underlying illness [17].

Hyponatremia is generally is secondary manifestation of another primary disease state. Symptoms of low serum sodium are merged with those of the underlying disease state [14] .

Clinical pathophysiology

Clinical manifestation of hyponatremia is obvious when serum sodium level falls rapidly below 120mEq/L [14] .Severe hyponatremia (S. Na+ 110mEq/L or less) can cause crippling fatal brain damage [18] .

Central nervous system

When the serum sodium decreases, plasma osmolality is reduced, water moves into the brain. Cerebral over hydration is the major cause of neurologic manifestation of hyponatremia[14].

Apathy, anorexia, nausea, vomiting, agitation headache, altered consciousness, seizures and coma. The severity of symptoms is often dependent on the magnitude and rapidity of the depression of the serum sodium concentration. Acute hyponatremia from water intoxication,

occurring during 24hrs or fewer hours and resulting in a serum sodium concentration less than 120mEq/L; causes nausea, vomiting, muscular twitching, seizures and coma. The mortality rate has been reported to be as high as 50% in adults.

As hyponatremia develops, osmotic equilibrium between brain and plasma is disturbed. The brain adaptation to hyponatremia is accomplished by two mechanisms.

(1) Loss of interstitial fluid into the cerebrospinal fluid.

(2) Loss of cellular solute, mainly potassium and organic osmolytes.

The loss of sodium, potassium and chloride from the brain provides most of the protection against cerebral edema in the first hour of hyponatremia. When hyponatremia is sustained the brain slowly loses intracellular osmolytes, mainly aminoacids. Losses of organic osmolytes during prolonged or severe hyponatremia are especially important in defending the brain against swelling because the amount of electrolytes that can be lost from the brain in responses of hyponatremia is limited.

The rate of recovery of brain intracellular potassium and organic osmolytes is much slower during correction of hyponatremia.

If correction of the low serum sodium occurs more rapidly than the brain can recover solute, the higher plasma osmolality may dehydrate and injure the brain.

Cardiovascular Response

Cardiovascular response to hyponatremia depends mostly on the effective arterial blood volume, which may be increased, decreased or, normal depending on the underlying disorder.

Intravascular volume is determined by the distribution of water between the intracellular and extracellular fluid compartments.

In a volume-depleted child, hyponatremia induces a further decrementin the intravascular volume by allowing movement of water out of the ECF compartment into the intracellular fluid space. The primary stimulus to the release of ADH is an increase in serum tonicity (osmolality); however, it is also released in response to decrease effective arterial blood volume accompanying hyponatremic edematous disorders and ECF volume depletion. The ADH potentiates the hyponatremic state by increasing water reabsorption by the renal tubules.

Musculoskeletal

Occasionally muscular cramps and weakness occurs, but these symptom resolves rapidly when the serum sodium is corrected.

Knochel JP et al found muscular cramp and weakness occurs more commonly in acute hyponatremia than chronic hyponatremia[19] .

Renal function

The usual renal response to hyponatremia is production of dilute urine.

But this is impaired by ADH effect. Depending on the primary disease process and the effective arterial blood volume, the kidney will regulate the volume of urine output.

If urine sodium <10mEq/L, it indicates renal handling of sodium is intact.

If urine sodium >20mEq/L it indicates intrinsic renal tubular damage or a natriuretic response to hypervolemia [14] .

CAUSES OF HYPONATREMIA [14]

Isotonic pseudohyponatremia (POsm = 280 – 295mOsm/L)

(a)Hyperproteinemia. (b) Hyperlipidemia.

1. Hypertonic hyponatremia

POsm > 295.

a. Hyperglycemia.

b. Mannitol, glycerol.

2. Hypotonic hyponatremia

a. Hypovolemic hyponatremia

(i) Renal losses (UNa > 20mEq/L)

1. Diuretic excess.

2. Osmotic dieresis

3. Salt wasting nephropathy

4. Adrenal insufficiency

5. Proximal RTA.

6. Metabolic alkalosis.

7. Pseudohypoaldosteronism.

(ii) Extra renal losses (UNa <20mEq/L.)

1. Gastrointestinal

• Vomiting

• Diarrhoea

• Drainage tubes / fistula

2. Sweat

3. Third space

- Pancreatitis

- Burns

- Muscle trauma

- Peritonitis

- Effusions

- Ascites

b. Euvolemic hyponatremia (UNa > 20mEq/L) (POsm <280mOsm/L)

1. SIADH

2. Reset osmostat

3. Glucocorticoid deficiency

4. Hypothyroidism

5. Water intoxication

- IV therapy

- Tap water enema

- Psychogenic water drinking

Causes of syndrome of inappropriate antidiuretic hormone (SIADH) [14] .

1. CNS disorder

- Infection: TBM, bacterial meningitis, encephalitis..

- Hypoxia - ischemia.

- Trauma, brain tumors

2. Chest diseases

Infection: Bacterial pneumonia, tuberculosis, mycoplasma, viral.

- Pneumothorax, asthma, cystic fibrosis.

- Positive pressure ventilation.

3. Tumours

- Bronchogenic carcinoma, Hodgkin's disease.

- Acute leukemia, thymoma, lymphosarcoma.

4. Drugs

Chlorpropamide, vincristine, vinblastine, diuretics, clofibrate,

carbamazepine ,barbiturates.

5. Hyper volemic hyponatremia

(i) Odema forming states (UNa <20 mEq/L)

1. CCF, cirrhosis of liver.

2. Neptrotic syndrome.

(ii) Renal failure (UNa > 40 mEq/L)

Acute / Chronic

Pseudohyponatremia

Normally plasma proteins and lipids occupy only 70ml out of each 1000ml of plasma. Plasma water occupies approximately 930ml of each liter of plasma. High levels of plasma lipids or protein increase plasma volume decreasing the percentage that is water. Sodium in most of the laboratories are measured by flame emission spectro-photometry and report of sodium concentration in mEq per liter of plasma not in mEq per liter of plasma water, so sodium concentration will be artificially low. Ion selective electrode method, which measures sodium activity in plasma water, is not affected by the proportion of serum occupied by lipids and proteins. Plasma osmolality will be normal or isotonic in such conditions [14] .

Factitious hyponatremia

Mainly glucose and mannitol cause this condition, Plasma osmolality in this case is abnormally high. For every 100mg per dl, increase in blood glucose or mannitol concentration, the serum sodium concentration will be lowered by impermeant solute is removed from the plasma [14] .

Hypovolemic hyponatremia

The condition in this category are associated with total body sodium depletion and ECF volume contraction owing to losses of sodium is relative excess to water, either through renal or extrarenal routes, or through intracorporeal sequestration, often called the third space effect.

By far the most common cause of hypovolemic hyponatremia is viral gastroenteritis causing vomiting and diarrhoea [20] .

Whattad A et al (1992) had noticed that acute gastroenteritis is the leading cause of hyponatremia, present on admission [16] .

In a study in Bangladesh, it was found that out of 1330 children under the age of 3 years with diarrhea, 276 (20.8%) were hyponatremic. The incidence of hyponatremia was related to the degree of malnutrition [21] .

English MC et al in a prospective study of 47 children with cerebral malaria found that 55% had hyponatremia (serum sodium <135 mmol/L) on admission.Hyponatremia pronounced (serum sodium <130 mmol/L) in 21%. The results suggested that dehydration was common in severe childhood malaria [22] .

Fistula and various types of gastrointestinal tubes for drainage occasionally cause this condition. Excessive sodium losses in sweat are found in patients with cystic fibrosis and adrenal insufficiency [23] .

A similar disturbance occurs when isotonic body fluid is translocated within the body to a third space.

Excessive renal loss of sodium is caused by exogenous (drugs) and endogenous (osmotic) diuretics,mineralcorticodis deficiency and certain primary kidney disorders.

The urine sodium concentration is greater than 20mEq/L and will continue to be so despite the presence of hypovolemia for as long as diuretics are used.

Hyponatremia occurring with ECF volume contraction hyperkalemia and renal sodium wasting without renal failure suggest the possibility of adrenal insufficiency.

Salt wasting sufficient to cause hyponatremia occurs in certain renal disorders. Such as medullary cystic disease, obstructive uropathy and various tubulointerstitial diseases, even in the absence of renal excretory impairment.

Gerigk M et al concluded that hyponatremia occurs in young infants in severe acute pyelonephrits in the absence of obstructive uropathy or vesicouretric reflux. The severe inflammation of the kidney itself causes electrolyte disturbance by transient resistance of the kidney itself causes electrolyte disturbance by transient resistance of the distal tubule to aldosterone [24] .

But **Melzi ML** had outlined that a salt-losing syndrome with tubular resistance to aldosterone can occur during pyelonephritis in young infants with congenital urinary tract malformation[25].

Euvolemic hyponatremia

These patients will have normal or near normal total body sodium,despite the presence of hyponatremia. They lack overt signs of water imbalance, specifically they are not edematous. The symptoms are usually, the central nervous system manifestations of hypotonicity owing to slight ECF volume expansion; urinary sodium concentration is usually greater than 20mEq per litre.

The syndrome of inappropriate secretion of ADH (SIADH) is the most common cause of euvolemic hyponatremia in children.

Shann F et al (1985) observed that 45% of children with pneumonia and 50% of children with bacterial meningitis had hyponatremia. Increased secretion of antidiuretic hormone was common with pneumonia and meningitis [26] .

Similarly in a study in PGI Chandigarh, out of 264 children with pneumonia 27% of cases were associated with hyponatremia. Of all the hyponatremia, 68% were secondary to SIADH. The hospital stay associated with hyponatremia was 60% longer, two-fold increase in complications and 3.5 times higher mortality compared to normonatremic children [27] .

The diagnostic criteria of SIADH are as follows:-

1. Hypotonic hyponatremia.

2. Inappropriate antidiuresis-excretion of urine, with on osmolality higher than would be expected for the degree of hyponatremia.

The urine is never maximally dilute and the osmolality is usually greater than, but may be slightly less than, the corresponding serum osmolality.

3. Normal renal excretory function.

4. Normal adrenal, pituitary, and thyroid function.

5. High urine sodium concentration. This is not an absolute criteria urinary sodium may become low in patients who are severely sodium restricted, or volume depleted.

6. Absence of hypovolemia or dehydration.

7. Absence of edema.

8. The condition is corrected by severe water restriction.

SIADH is a problem of water retention, not sodium depletion. Aggressive sodium administration is appropriate only to relieve neurologic symptoms.

Attempt to correct the hyponatremia with sodium rich solution will cause an increase in urinary sodium excretion and little change in the serum sodium.

Other than treatment of the underlying disease no therapy is indicated to correct the hyponatremia.

Acute water intoxication accounts for the diagnosis of hyponatremia in a few children, most of whom are hospitalized and receiving intravenous fluid. Most affected patients have a superimposed condition that impairs free water excretion. Postoperative patients have increased risk owing to high ADH secretion secondary to pain and emotional stress.

Arieff Al et al in their study had found the incidence of postoperative hyponatremia among 24412 patients were 0.3% (83 cases). The mortality of those afflicted was 8.4% [28] .

Acute water intoxication can occur secondary to ingestion of low solute formula feeds. Hyponatremia affecting infants less the 3-6 months of age can happen due to dilute administration of formula feeds, plus 1 liter of water, a day[29] .

Chronic water intoxication or psychogenic polydipsia leads to hyponatremia occurs in mentally disturbed patients.

Luchins DJ et al studied eight patients with psychogenic polydipsia and found that they had repeated bouts of hyponatremia [30] .

The mechanism resulting in hyponatremia in these patients include the washing out of the normal renal medullary concentrating gradient and perhaps an increased sensitivity to ADH.

Hypervolemic hyponatremia

This includes patients with total body sodium and water in great excess such that they present with pulmonary or peripheral edema. They also have impaired ability to excrete water load, promoting water retention that is proportionately, greater than sodium retention hence, hypotonic hyponatremia.

These patients may be subcategorized into two groups.

1. The generalized edematous states of congestive heart failure, cirrhosis of liver, and the nephritic syndrome.
2. Advanced acute or chronic renal insufficiency.

Schrier RW et al studied the pathogenesis of sodium are water retention in high output and low output cardiac failure, nephrotic syndrome, cirrhosis of liver and found that in generalized edematous patients, hyponatremia is due to decreased effective arterial blood volume [31] .In heart failure decrease effective blood volume is caused by low cardiac output, whereas in cirrhosis of liver, decrease effective arterial blood volume is related to decrease peripheral resistance with arteriovenous shunting and splanchnic venous pooling. The low blood volume found in the nephritic syndrome is a result of low capillary oncotic pressure with resultant loss of fluid from the intravascular to the interstitial space. In each of these disorders, a decline in the effective arterial blood volume activates baroreceptors leading to decreased afferent parasympathetic tone, increased ADH release, renal water retention, dilution of ECF solutes and hyponatremia.

Edematous disorders are characterized by decreased glomerular filtration rate and enhanced proximal tubular reabsorption of fluid by the nephron. The retention of sodium causes the urinary sodium concentration to be less than 20mEq/liter, unless diuretics are being used.

Renal failure

The diseased kidney exhibits a dramatically elevated fractional sodium excretion in efforts to maintain sodium balance. Edema develops when large quantities of sodium are ingested than can be excreted. The ability to excrete water is also impaired, primarily as a result of a dramatic decrease in GFR Hyponatremia occurs when water intake exceeds induced non-renal losses, insensible losses and the maximum volume that can be excreted.

Urinary sodium concentrations are variable, but it usually exceeds 40mEq/L per litre .

Clinical features

Hyponatremia is symptomatic when serum sodium falls rapidly below 120mEq/L [14].

Symptoms

Anorexia, nausea, muscle cramps, lethargy, apathy, disorientation, agitation [32] .

Signs

Clouded sensorium, decreased tendon reflexes, pathologic reflexes, cheyne stokes respiration, hypothermia, pseudobulbar palsy and seizures [32] .

Chronic hyponatremia may cause focal weakness, hemiparesis, ataxia and positive Babinski sign[14].

Gruskin et al studies serum sodium abnormalities in children and found hyponatremia causes shock[33] . Hyponatremia enhances the susceptibility to seizures associated with febrile illness in childhood [34] .

Hugen CA et al had found that the probability of a repeat convulsion within the same febrile period appeared to be significantly related to the serum sodium level. Measurement of the serum sodium is valuable investigation in the child with febrile convulsion [35].

Farrar HC et al in their retrospective chart review of infants found hyponatremia was cause of seizures in 70% of infants younger than 6 months who lacked other findings suggesting a cause. The median temperature was lower in hyponatremic infants than normonatremic patients [36].

Pseudo hyponatremia

True hyponatremia can be differentiated from pseudohyponatremia by using ion-specific electrode measure. Alternatively, serum water sodium concentration can be calculated from the formula.

$$\frac{100 \times \text{observed serum sodium}}{Ws}$$

Where Ws (water content of the serum) equals 99.1 - 1.03 Ls - 0.73Ps.

Ls and Ps are the total serum lipid and protein concentrations expressed as gram perdecilitre of serum and 99.1 represents the volume of serum minus crystalloids [37].

Factitious hyponatremia

Glucose and mannitol increases plasma osmolality. Each 100mg/dl increase in blood glucose or mannitol concentration, serum sodium concentration decreases by 1.6mEq/L2.

$$\textbf{Serum sodium} + 1.6 \times \frac{\textbf{Blood glucose} - 100}{100}$$

MANAGEMENT [4,10]

Hyponatremia with contraction of ECF volume re-expansion with isotonic saline is appropriate therapy. The volume and rate of infusion are dictated by estimates of fluid loss made from the history and physical examination.

In shock, 20ml/kg isotonic saline can be administered rapidly over 1hr or more as needed and then repeated as necessary until blood pressure and peripheral circulation return to normal. Underlying diseases such as renal tubular acidosis and adrenal insufficiency can be treated most effectively by specific replacement therapy.

The number of milliequivalents of sodium necessary to achieve the desired concentration in the patient's blood can be calculated by the following formula [10] :-

Sodium deficit [mEq]

= (Desired S_{Na} – Actual S_{Na}) X Total body water (in L) (0.6 X weight in Kg.)

S_{Na} = serum sodium

Desired Serum Sodium is 130 mEq/L.

0.6 is a constant for total body water.

Hyponatremia should be corrected in 36-48hours. Symptomatic hyponatremia can be corrected by administrating 3% of NaCI or 5% NaCI.

1ml 3% NaCI will give-0.5mEq of sodium.

Sarnaik AD et al had stated that treatment of hyponatremic seizures with routine anticonvulsant may be ineffective and is associated with a considerable incidence of apnea. A rapid increase in the serum sodium concentration by 3 to 5 mmol/L with the use of hypertonic saline is safe and efficacious in managing acute symptomatic hyponatremia [38] .

Mon SOH et al had recommended that 5-6 mE/L of increase of serum sodium in 2-3 hr for the patients who presents with convulsion and does not respond to conventional anticonvulsant therapy [39] .

Sterns RH outlined that emergency treatment of hyponatremia should be reserved for a patients who had no time to fully adapt to the disturbances, when the clinical situation demands, treatment can be initiated by infusing 3% saline at 1 to 2 ml/kg/l hour for 2-3 hours [40] . Rapid correction of hyponatremia that is an increase serum sodium of >2mEq / litre per hour or more than 10-12 mEq/L per day may be dangerous.

As the produced potentially fatal neurologic syndrome known as osmotic demyelination syndrome [41] .

Brunner JE et al (1990) concluded that correction rate of hyponatremia plays on a significant role in the pathogenesis of pontine lesions, in individuals with profound hyponatremia who undergo large increases in sodium concentration as a result of severe initial hyponatremia [42].Rapid correction of hyponatremia leads to central pontine myelionolysis.[42,43,44] ·Demyelinating brain stem lesion manifested as papillary changes, decerebrate posture and coma.

Arieff et al had reported in his studies that brain damage is independent of rate of correction; rapid correction to mildly hyponatremia level is safe. Brain damage is related to magnitude of osmolar change and the presence of hypoxic episode [45] .

Mickel HS et al had found that rapid rise in serum sodium following hyponatremia potentiates an oxidative stress and results in oxidation of cellular proteins leading to myelinolysis [46] . In the euvolemic patients, simple water restriction is treatment of

choice. For the seriously symptomatic patients with SIADH or acute water intoxication, giving Intravenous furosemide followed by intravenous hypertonic saline increases serum sodium concentration. Sodium and potassium loses into the urine must be measured and replaced intravenously,while holding water replacement to a minimum.[14]

Hannon RJ et al had reported that in septicemia hyponatremia and plasma hypo-osmolality occurs due to combination of intracellular shift of sodium and water, and dilution of the extracellular space, probably on the basis of physiological antidiuretic hormone (ADH) secretion. Dextrose is in appropriate, potentially dangerous and should be avoided in these circumstances [47].

For hypervolemic hyponatremic patients, salt and water restriction is appropriate therapy. Hypertonic saline may be necessary to alleviate symptoms of hyponatremia but this maneuver is most successful when combined with rigorous water restriction[14]**.**

Subba Rao SD et al in their study of electrolyte abnormalities in children admitted to pediatric intensive care unit found that out of 305 patients, 29 (9.5%) had hyponatremia 19 (65.5%) were clinically euvolemic, 8 (27.6%) were hyponatremic, and 2 (6.9%) were hypervolemic. The mortality associated with hyponatremia was (20.7%) [48] .

Baron D et al in their study reported that highest mortality (64%) was in the patients with CNS symptoms related to factors other than hyponatremia. Patients with CNS symptoms due to hyponatremia had a mortality rate (9%) similar to that of the patients without CNS symptoms (10%). So hyponatremia appears to be a marker for severe underlying

disease that caries a poor prognosis [49] .

Singhi S et al in their prospective study of 727 sick children found that, the mean duration of hospital stay (7.7 + 0.4 days) among 217 children with serum sodium < 130mEq/L about 30% longer than that of 510 children with serum sodium concentration (> 131 mEq/L) (5.9 + 0.3 days). The mortality rate in children with normal serum sodium concentration (> 131 mEq/L) was 5.3% and the mortality rate was 17% in 47 children with serum sodium <125 mEq/L [50] .

Treatment guidelines for childhood hyponatremia [4,33] :

I. Hyponatremic dehydration

Establish circulatory adequacy with 20-40 ml/kg 0.9% saline or Ringer'sLactate.

A. Symptomatic

Rapidly raise serum sodium by giving 5-6ml 3% saline /kg body weight over 10-15 min. If clinical improvement does not occur, an additional 3-4 ml/kg may be given. Further increase serum sodium at a rate of 0.5mEq/L per hour.

B. Asymptomatic

Provide maintenance and replacement therapy with 5% dextrose plus 0.45% saline over 24 hours. Increase serum sodium at a rate of 0.5mEq/L per hour.

II. Symptomatic hyponatremia

A. Water overload

Rapidly raise serum sodium by giving 5-6ml 3% saline/kg body weight over 10-15min. If clinical improvement does not occur, an additional 3-4 ml/kg may be given. Further increase serum sodium at a rate of 0.5 mEq/L per hour by combining fluid restriction, 0.9% saline, if needed, and spontaneous diuresis.

B. With SIADH

Administer furosemide (1-2mgl/kg) and treat with hypertonic saline as above. Treat the underlying disorder.

III. Chronic, asymptomatic hyponatremia

Treat the underlying cause. Administer isotonic fluids for replacement and maintenance therapy. Avoid increasing serum sodium more than 12 mEq /L per day.

Similarly, a 2-year retrospective case-control study on patients with bronchiolitis admitted in the critical care unit (ICU) had 73% (n = 59) of their subjects eventually developing hyponatremia and from this group 88% required ventilatory support. Results also showed that 39% of those with hyponatremia have been infused with hypotonic intravenous fluids at least 6 hours prior to sodium determination.[51] This correlated disease severity with plasma sodium levels, low sodium levels depicted was consistent with higher morbidity and mortality rates, and longer ICU admission. However, other than having a small sample size, the authors were not able to account for other confounders for the development of hyponatremia such as use of diuretics and type of nasogastric feeding.

A study done by Singhi and co-workers identified that electrolyte disturbances in children with pneumonia were quite common.The most frequent abnormalities reported were hyponatraemia (25%), followed by hypokalemia (12%), hypernatraemia(3.7%) and hyperkalemia(2.0%). They reported that the period of hospitalization prolonged by 60% and mortality increased by 3.5 times when hyponatraemia was associated with pneumonia [50] .

It was in 1920 when Lussky and co-workers described the retention of water in children with pneumonia ,which was found to be associated with an increased blood volume and a low plasma chloride value. These findings are explained by the syndrome of inappropriate secretion of anti-diuretic hormone, which has been described in children with pneumonia[6].

However it was in 1962 the correlation of hyponatraemia and pneumonia in children was first described by Stormont and Waterhouse [52].

Afroditi Sakellaropoulou and co-workers in their study from Greece reported that 33.3% of children with community acquired pneumonia had mild hyponatraemia and 1.9% had moderate hyponatraemia [6] .

Dhavan and colleagues from Chandigharh, reported that hyponatraemia was found in 31% of children with pneumonia and syndrome of inappropriate secretion of antidiuretic hormone was the cause in almost 94% of these cases [53].

Singhi and coworkers from Chandigarh reported that hypokalemia was associated with 12.4% and hyperkalemia in 6.8% of children with pneumonia and it was associated with higher mortality in these sick children [54] .

In Japan, a study done by Kaneko et al found the prevalence of hyponatremia in children with pneumonia to be 38.7%. This was higher as compared to a prevalence rate of 13.3% found in children who had pharyngitis or laryngitis. The conclusion from this study was that, the deeper the site of inflammation in the respiratory tract, the higher the prevalence of hyponatremia [55].

In Italy, a study done to determine the incidence and the risk factors for hyponatremia in children with community acquired pneumonia found out that the commonest pathogens were mycoplasma pneumonia, viruses and streptococcus pneumonia in

descending order. Hyponatremia was present in 45.4% of patients. The children with hyponatremia had significantly higher mean white blood cell counts, neutrophil proportion, C-reactive protein, procalcitonin levels, and initial temperature. The mean sodium concentration was almost identical in the children with pneumococcal, atypical, and viral pneumonia[56].

S.D. Subba rao. et al and Biju Thomas et al analysed 305 patients in St.john's hospital aged between 1 month and 14 years , who were admitted in PICU during the period. Ninety nine (32.45%) had electrolyte abnormalities. Of these 24 (7.9%) had mixed electrolyte imbalance, hyperkalemia was the commonest found in 44 (14.4%)cases, hyponatremia was seen in 11 (3.6%) cases which is second commonest abnormality noted. Of the 99 patients with electrolyte imbalance, 24 (24.2%) expired. In these 24 patients 10 (41.6%) had hyperkalemia , 6(25%) had hyponatremia , 5(20.8%) had hypernatremia , 3(12.5%) had hypokalemia[48] .

SV.S.S. Prasad , Sunit Singhi , K.S.Chugh et al analysed a total of 727 Children admitted in PICU the frequency distribution of serum sodium concentration in 727 children , hyponatremia was present in 217(29.8%) children while severe hyponatremia (serum sodium <125 mEq/l) was found in 47(6.4%). Amongst those with severe hyponatremia in 21 the serum sodium ranged between 121-125 mEq/l. in 17 between 116-120 mEq/l and in 9 patients it was 115mEq/l or less. hypokalemia was found in 101 (13.9%) and 39 (5.4%) had hyperkalemia while 587 had normokalemic . in their study pneumonia and diarrheal disease, each accounted for about 20% cases of hyponatremia. The frequency was

26% in pneumonia and 33% in meningoencephalitic illness, being higher in summer as compared winter seasonal difference was observed. [57]

Lamia. M Al Naama, Jawad Kadhum Abdul –Hassan et al, performed case control study on 150 children (87 boys and 63 girls), of age group between 2 months and 9yrs. 75 of them presented with acute CNS manifestations while the rest were considered as control. Eight of 75 pediatric patients (10.7%) with acute CNS diseases had hyponatremia syndrome, three were diagnosed with inappropriate antidiuretic hormone secretion. The highest percentage of hyponatremia (3 out of 6 patients) was found in patients with intracranial diseases. Four out of 38 patients(15.5%) presented with CNS infections. [58] Hoorn et al found a 30% overall incidence of pNa less than 136mmol/l and a 38% incidence in the ICU. Severe hyponatriemia P_{Na} less than 125mmol/l, was present in 3% of hospitalized patients and occurred during hospitalization in fully half of cases of the cases.[15]

Stelfox et al reported ICU acquired hyponatremia in 11% of patients and that this was associated with an increase in hospital mortality from 16 to 28% [60].

In a prospective study, 27% of patients with infectious diseases admitted to a PICU, were seen to have hyponatremia [50]**.** Other patients with hyponatremia had either diarrheal disease(20%), pneumonia(19%) or central nervous system disorders (12%)(10). **Pizzotti *et al*** showed that 10.8% of patients with hyponatremia had an under-lying neurologic disorder and hyponatremia persisted in 4.3% of them after treatment of the underlying disorder. This could be due to the difference in the pattern of PICU admissions in these studies where only 10.3% were CNS diseases [61] .

MATERIALS & METHODS

MATERIALS AND METHODS

STUDY HOSPITAL

R L Jalappa hospital Kolar,is a tertiary level hospital catering to the local needs of the people of kolar district.

STUDY POPULATION

Children between 1 month to 14 years of age admitted to pediatric intensive care unit during January 2015 to December 2015 will be registered for the study.

STUDY DESIGN : Prospective study

INCLUSION CRITERIA : All patients admitted in pediatric intensive care unit between

1 month to 14 years were included in the study

EXCLUSION CRITERIA : Children discharged against medical advice were not included in the study

SAMPLE SIZE:

Was estimated based on the proportion of hyponatremia in critically ill children from the pilot study in our settings.

Incidence of hyponatremia in critically ill children was 10% from the pilot study.

Sample size was estimated based on the formula

$$\mathbf{N= Z^2\alpha/2\ p\ (1\text{-}p)\ /\ d^2}$$

p = 0.1

1-p = 0.9

d = absolute error = 5%

N = 127 at 95% confidence interval

With 10% as Non response final sample size will be **127 + 13 = 140** critically ill children will be included in the study

STUDY METHODOLOGY :

This was a prospective study on the incidence and outcome of hyponatremia in patients aged 1 month to 14 years old admitted in the pediatric intensive care unit in R L Jalappa hospital, kolar

After obtaining permission from the institutional review board and written informed consent from the parents of the patient all the detailed information was entered in the Performa. Consent was taken from the parents of all the 152 children enrolled in the study to detect the serum sodium levels at the time of admission to the paediatric intensive care unit along with other necessary tests. The only significant risk involved in this study was the development of infection on the blood extraction site. This was avoided by proper handwashing and utilization of the blood extraction kit (in accordance with the policy of pediatric infectious disease section), which was readily available in the paediatric intensive care unit.

The following clinical and demographic data were gathered: 1) case number, 2) age, 3) sex, 4) presenting symptom, 5) systemic examination 6) primary diagnosis, 7) serum sodium level, 8) outcome, and 9) final diagnosis . Single determination of serum sodium was done at time of admission to paediatric intensive care unit and at periodic interval if required. Venous blood samples about 1 to 2 mL were drawn upon admission (together with the other blood work-up done if necessary) and sent for

analysis in RL Jalappa Department of Laboratories. Serum samples were analyzed using VITROS , a machine which utilizes indirect ion-specific electrode potentiometry for sodium determination. Specimens were not subjected to other tests without the permission of the participant and parent/guardian. Apart from these data which were collected on admission, the patient's length of ICU stay, outcome (discharged or died) and whether the patient developed complications due to hyponatremia (seizures and altered sensorium)were also determined.

FIGURE 1 : VITROS , a machine which utilizes indirect ion-specific electrode potentiometry for sodium determination

Statistical analysis:

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square** was used as test of significance for qualitative data.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram and Pie diagram.

p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data. EPI Info (CDC Atlanta), Open Epi, Med calc and Medley's desktop were used to estimate sample size, odds ratio and reference management in the study.

RESULTS

RESULTS AND ANALYSIS

Table 4 : Age distribution of subjects

Age	Frequency	Percent
1month - < 1yr	58	38.2
1 yr - < 5 yr	42	27.6
5 yr - <10 yr	25	16.4
10 yr -14 yr	27	17.8
Total	152	100.0

Majority of subjects in the study were in the age group of 1 month to 1 year (38.2%), followed by 27.6% in 1 to 5 years, 16.4% in 5 to 10 years and 17.8% in >10 years.

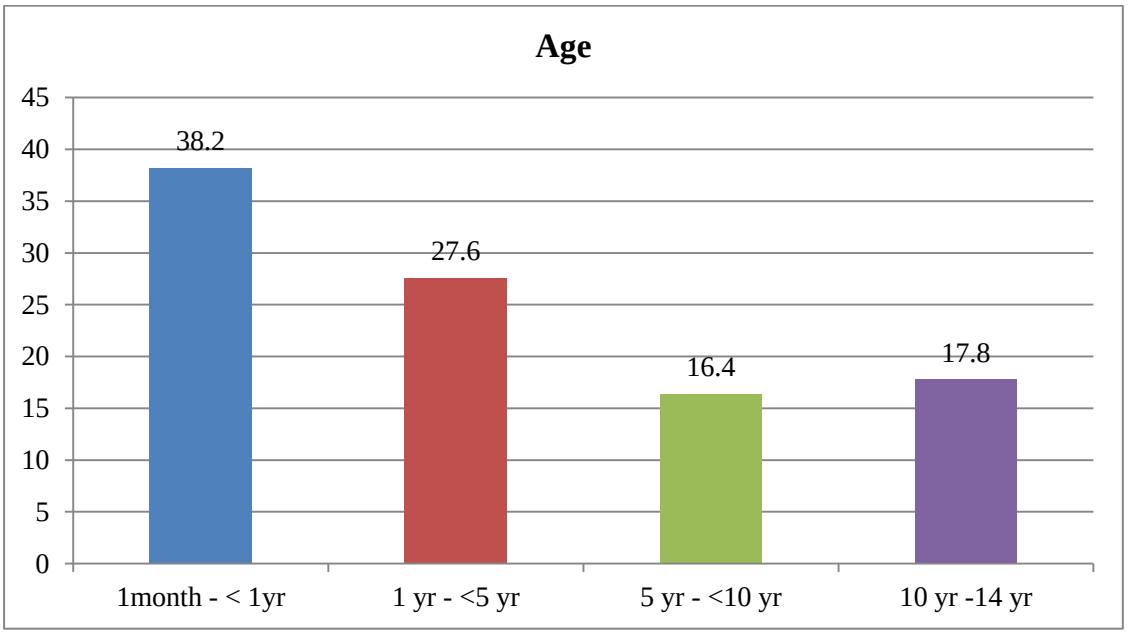


Figure 2: Bar diagram showing Age distribution of subjects

Table 5 : Gender distribution of subjects

Gender	Frequency	Percent
Male	92	60.5
Female	60	39.5
Total	152	100.0

Majority of the subjects were males (60.5%) and 39.5% were females.

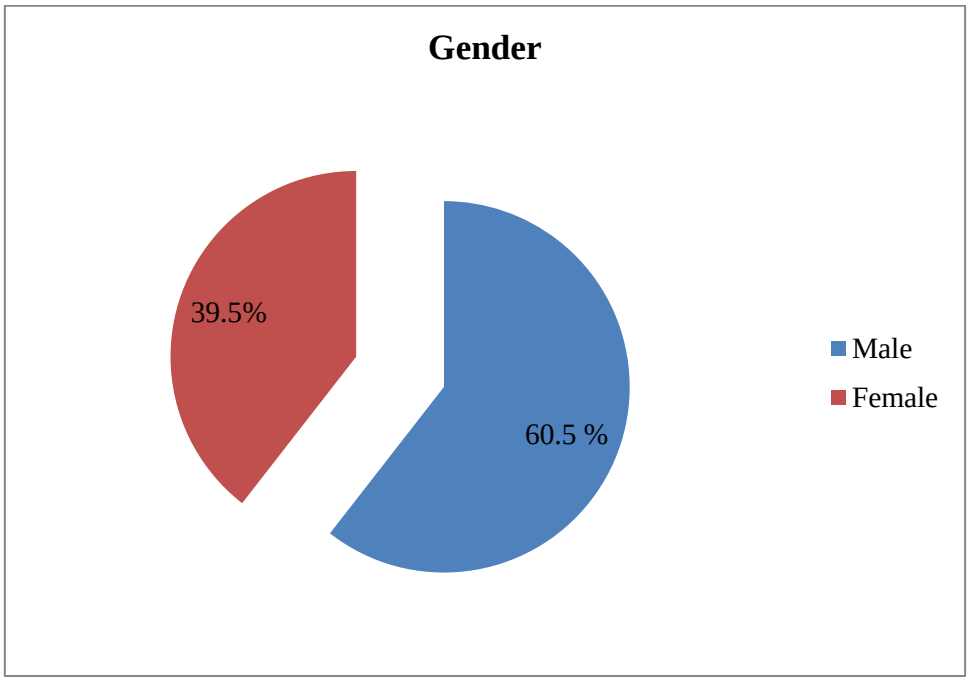


Figure 3 : Pie diagram showing Gender distribution of subjects

Table 6 : Comparison between Age and Gender

	Gender		Total	$\chi 2$ value*	P value
	Male	Female			
1month - < 1yr	38	20	58	1.476	0.688
	65.5%	34.5%	100.0%		
1 yr - < 5 yr	25	17	42		
	59.5%	40.5%	100.0%		
5 yr - <10 yr	15	10	25		
	60.0%	40.0%	100.0%		
10 yr -14 yr	14	13	27		
	51.9%	48.1%	100.0%		
Total	92	60	152		
	60.5%	39.5%	100.0%		

65.5% were males and 34.5% were females in the age group 1 month to 1 year, 59.5% were males and 40.5% were females in the age group 1 to 5 year, 60% were males and 40% were females in the age group 5 to 10 years and 51.9% were males and 48.1% were females in the age group >10 years.

Majority of the subjects were males irrespective of age distribution. There was no significant association between age and gender.

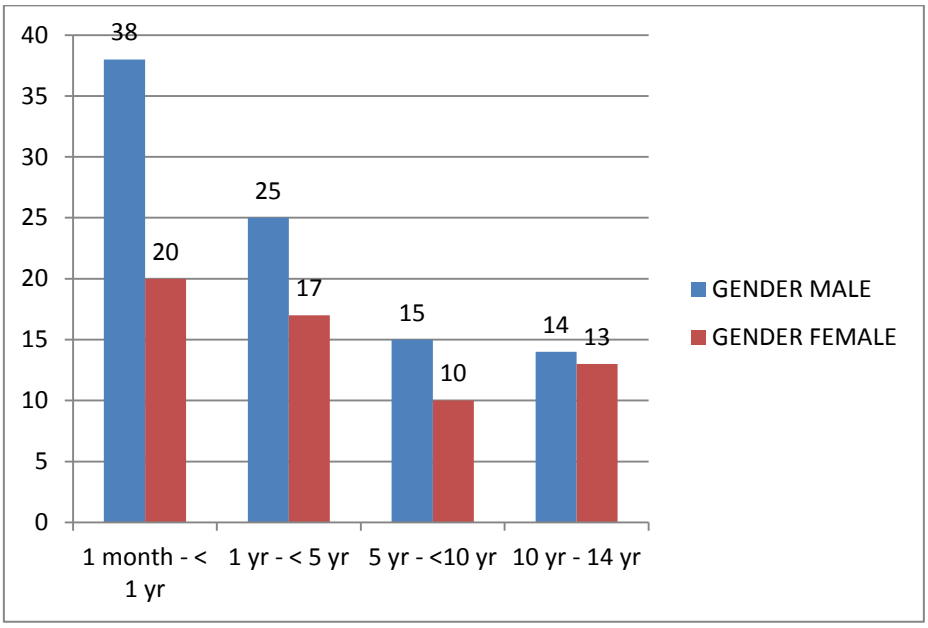


Figure 4 : Bar diagram showing Comparison between Age and Gender

Table 7: Diagnosis among subjects in the study

Diagnosis	Frequency	Percent
Acute Encephalitis	39	25.7
Blunt Trauma ABD	1	0.7
Bronchiolitis	11	7.2
Bronchopneumonia	61	40.1
DF With WS	9	5.9
Drowning	2	1.3
Sepsis With Acute GE	13	8.6
Severe Dengue	16	10.5
Total	152	100.0

In the study majority 40.1% of subjects were diagnosed to have bronchopneumonia, 25.7% had acute encephalitis, 10.5% had severe dengue and others as shown in table 4.

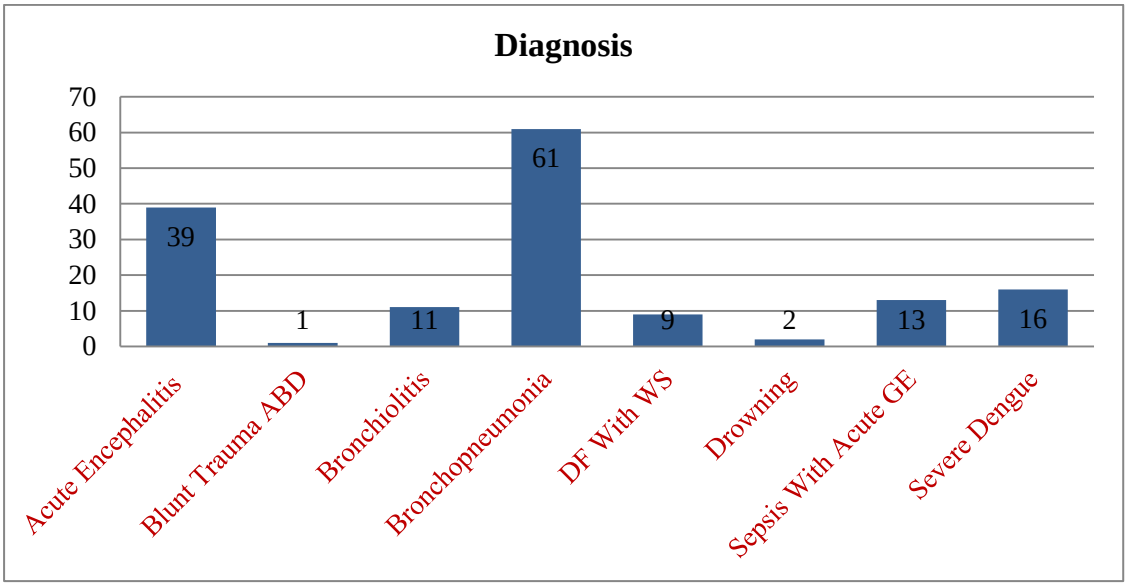


Figure 5: Bar diagram showing distribution of Diagnosis among subjects in the study

Table 8: Percentage of Hyponatremia cases in the study

Group	Frequency	Percent
Normonatremia	101	66.44
Hyponatremia	51	33.56
Total	152	100.0

In the study out of 152 cases, 51 (33.56%) had hyponatremia and 101 (66.44%) had no hyponatremia.

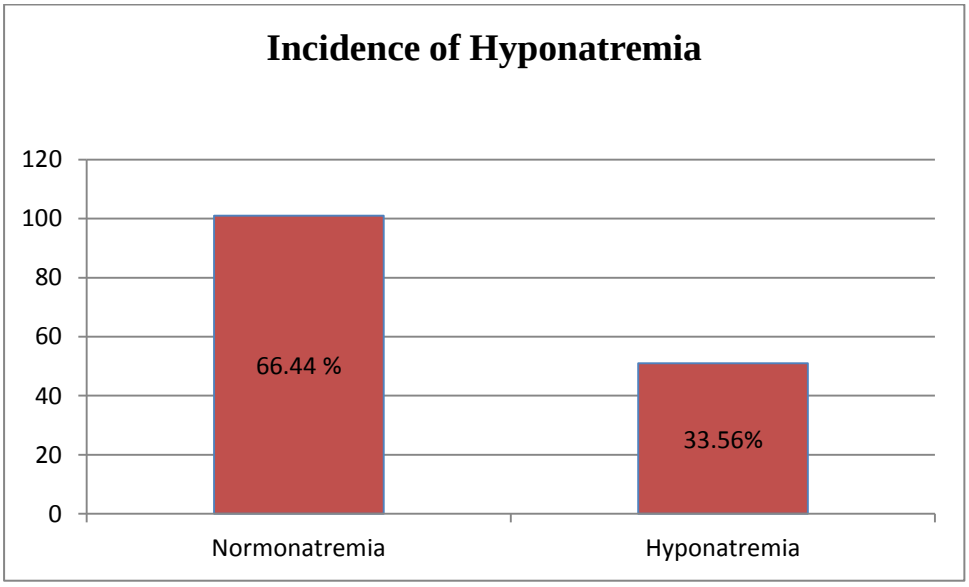
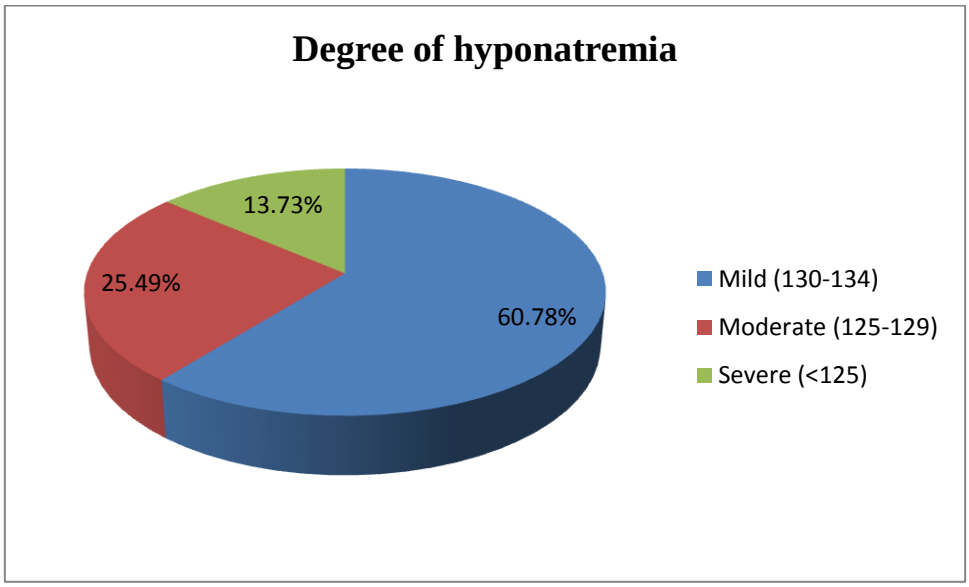


Figure 6 : Bar diagram showing Percentage of Hyponatremia cases in the study

Table 9 : Degree of Hyponatremia among subjects

Degree of Hyponatremia	Frequency	Valid Percent
Mild (130-134)	31	60.78
Moderate (125-129)	13	25.49
Severe (<125)	7	13.73
Total	51	100.0

In the study majority 25.49 % had moderate hyponatremia, 60.78 % had mild hyponatremia and 13.73 % had severe hyponatremia.



Figre 7: Pie chart showing Degree of Hyponatremia

Table 10 : Association between Hyponatremia and age distribution

Group	Age				Total	χ^2 value*	P value
	1month - < 1yr	1 yr - <5 yr	5 yr - <10 yr	10 yr - 14 yr			
Normonatremia	34	30	15	22	101	5.264	0.15
	33.8%	29.8%	14.8%	21.6%	100.0%		
Hyponatremia	24	12	10	5	51		
	47.1 %	23.5 %	19.6%	9.8 %	100.0%		

*Chi Square test

In the study among subjects with hyponatremia, 47.1 % were in the age group 1 month to 1 year, 23.5% were in the age group 1 to 5 years, 19.6% were in the age group 5 to 10 year and 9.8% were in the age group 10 to 14 years. Similarly among subjects with Normonatremia, 33.8% were in the age group 1 month to 1 year, 29.8% were in the age group 1 to 5 years, 14.8% were in the age group 5 to 10 year and 21.6% were in the age group 10 to 14 years.

There was no significant association between Hyponatremia and age distribution.

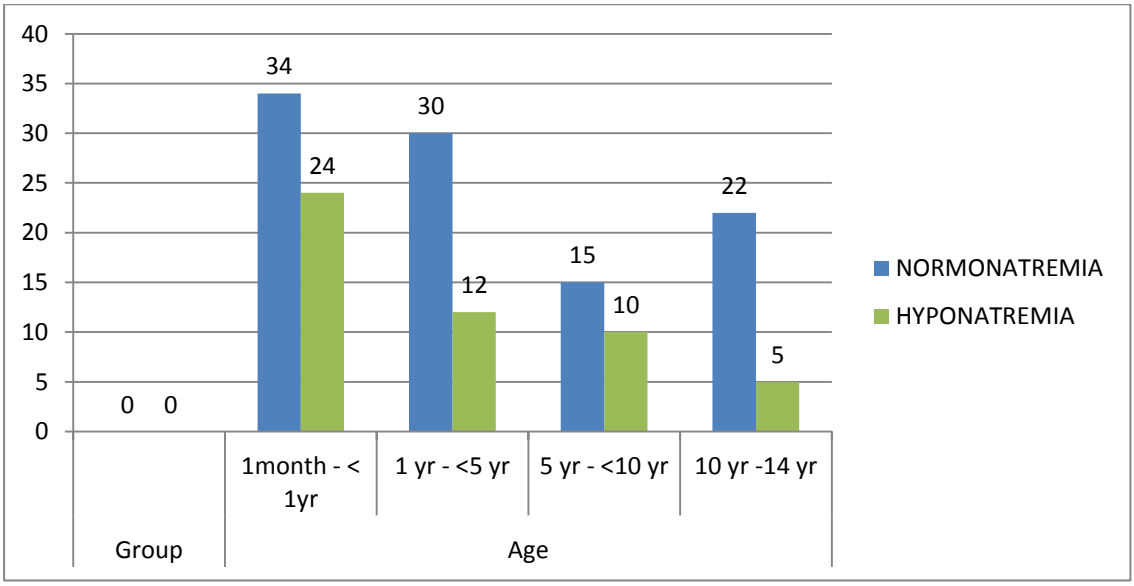


Figure 8 : Bar diagram showing Association between Hyponatremia and age distribution

Table 11 : Association between Hyponatremia and Gender

	Gender		Total	$\chi 2$ value*	P value
	Male	Female			
Normonatremia	62	39	101	0.093	0.76
	61.4%	38.6%	100.0%		
Hyponatremia	30	21	51		
	58.8%	41.2%	100.0%		

Among subjects with Normonatremia 61.4% were males and 38.6% were females, similarly among subjects with hyponatremia 58.8% were males and 41.2% were females. There was no significant association between hyponatremia and gender.

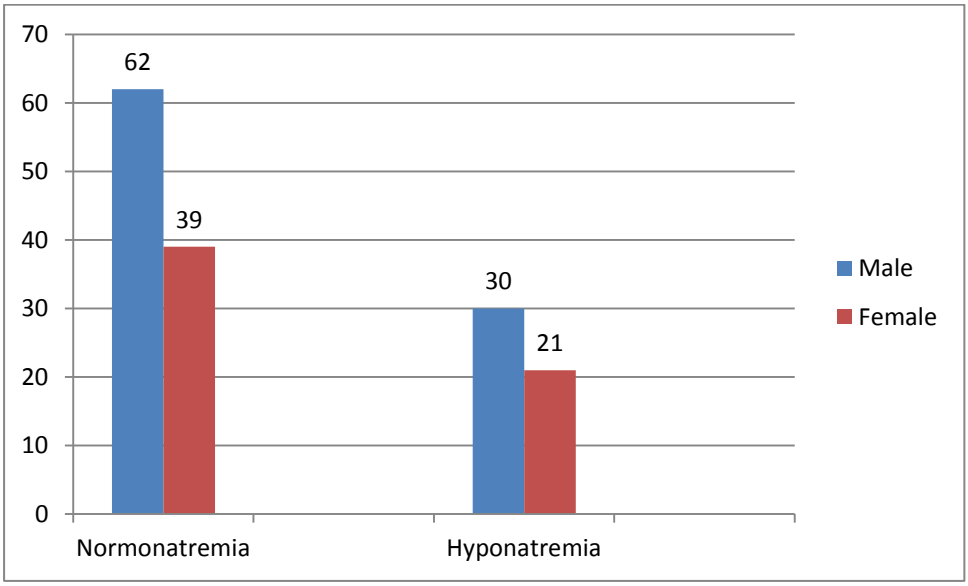


Figure 9 : Bar diagram showing Association between Hyponatremia and Gender

Table 12 : Association between diagnosis and Hyponatremia

	Diagnosis								Total	χ2 value*	P value
	Acute Encephalitis	Blunt Trauma Abd	Bronchiolitis	Bronchopneumonia	DF With WS	Drowning	Sepsis With Acute GE	Severe Dengue			
Normonatremia	23	1	5	51	6	0	6	9	101	18.82	0.0087
	22.8%	1%	5%	50.5 %	6%	0 %	6%	9%	100 %		
Hyponatremia	16	0	6	10	3	2	7	7	51		
	24.0%	0.8%	5.8%	41.3 %	5.8%	1.7 %	8.3 %	12.4 %	100.0%		

Among subjects with Hyponatremia and Normonatremia majority of the subjects had Bronchopneumonia (41.3 % and 50.5 % respectively). Second most common diagnosis in both hyponatremia and Normonatremia was acute encephalitis (24% and 22.8% respectively). There was significant association between diagnosis and hyponatremia among subjects.

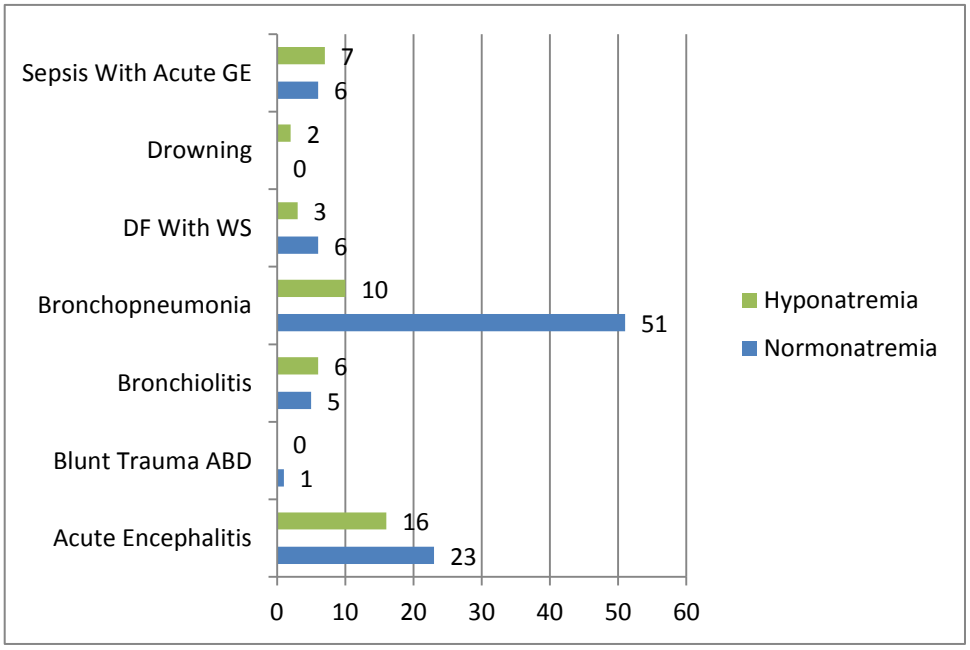


FIGURE 10 :Bar diagram showing the association between diagnosis and hyponatremia

Table 13 : Association between Diagnosis and degree of hyponatremia among subjects

Diagnosis	Degree of Hyponatremia			Total
	Mild (130-134)	Moderate (125-129)	Severe (<125)	
Acute Encephalitis	11	3	2	16
	68.8%	18.7%	12.5%	100.0%
Blunt Trauma ABD	0	0	0	0
	0.0%	0.0%	0.0%	0.0%
Bronchiolitis	2	3	1	6
	33.3%	50%	16.7%	100.0%
Bronchopneumonia	7	2	1	10
	70%	20.0%	10%	100.0%
DF With WS	3	0	0	3
	100.0%	0.0%	.0%	100.0%
Drowning	0	2	0	2
	.0%	100.0%	.0%	100.0%
Sepsis With Acute GE	5	2	0	7
	71.5 .0%	28.5%	.0%	100.0%
Severe Dengue	3	1	3	7
	42.9%	14.2%	42.9%	100.0%
Total	31	13	7	121
	25.6%	10.7%	63.7%	100.0%

Among subjects with acute encephalitis 68.8 % had mild, 18.7% had moderate and 12.5% had severe hyponatremia.

In Blunt trauma ABD subjects, no hyponatremia

In Bronchiolitis subjects 33.3% had mild, 50% had moderate and 16.7% had severe hyponatremia.

In Bronchopneumonia subjects 70% had mild, 20% had moderate and 10% had severe hyponatremia.

In dengue fever subjects 100% had mild and none had moderate and severe hyponatremia.

In Drowning subjects 100% had moderate hyponatremia.

In Sepsis with acute GE, 71.5% had mild, 28.5% had moderate hyponatremia and none had severe hyponatremia

Lastly among subjects with severe dengue fever 42.9% had mild, 14.2% had moderate and 42.9% had severe hyponatremia.

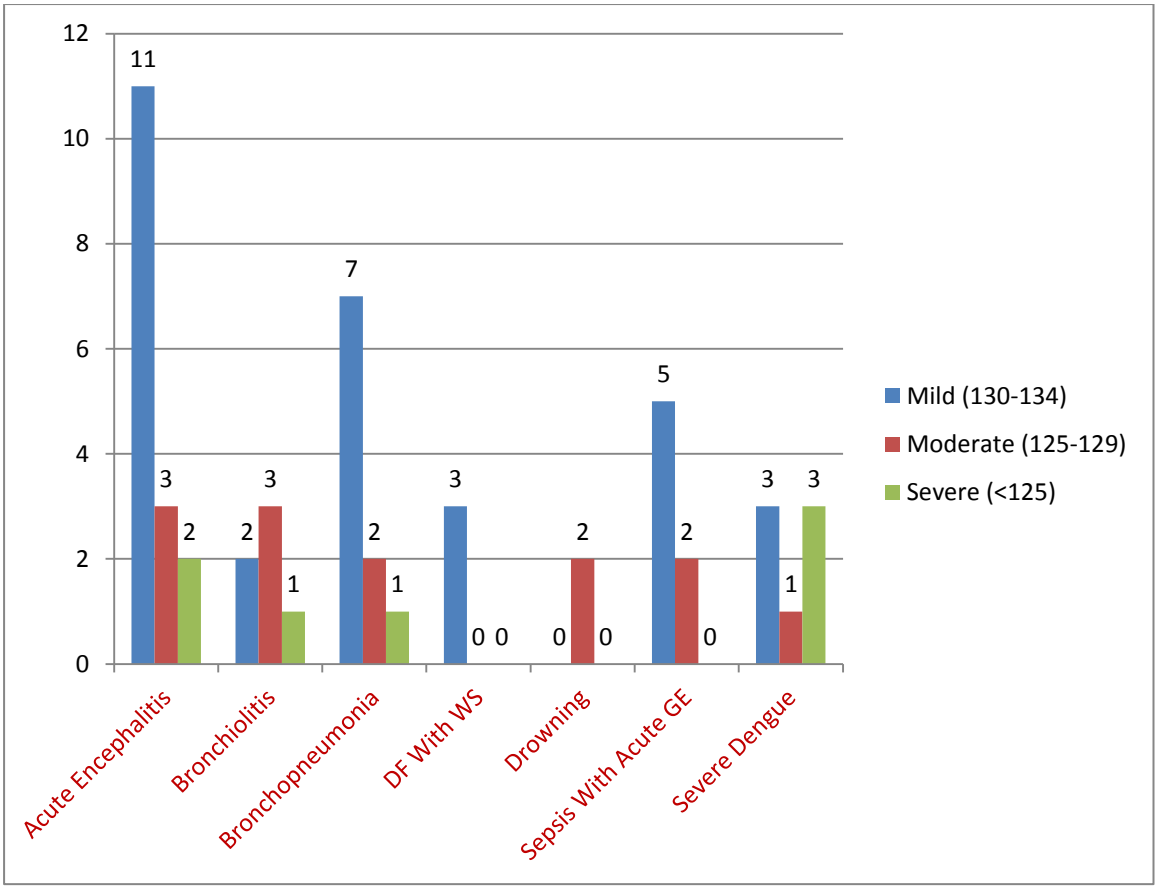


Figure 11 : Bar diagram showing Association between Diagnosis and degree of hyponatremia among subjects

Table 14 : Morbidity distribution of subjects

Morbidity	Frequency	Percent
Yes	72	47.4
No	80	52.6
Total	152	100.0

47.4% of subjects in the study had morbidity and 52.6% had no morbidity.

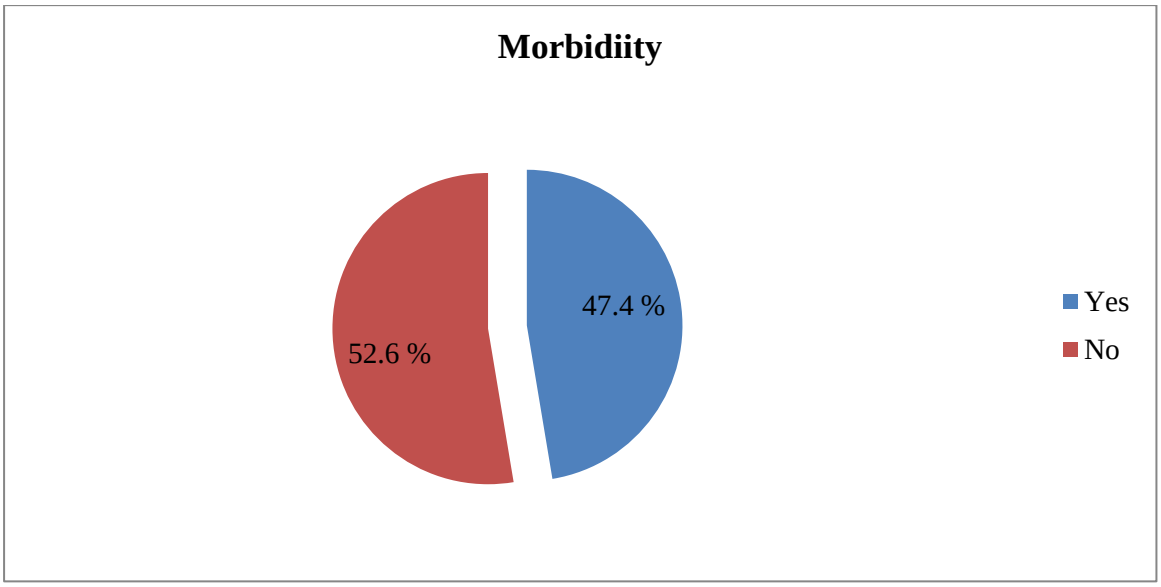


Figure 12 : Pie diagram showing Morbidity distribution of subjects

TABLE 15 : ASSOCIATION BETWEEN MORBIDITY AND AGE DISTRIBUTION

Morbidity	Age				Total	χ^2 value*	P value
	1month - < 1yr	1 yr - <5 yr	5 yr - <10 yr	10 yr - 14 yr			
Yes	29	18	14	11	72	1.727	0.631
	40.3%	25.0%	19.4%	15.3%	100.0%		
No	29	24	11	16	80		
	36.3%	30.0%	13.8%	20.0%	100.0%		
Total	58	42	25	27	152		
	38.2%	27.6%	16.4%	17.8%	100.0%		

Among subjects with morbidity, 40.3% were in the age group 1month to 1 year, 25% in the age group 1 to 5 year, 19.4% in the age group 5 to 10 years and 15.3% were in the age group 10 to 14 years. There was no significant association between morbidity and age distribution.

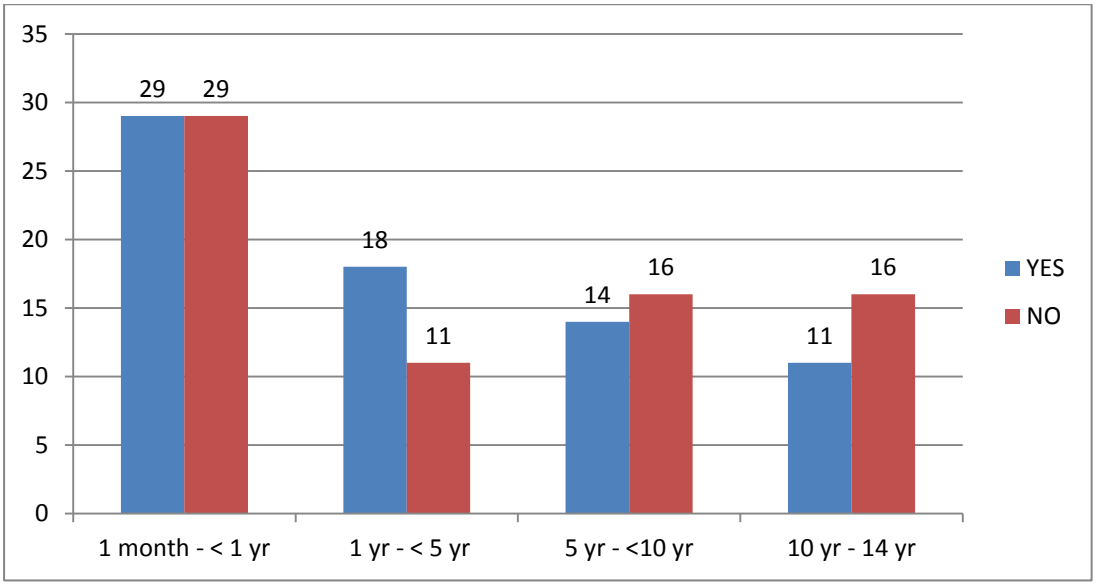


Figure 13 : Bar diagram showing Association between Morbidity and age distribution

TABLE 16 : Association of Morbidity with Hyponatremia

Morbidity	Frequency	Percent
Yes	40	78.4
No	11	21.6
Total	51	100.0

In the hyponatremic subjects ,78.4% of subjects had morbidity and 21.6% had no morbidity.

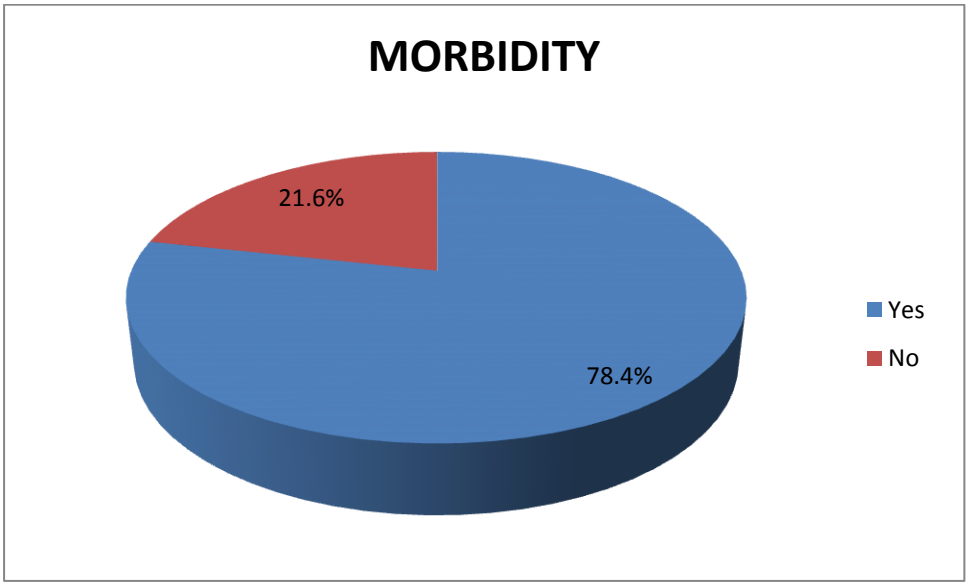


FIGURE 14: PIE DIAGRAM SHOWING ASSOCIATION OF MORBIDITY WITH HYPONATREMIA

TABLE 17 : Association between diagnosis and morbidity

	Morbidity		Total	$\chi 2$ value*	P value
	Yes	No			
Acute Encephalitis	22	17	39	7.951	0.337
	30.6%	21.3%	25.7%		
Blunt Trauma ABD	0	1	1		
	.0%	1.3%	.7%		
Bronchiolitis	5	6	11		
	6.9%	7.5%	7.2%		
Bronchopneumonia	32	29	61		
	44.4%	36.3%	40.1%		
DF With WS	2	7	9		
	2.8%	8.8%	5.9%		
Drowning	0	2	2		
	.0%	2.5%	1.3%		
Sepsis With Acute GE	5	8	13		
	6.9%	10.0%	8.6%		
Severe Dengue	6	10	16		
	8.3%	12.5%	10.5%		
Total	72	80	152		
	100.0%	100.0%	100.0%		

Out of 72 subjects who had morbidity during the course of treatment, 30.6% had acute encephalitis, 6.9% had bronchiolitis, 44.4% had bronchopneumonia, 2.8% had DF with WS, 6.9% had sepsis with GE and 8.3% severe dengue. There was no significant association between diagnosis and morbidity.

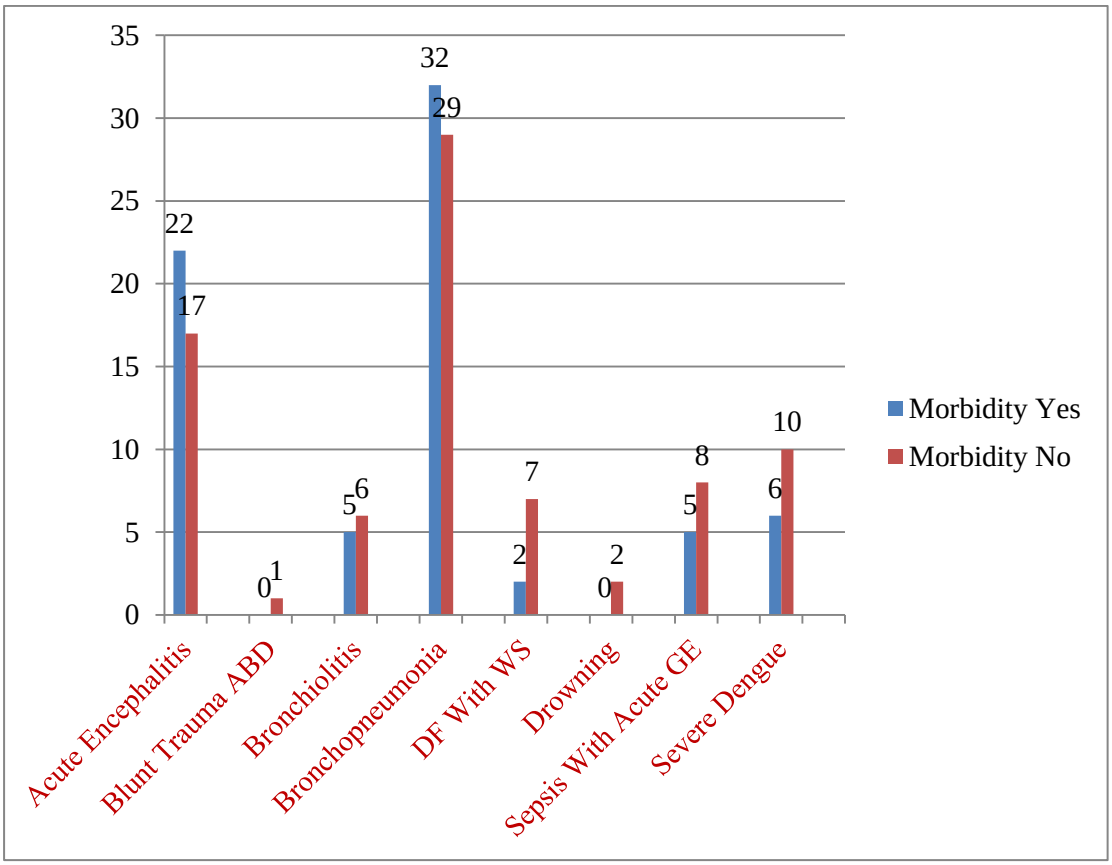


Figure 15 : Bar diagram showing association between diagnosis and morbidity

Table 18 : Seizures incidence among subjects

Seizures	Frequency	Percent
Yes	19	12.5
No	133	87.5
Total	152	100.0

12.5% of subjects in the study had seizure episodes.

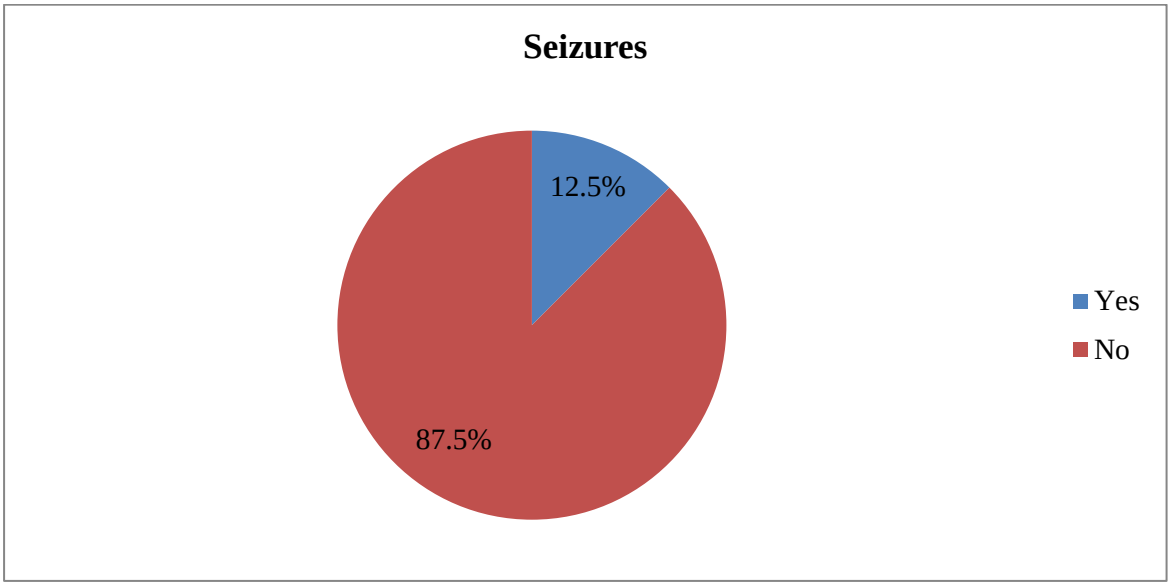


Figure 1: Pie diagram showing Seizures incidence among subjects

Table 19 : Incidence of seizures in Hyponatremia cases

Seizures	Frequency	Percent
Yes	12	23.5
No	39	76.5
Total	51	100.0

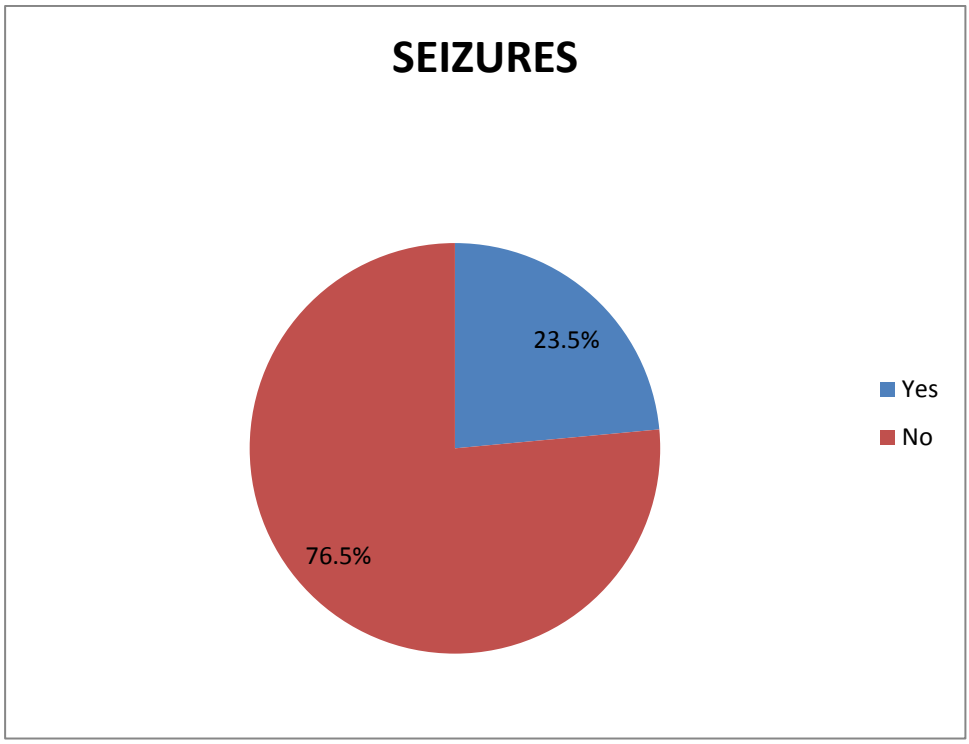


Figure 17 : Pie diagram showing Incidence of seizures in Hyponatremic cases

Table 20 : Altered Sensorium incidence among subjects

Altered Sensorium	Frequency	Percent
Yes	39	25.7
No	113	74.3
Total	152	100.0

25.7% of study subjects had altered Sensorium.

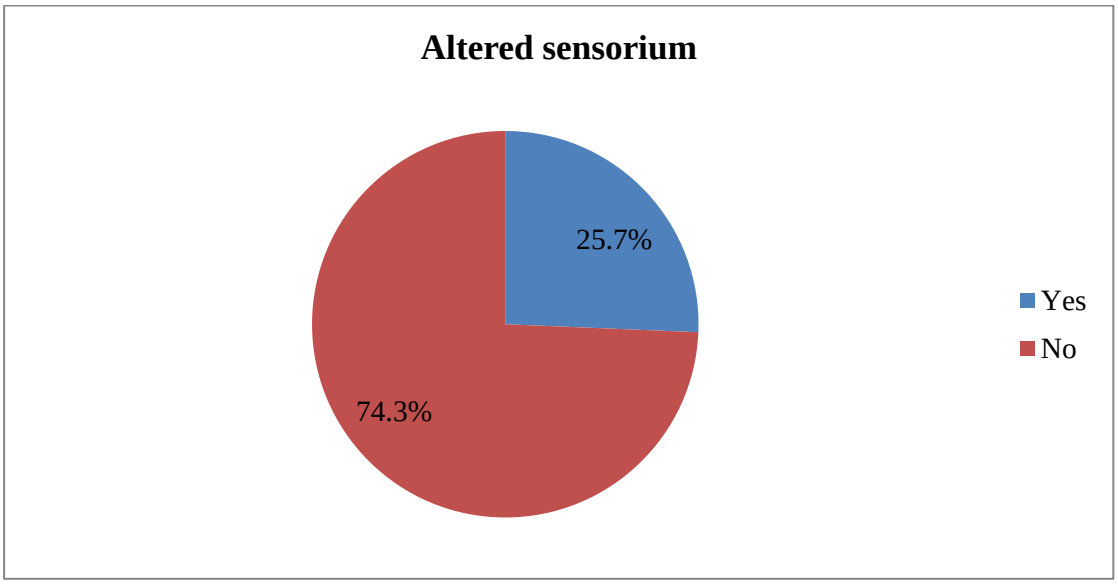


Figure 18 : Pie diagram showing Altered Sensorium incidence among subjects

Table 21 : Incidence of Altered sensorium among Hyponatremic cases

Altered Sensorium	Frequency	Percent
Yes	20	39.2
No	31	60.8
Total	51	100.0

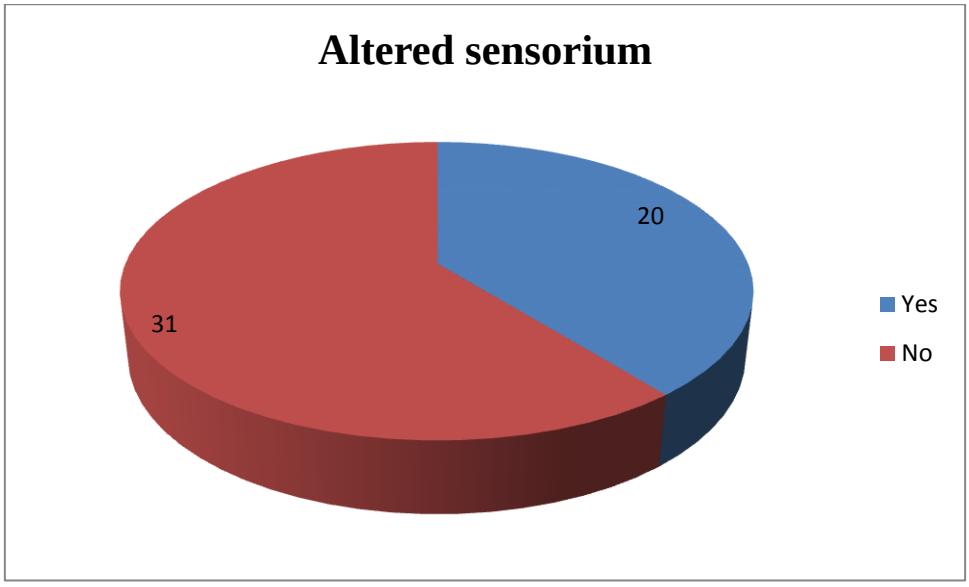


Figure 19 : Pie diagram showing Incidence of Altered sensorium among Hyponatremic cases

Table 22 : Distribution of subjects according to Mortality

Mortality	Frequency	Percent
Yes	7	4.6
No	145	95.4
Total	152	100.0

In the study 4.6% had mortality and 95.4% improved during the course of treatment.

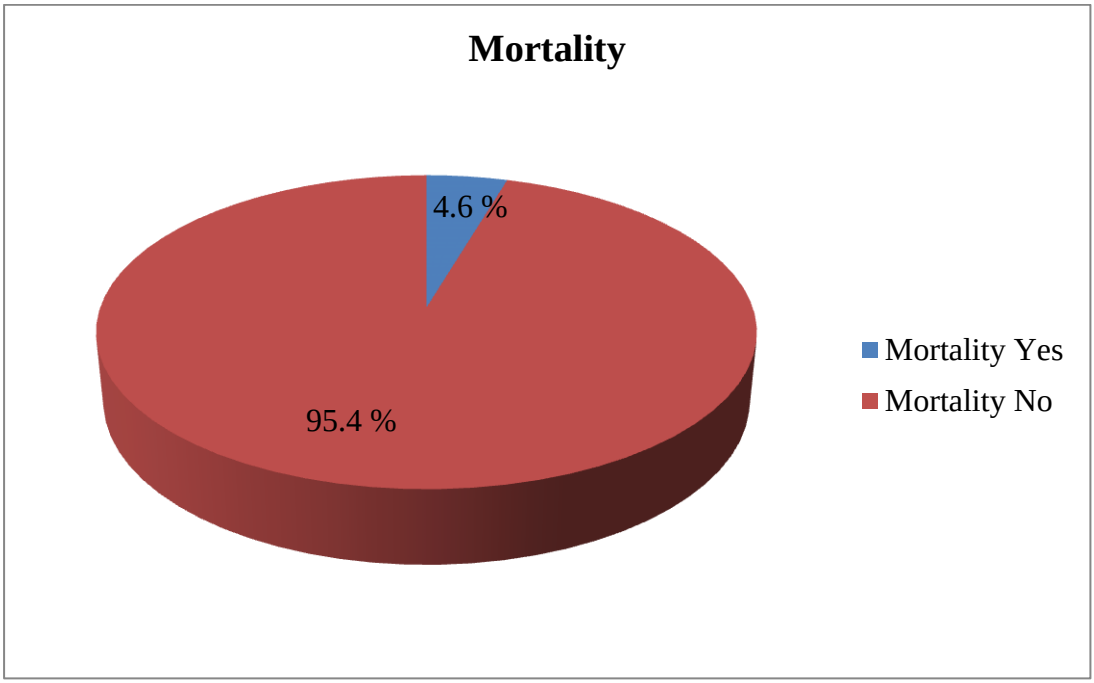


Figure 2: Pie diagram showing Distribution of subjects according to Mortality

Table 23 : Association between Mortality and age distribution

Mortality	Age				Total	χ^2 value*	P value
	1month - < 1yr	1 yr - <5 yr	5 yr - <10 yr	10 yr - 14 yr			
Yes	4	1	0	2	7	2.856	0.414
	57.1%	14.3%	.0%	28.6%	100.0%		
No	54	41	25	25	145		
	37.2%	28.3%	17.2%	17.2%	100.0%		
Total	58	42	25	27	152		
	38.2%	27.6%	16.4%	17.8%	100.0%		

Among subjects with mortality, 57.1% were in the age group 1month to 1 year, 14.3% in the age group 1 to 5 year, 0% in the age group 5 to 10 years and 28.6% were in the age group 10 to 14 years. There was no significant association between mortality and age distribution.

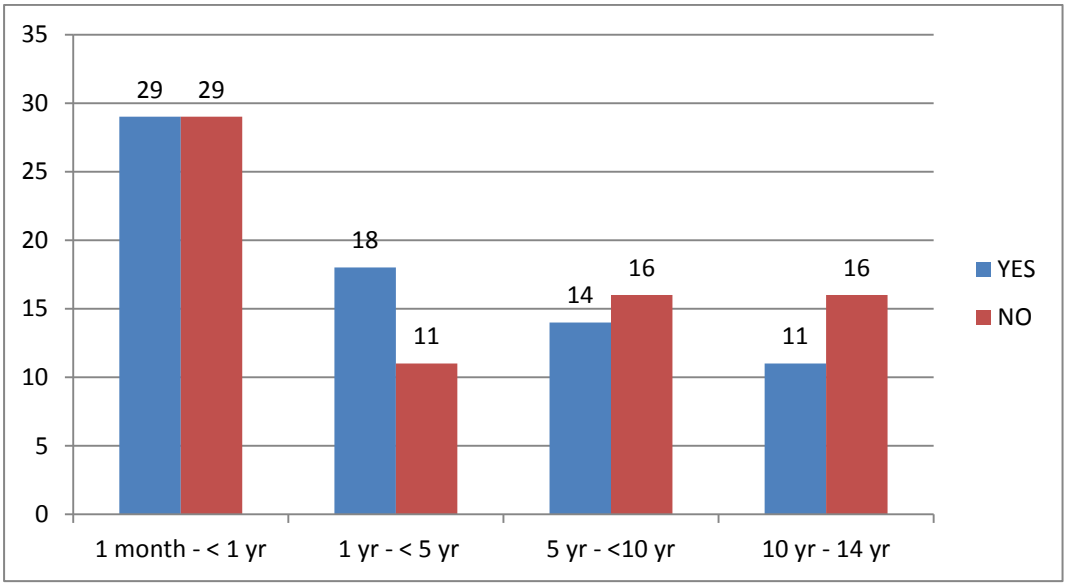


Figure 21 : Bar diagram showing Association between Mortality and age distribution

Table 24 : Association between Mortality and Diagnosis

	Mortality		Total	$\chi 2$ value*	P value
	Yes	No			
Acute Encephalitis	2	37	39	11.363	0.124
	28.6%	25.5%	25.7%		
Blunt Trauma ABD	0	1	1		
	.0%	.7%	.7%		
Bronchiolitis	1	10	11		
	14.3%	6.9%	7.2%		
Bronchopneumonia	2	59	61		
	28.6%	40.7%	40.1%		
DF With WS	0	9	9		
	.0%	6.2%	5.9%		
Drowning	1	1	2		
	14.3%	.7%	1.3%		
Sepsis With Acute GE	0	13	13		
	.0%	9.0%	8.6%		
Severe Dengue	1	15	16		
	14.3%	10.3%	10.5%		
Total	7	145	152		
	100.0%	100.0%	100.0%		

Out of 7 subjects who died during the course of treatment, 28.6% had acute encephalitis, 14.3% had bronchiolitis, 28.6% had bronchopneumonia, 14.3% had drowning, sepsis with GE and severe dengue. There was no significant association between diagnosis and mortality.

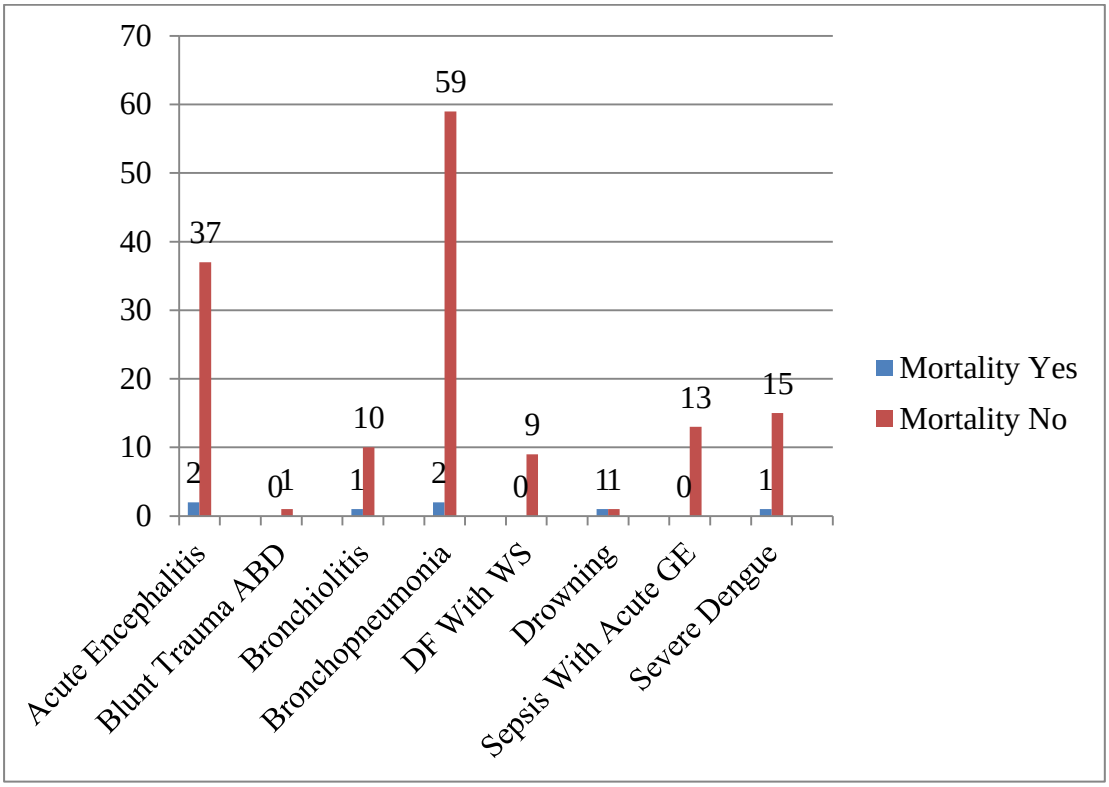


Figure 22: Bar diagram showing Association between Mortality and Diagnosis

Table 25 : Incidence of Mortality among Hyponatremia cases

Mortality	Frequency	Percent
Yes	4	7.8%
No	47	92.2%
Total	51	100.0

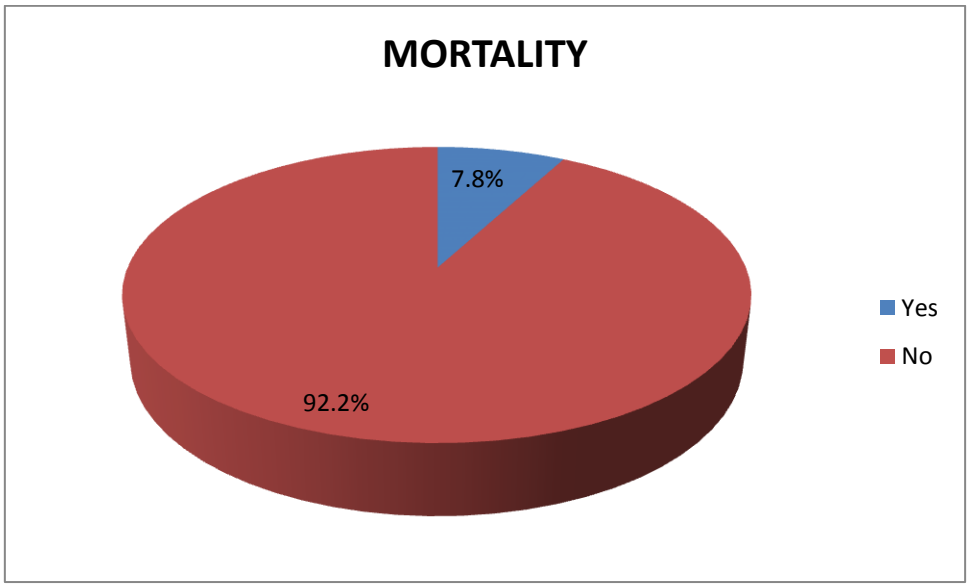


Figure 23 : Pie diagram showing Incidence of Mortality among Hyponatremia cases

DISCUSSION

DISCUSSION

The present study was conducted from January 2015 to December 2015. In this period 152 cases were studied swho was admitted to Pediatric intensive care unit with various illness. Serum sodium concentration was estimated in these children at the time of admission.

Out of 152 cases admitted in our hospital , majority of the subjects were males (60.5%) and 39.5% were females.

Majority of the subjects in the study were in the age group of 1 month to 1 year (38.2%), followed by 27.6% in 1 to 5 years, 16.4% in 5 to 10 years and 17.8% in >10 years.

In our study , majority of PICU admission were in the age group of 1month to 1 year. This can be supported by the studies done by **Prasad et al**[3] and **Subba Rao et al**[48] where the mean age was 2-14 years and 4.09 years respectively.

In the study out of 152 cases,51 (33.5%) had hyponatremia and 101 (66.5%) had normonatremia .Hyponatremia was defined as less than 135 mEq/L in our study [4]. One third of patients had hyponatremia at admission to PICU. and further administration of hypoosmotic fluids is more likely to increase the proportion of children who will have hyponatremia. When compared to other reported data in children by **Sunit Singhi *et al*** who reported 29.8% incidence of hyponatremia, the present study showed higher incidence [3]. **Anderson RJ et al** (1986) found clinically significant symptomatic hyponatremia (serum sodium less than 130mEq/L) was a frequent occurrence with incidence 1-2% of total hospitalized patients with acute or chronic illness [15] .**Wattad A et al** in (1992) observed that, out of 11,702 total hospital

admissions, 161 patients were hyponatremic (serum sodium less than 130 mEq/l), an overall frequency of 1.38% [16].

In our study ,we have calculated incidence only to critically ill children admitted to PICU, hence clinical significant incidence of hyponatremia was high in our study.

In the present study there was no significant age or sex difference in frequency of hyponatremia.

Out of 152 admissions, the causes of admission are Bronchopneumonia(40.1%), Acute Encephalitis(25.7%), Severe Dengue(10.5%), Sepsis With Acute GE(8.6%), DF With WS(5.9%), Bronchiolitis(7.2%),Drowning(1.3%) and Blunt Trauma abdomen(0.7%).

The most common association with hyponatremia were pneumonia, encephalitis and acute gastroenteritis. The proportion of cases with hyponatremia across various diagnostic categories ranged from 0.8 to 41.3 %.

In a recent study by **Don M *et al***, hyponatremia was found in 45.4% of children with community acquired pneumonia incidence of which was not similar to present study [7]. 19.6 % children with pneumonia had hyponatremia in our study. A study done by Singhi and co-workers identified that electrolyte disturbances in children with pneumonia were quite common. The most frequent abnormality reported was hyponatraemia (25%) [62]. **Shann F et al** (1985) observed that 45% of children with pneumonia and 50% of children with bacterial meningitis had hyponatremia. Increased secretion of antidiuretic hormone was common with pneumonia and meningitis [26]. Similarly in a study in PGI Chandigarh, out of 264 children with pneumonia 27% of cases were associated with hyponatremia. Of all the

hyponatremia, 68% were secondary to SIADH [27]. In another study, mild hyponatremia was found in 70 % of hyponatremia and moderate hyponatremia was found in 20 % of children with pneumonia [6] **.**

Encephalitis was the 2nd most common cause of hyponatremia in our study accounting to about 31.3%(16/51) of all cases of hyponatremia. Incidence of hyponatremia among acute encephalitis cases was 41 %(16/39) in our study. In a work by **Bussmann C** 10.3% of children with various acute central nervous system disorders had sodium level below 130mmol/L [20].In another study by **von Vigier RO *et al*** who studied circulating sodium in acute meningitis and found that 32.3% (97 / 300) children with meningitis had sodium level below 133 mmol/L [63] . **Lamia et al**, performed case control study on 150 children (87 boys and 63 girls), of age group between 2 months and 9yrs. 75 of them presented with acute CNS manifestations while the rest were considered as control. Eight of 75 pediatric patients (10.7%) with acute CNS diseases had hyponatremia syndrome, three were diagnosed with inappropriate antidiuretic hormone secretion. The highest percentage of hyponatremia (3 out of 6 patients) was found in patients with intracranial diseases. Four out of 38 patients(15.5%) presented with CNS infections [58].

In a study done by **Shann and Germer** had found 50% of cases with bacterial meningitis had hyponatremia [26] . Similar study was done by **Subba Rao et al** with 41.4% (12/29) cases of hyponatremia suffering from central nervous system diseases [48] .**Prasad et al** have found 21% of children with central nervous system disease (meningo encephalitis) had hyponatremia.[3]

In a recent published literature by **Shah GS *et al*** hyponatremia was present in 56% of children presenting with diarrhea[7]. It was much higher than present study where 53.8% (7/13) of children with diarrhea had hyponatremia. By far the most common cause of hypovolemic hyponatremia is viral gastroenteritis causing vomiting and diarrhoea [3] .**Whattad A et al** (1992) had noticed that acute gastroenteritis is the leading cause of hyponatremia, present on admission [16] . In a study in Bangladesh, it was found that out of 1330 children under the age of 3 years with diarrhoea, 276 (20.8%) were hyponatremic. The incidence of hyponatremia was related to the degree of malnutrition [21] .Incidence of hyponatremia among Acute gastroenteritis cases was high in our study as we admit only acute gastroenteritis with severe dehydration cases to PICU.

Morbidity

The morbidity, as determined by the PICU stay(more than 5 days) was significantly higher in patients with hyponatremia when compared to those with normonatremia. This observation is similar to the study done by prasad et al **[50]** and singhi et al [27] The severity of the underlying disease may also contribute to prolonged PICU stay. They reported that the period of hospitalization prolonged by 60% and mortality increased by 3.5 times when hyponatraemia was associated with pneumonia [62] .

In the study done by **singhi et al** ,the hospital stay associated with hyponatremia was 60% longer and two-fold increase in complications [27]. **Singhi S et al** in their prospective study of 727 sick children found that, the mean duration of hospital stay (7.7 + 0.4 days) among 217 children with serum sodium < 130mEq/L about 30% longer than that of 510 children with serum sodium concentration (> 131 mEq/L) (5.9 + 0.3 days) [50].

Incidence of seizure and altered sensorium among hyponatremic group was 23.5% and 39.2% respectively in our study. These complications are seen only in the moderate and severe hyponatremia group. **Farrar HC et al** in their retrospective chart review of infants found hyponatremia was cause of seizures in 70% of infants younger than 6 months who lacked other findings suggesting a cause. The median temperature was lower in hyponatremic infants than normonatremic patients [36] .

Mortality

Incidence of mortality in hyponatremic group was 7.8% (4/51) but hyponatremia cannot be attributed to mortality as the confounding factors are not excluded. **Baron D et al** in their study reported that highest mortality (64%) was in the patients with CNS symptoms related to factors other than hyponatremia. Patients with CNS symptoms due to hyponatremia had a mortality rate (9%) similar to that of the patients without CNS symptoms (10%). So hyponatremia appears to be a marker for severe underlying disease that carries a poor prognosis prognosis [49] **.**The risk of mortality is increased by 3-3.5 times in patients with hyponatremia when compared to those with normal serum sodium in a study done by prasad et al [50] . In a study done by **singhi et al**, 3.5 times higher mortality in the hyponatremic children compared to normonatremic children [27]**.** In a study done by Prasad et al , mortality rate in children with normal serum sodium concentration (> 131 mEq/L) was 5.3% and the mortality rate was 17% in 47 children with serum sodium <125 mEq/L [50] **.**

LIMITATION OF THE STUDY :

- Small sample size
- Patients were not worked up for the primary cause of hyponatremia
- Further studies are required to find out underlying mechanism of hyponatremia in various clinical states and to design rational fluid protocols for sick children.

CONCLUSION

CONCLUSION :

- This study shows that hyponatremia (33.5%) occurs frequently in children requiring critical care management and thus should be anticipated and given attention in management plan along with treating the underlying cause.
- One third of patients had hyponatremia at admission to PICU. and further administration of hypoosmotic fluids is more likely to increase the proportion of children who will have hyponatremia.
- Giving hypotonic fluids to these diseases put them at higher risk of developing hospital-acquired hyponatremia which poses greater risk for severe metabolic complications, and higher rates of morbidity and mortality.
- Detection and management of hyponatremia among the PICU admitted cases will reduce the hospital stay and indirectly the cost of treatment.

SUMMARY

SUMMARY

A total of 152 acutely ill children admitted in PICU were taken up for this study. Incidence of hyponatremia was 33.5% in our study. There is significant association between diagnosis and hyponatremia among subjects in our study.

Out of 51 cases of hyponatremia , 25.49 % had moderate hyponatremia and 13.73% had severe hyponatremia. The incidence of hyponatremia in bronchopneumonia was 41.3% followed by acute encephalitis 24%. The duration of hospital stay (days) was longer in severe hyponatremic cases than moderate hyponatremic cases.

From our study we infer that hyponatremia is common entity in varied clinical conditions in children. Hyponatremia is mild in variety though in acute neurological disease more severe variety have higher propensity to occur. Further studies are required to find out underlying mechanism of hyponatremia in various clinical states and to design rational fluid protocols for sick children. It is therefore essential to give the appropriate intravenous fluids, whenever deemed necessary in the management, especially to infectious cases, surgical abdomen, malignancy and neurologic diseases. Giving hypotonic fluids to these diseases put them at higher risk of developing hospital-acquired hyponatremia which poses greater risk for severe metabolic complications, and higher rates of morbidity and mortality.

Thus this study brings out the salient aspects of hyponatremia in sick children, focuses on the importance and need to recognise and correct hyponatremia in children for better outcome.

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ANNEXURES

ANNEXURE 1

PROFORMA FOR THE STUDY

TITLE : STUDY OF HYPONATREMIA IN CRITICALLY ILL CHILDREN ADMITTED TO PEDIATRIC INTENSIVE CARE UNIT .

Serial No.: Hospital number :

Name:

Age : Sex: Religion:

Address :

Informant:

Date of admission:

Date of discharge:

COMPLAINTS:

PAST HISTORY:

BIRTH HISTORY:

DEVELOPMENTAL HISTORY:

TREATMENT HISTORY:

IMMUNISATION HISTORY:

DIETARY HISTORY:

FAMILY HISTORY:

GENERAL EXAMINATION

Conscious: YES/NO

Temperature

Pulse rate Respiratory rate

BP

Pallor Icterus Cyanosis Clubbing Edema Lymphadenopathy

ANTHROPOMENTRY

WEIGHT:

HEIGHT:

HEAD CIRCUMFERENCE:

CNS-

Higher functions

1)Level of consciousness :Conscious/Drowsy-Arousable/Stupor/Coma

2)GCS

Cranial nerves

Motor system Right Left

Limb position

Bulk

Power (Proximal/distal)

Tone

REFLEXES

1)Superficial reflexes

2)Deep tendon reflexes

SENSORY EXAMINATION:

MENINGEAL SIGNS: Yes No

CEREBELLAR SIGNS : Yes No

Abdominal Examination:

Liver Spleen

Respiratory system

Air entry

Crepts

Rhonchi

CVS - S1,S2

PROVISIONAL DIAGNOSIS:

SERUM SODIUM LEVELS

1)AT THE TIME OF ADMISSION :

2)AT PERIODIC INTERVALS :

MORBIDITY :

a)Hospital stay > 5 days - YES / NO

b)Seizures YES / NO

c)Altered sensorium YES / NO

MORTALITY : YES NO

FINAL DIAGNOSIS :

ANNEXURE 2

PATIENT INFORMATION SHEET

I Dr Malikireddy Hima bindu, Post Graduate in Department of Paediatrics am conducting a study on "**STUDY OF HYPONATREMIA IN CRITICALLY ILL CHILDREN ADMITTED TO PEDIATRIC INTENSIVE CARE UNIT "** this is a prospective study to measure the serum sodium levels in children and to find association of hyponatremia with morbidity and mortality .There will not be any additional expenditure other than routine care incurred because of this study other than 2ml of venous blood collected. Personal information will not be revealed and the scientific data obtained through the study will be communicated to other Pediatricians.

(Principle investigator)

Date:

ANNEXURE 3

CONSENT FORM

I/We ,have been told to participate in the study **"STUDY OF HYPONATREMIA IN CRITICALLY ILL CHILDREN ADMITTED TO PEDIATRIC INTENSIVE CARE UNIT"** I/We have been told in my mother tongue and completely understood regarding the purpose of the study. I have been informed that no additional expenditure will be incurred for this study other than routine care. I have also been informed that any personal information of myself/ my child will not be revealed and I can withdraw from the study at any point of time without reason.

I,voluntarily give consent to participate in this study and allow to draw 2 ml of venous blood from my ward for the purpose of this study and to use the data or results that arise from this study for scientific purpose.

Signature Witness 1.

2.

KEY TO MASTER CHART

N – Normal

ABN – Abnormal

Y – Yes

Morbidity – Morbidity (ICU stay more than 5 days)

Sepsis with Acute GE – Sepsis with Acute gastroenteritis

AGE	SEX	DOA	DOD	HOSPITAL NO	PAST HISTORY	BIRTH HISTORY	DEVELOPMENTAL HISTORY	TREATMENT HISTORY	IMMUNISATION HISTORY	DIETARY HISTORY	FAMILY HISTORY	GENERAL EXAMINATION	ANTHROPOMETRY	VITALS	CNS	RS	CVS	P/A	PROVISIONAL DIAGNOSIS	SODIUM LEVELS AT TIME OF DISCHARGE	SODIUM LEVELS AT TIME OF DISCHARGE	MORTALITY	OUTCOME	MORBIDITY	FINAL DIAGNOSIS	SEIZURES	ALTERED SENSORIUM
10 years	M	2/28/2015	3/7/2015	191396	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	124	138	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
9M	M	3/1/2015	3/7/2015	190568	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEPSIS WITH ACUTE GE	131	139	NO	GOOD	Y	SEPSIS WITH ACUTE GE	NO	NO
13 YEARS	F	3/3/2015	3/15/2015	191020	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	123	135	NO	GOOD	Y	ACUTE ENCEPHALITIS	NO	Y
7 YEARS	M	4/12/2015	4/23/2015	194336	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	131	141	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
11 YEARS	M	4/15/2015	4/20/2015	194629	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	DENGUE FEVER WITH WARNING SIGNS	132	137	NO	GOOD	NO	DENGUE FEVER WITH WARNING SIGNS	NO	NO
2.5 MONTHS	F	4/20/2015	4/29/2015	194663	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEPSIS WITH ACUTE GE	128	141	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
7 MONTHS	M	6/1/2015	6/9/2015	194677	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEPSIS WITH ACUTE GE	123	135	NO	GOOD	Y	SEPSIS WITH ACUTE GE	NO	NO
4 YEARS	F	7/1/2015	7/11/2015	194635	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	SEVERE DENGUE	131	137	NO	GOOD	Y	SEVERE DENGUE	NO	NO
12 YEARS	F	7/12/2015	7/19/2015	155172	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	SEVERE DENGUE	134	141	NO	GOOD	NO	SEVERE DENGUE	NO	NO
1.5 YEARS	F	8/15/2015	8/18/2015	195995	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	DROWNING	129	135	NO	GOOD	NO	DROWNING	NO	NO
4 M	F	8/10/2015	8/18/2015	164781	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEPSIS WITH AGE	136	139	NO	GOOD	NO	SEPSIS WITH AGE	NO	NO
11M	M	6/13/2015	6/15/2015	165609	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	SEVERE DENGUE FEVER	139	137	Y	BAD	Y	SEVERE DENGUE FEVER	NO	NO
13 YEARS	M	6/16/2015	6/22/2015	203930	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEPSIS WITH ACUTE GE	128	138	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
12YEARS	F	6/20/2015	6/27/2015	204017	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	DENGUE FEVER WITH WARNING SIGNS	136	139	NO	GOOD	Y	DENGUE FEVER WITH WARNING SIGNS	NO	NO
13 YEARS	M	6/18/2015	6/25/2015	196139	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	DENGUE FEVER WITH WARNING SIGNS	133	143	NO	GOOD	NO	DENGUE FEVER WITH WARNING SIGNS	NO	NO
1.1 M	M	6/11/2015	6/16/2015	227091	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEPSIS WITH ACUTE GE	137	136	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
14 YEARS	F	6/10/2015	6/19/2015	227798	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	ACUTE ENCEPHALITIS	133	137	NO	GOOD	Y	ACUTE ENCEPHALITIS	NO	Y
9 YEARS	M	6/4/2015	6/10/2015	227396	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEPSIS WITH ACUTE GE	129	140	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
11 YEARS	F	6/3/2015	6/15/2015	228687	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	132	137	NO	GOOD	NO	ACUTE ENCEPHALITIS	Y	Y
2.3 YEARS	M	3/3/2015	3/11/2015	222089	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEPSIS WITH ACUTE GE	129	137	NO	GOOD	Y	SEPSIS WITH ACUTE GE	NO	NO
8 YEARS	F	7/3/2015	7/11/2015	228831	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BLUNT TRAUMA ABD	131	141	NO	GOOD	NO	BLUNT TRAUMA ABD	NO	NO
5YEARS	M	2/16/2015	2/22/2015	229118	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	DENGUE FEVER WITH WARNING SIGNS	130	135	NO	GOOD	NO	DENGUE FEVER WITH WARNING SIGNS	NO	NO
8 YEARS	M	2/16/2015	2/24/2015	229171	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	130	137	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
1.15 M	F	2/11/2015	2/19/2015	235054	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEPSIS WITH ACUTE GE	130	141	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
12 YEARS	F	2/5/2015	2/7/2015	235129	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	DROWNING	127	135	Y	GOOD	NO	DROWNING	NO	NO
10 YEARS	F	2/1/2015	2/15/2015	236010	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	132	139	NO	GOOD	Y	ACUTE ENCEPHALITIS	NO	Y
7 YEARS	F	1/21/2015	2/2/2015	236375	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	131	137	NO	GOOD	Y	ACUTE ENCEPHALITIS	NO	Y
3 MONTHS	M	1/12/2015	1/17/2015	236472	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEPSIS WITH ACUTE GE	128	138	NO	GOOD	Y	SEPSIS WITH ACUTE GE	NO	NO
1 YEAR	M	1/9/2015	1/15/2015	236904	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEPSIS WITH ACUTE GE	137	139	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
10 M	M	1/3/2015	1/9/2015	237270	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEPSIS WITH ACUTE GE	130	143	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
10 YEARS	M	1/3/2015	1/16/2015	237258	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	133	136	NO	GOOD	Y	ACUTE ENCEPHALITIS	NO	Y
7 YEARS	F	7/6/2015	7/11/2015	237311	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	DENGUE FEVER WITH WARNING SIGNS	131	137	NO	GOOD	NO	DENGUE FEVER WITH WARNING SIGNS	NO	NO
14 YEARS	M	7/5/2015	7/21/2015	237958	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	132	140	NO	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
10 YEARS	M	7/1/2015	7/13/2015	215090	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	SEVERE DENGUE	129	140	NO	GOOD	Y	SEVERE DENGUE	NO	NO
7 YEARS	F	7/13/2015	7/24/2015	240567	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	135	138	NO	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
4 YEARS	M	7/15/2015	7/24/2015	240570	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEPSIS WITH ACUTE GE	126	138	NO	GOOD	Y	SEPSIS WITH ACUTE GE	NO	NO
4 YEARS	F	7/17/2015	7/25/2015	238796	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEPSIS WITH ACUTE GE	128	139	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
7 YEARS	M	7/7/2015	7/15/2015	240998	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	129	138	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
9 YEARS	M	9/1/2015	9/6/2015	241055	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	136	136	NO	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
9 MONTHS	F	9/1/2015	9/7/2015	240177	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	139	135	NO	GOOD	NO	ACUTE ENCEPHALITIS	Y	Y
9 YEARS	M	9/2/2015	9/6/2015	241860	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	133	138	NO	BAD	Y	ACUTE ENCEPHALITIS	NO	Y
2 YEARS	M	9/5/2015	9/10/2015	242231	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	139	138	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
5 YEARS	M	9/3/2015	9/7/2015	242241	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	137	138	NO	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
3 MONTHS	M	10/5/2015	10/20/2015	242651	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	131	139	NO	GOOD	NO	ACUTE ENCEPHALITIS	Y	Y
10 YEARS	F	10/16/2015	10/29/2015	244234	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	139	140	NO	GOOD	Y	ACUTE ENCEPHALITIS	NO	Y
2 YEARS	F	9/26/2015	10/12/2015	243515	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	137	138	NO	GOOD	NO	ACUTE ENCEPHALITIS	Y	Y
14 YEARS	M	10/15/2015	10/27/2015	189220	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	135	137	Y	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
5 YEARS	F	10/22/2015	11/5/2015	192814	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	138	136	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
12 YEARS	F	10/21/2015	11/8/2015	191944	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	138	136	NO	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
14 YEARS	F	10/16/2015	11/2/2015	192878	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	127	139	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
4 YEARS	F	10/15/2015	11/2/2015	192087	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	136	139	NO	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
9 YEARS	M	10/15/2015	11/2/2015	194217	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	139	137	NO	GOOD	Y	ACUTE ENCEPHALITIS	NO	Y
4 YEARS	F	11/15/2015	12/1/2015	193988	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	137	136	NO	GOOD	NO	ACUTE ENCEPHALITIS	Y	Y
14 YEARS	M	12/15/2015	1/2/2016	193867	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	128	136	NO	GOOD	NO	ACUTE ENCEPHALITIS	Y	Y
5 YEARS	M	11/15/2015	11/29/2015	193871	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	139	138	NO	GOOD	Y	ACUTE ENCEPHALITIS	NO	Y
4 YEARSS	F	10/13/2015	10/30/2015	191396	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	138	136	NO	GOOD	NO	ACUTE ENCEPHALITIS	Y	Y
3 YEARS	M	10/4/2015	10/14/2015	190568	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	127	140	Y	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
7 YEARS	F	9/3/2015	9/7/2015	191020	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	138	138	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
2 YEARS	M	9/15/2015	9/21/2015	194336	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	132	136	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
14 YEARS	M	10/15/2015	10/29/2015	194629	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	131	136	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
3 YEARS	M	11/15/2015	12/2/2015	194663	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	135	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
2 MONTHS	M	10/9/2015	10/20/2015	194677	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	138	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
3 YEARS	F	11/9/2015	11/20/2015	194635	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	135	139	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
2.6 YEARS	F	12/10/2015	12/28/2015	155172	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	136	135	NO	GOOD	Y	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
4 YEARS	M	10/10/2015	10/14/2015	195995	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	136	137	NO	GOOD	Y	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
4 YEARS	F	6/10/2015	6/26/2015	164781	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	SEVERE DENGUE FEVER	129	136	NO	GOOD	NO	SEVERE DENGUE FEVER	NO	NO
7 MONTHS	M	5/5/2015	5/15/2015	165609	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEPSIS WITH ACUTE GE	128	138	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
11MONTHS	M	10/5/2015	10/15/2015	203930	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	140	140	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
13YEARS	F	5/5/2015	5/15/2015	204017	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEVERE DENGUE FEVER	135	138	NO	GOOD	NO	SEVERE DENGUE FEVER	NO	NO
8 YEARS	M	11/9/2015	11/26/2015	203534	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	SEVERE DENGUE FEVER	130	136	NO	GOOD	NO	SEVERE DENGUE FEVER	NO	NO
4 MONTH	M	9/14/2015	9/26/2015	202468	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	SEVERE DENGUE FEVER	138	135	NO	GOOD	Y	SEVERE DENGUE FEVER	NO	NO
8 YEARS	F	10/12/2015	10/20/2015	203344	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	SEVERE DENGUE FEVER	132	135	NO	GOOD	NO	SEVERE DENGUE FEVER	NO	NO
14 YEARS	M	10/10/2015	10/18/2015	203472	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEVERE DENGUE FEVER	131	137	NO	GOOD	Y	SEVERE DENGUE FEVER	NO	NO
10YEAR	M	10/16/2015	10/26/2015	202609	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEVERE DENGUE FEVER	136	138	NO	GOOD	NO	SEVERE DENGUE FEVER	NO	NO
9 YEARS	F	10/2/2015	14-Oct	202831	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEVERE DENGUE FEVER	136	135	NO	GOOD	Y	SEVERE DENGUE FEVER	NO	NO
2YRS	M	10/14/2015	10/24/2015	196139	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	140	137	NO	GOOD	Y	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
1.1 YEAR	F	12/1/2015	12/8/2015	227091	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	138	140	NO	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
1,6 YEARS	M	12/2/2015	12/5/2015	227798	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	133	134	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
4MONTH	M	12/2/2015	12/4/2015	227396	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	133	139	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
7 MONTHS	M	12/4/2015	12/8/2015	228687	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	141	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO

3 YRS	F	12/4/2015	12/10/2015	222089	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	139	132	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
14YRS	F	12/4/2015	12/8/2015	228831	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEVERE DENGUE FEVER	125	138	NO	GOOD	NO	SEVERE DENGUE FEVER	NO	NO
1REAR	M	12/5/2015	12/11/2015	229118	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	ACUTE ENCEPHALITIS	137	142	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
1 YEAR	M	12/5/2015	12/12/2015	229171	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	142	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
3MONT	M	12/7/2015	12/14/2015	229890	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH DISTRESS	137	139	NO	GOOD	NO	BRONCHOPNEUMONIA WITH DISTRESS	NO	NO
3 MNTHS	F	12/8/2015	12/16/2015	230450	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHITIS WITH RESPIRATORY DISTRESS	126	136	NO	GOOD	Y	BRONCHITIS WITH RESPIRATORY DISTRESS	NO	NO
1.5MONTHS	M	12/9/2015	12/16/2015	231039	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	140	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1YR	M	12/10/2015	12/17/2015	231575	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRES	137	138	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRES	NO	NO
4MONTH	F	12/10/2015	12/10/2015	231568	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	136	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
8MNTH	M	12/10/2015	12/17/2015	235169	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	131	138	NO	GOOD	NO	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
4MNTH	M	12/11/2015	12/18/2015	232068	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	139	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1MNTH	F	12/12/2015	12/12/2015	232241	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	139	135	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
4MONTH	F	12/12/2015	12/12/2015	232361	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	137	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1.5YEAR	M	12/12/2015	12/16/2015	232365	N	N	N	N	N	N	N	N	N	AB N	N	N	N	ABN	SEPSIS WITH ACUTE GE	126	140	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
5MONTH	F	12/12/2015	12/17/2015	232201	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	134	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
2MONTH	M	12/12/2015	12/17/2015	232461	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	122	139	NO	GOOD	NO	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
3YEARS MALE		12/13/2015	12/18/2015	232556	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	SEVERE DENGUE FEVER	129	141	NO	GOOD	NO	SEVERE DENGUE FEVER	NO	NO
3MNTH	M	12/13/2015	12/16/2015	232541	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	135	132	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1.5YEAR	M	12/14/2015	12/17/2015	233155	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	138	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
11Y	M	12/15/2015	12/15/2015	233217	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	128	142	NO	GOOD	NO	ACUTE ENCEPHALITIS	NO	Y
7M	M	12/15/2015	12/18/2015	233749	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	132	142	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
6M	M	12/15/2015	12/19/2015	233747	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	133	139	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
3M	M	12/16/2015	12/20/2015	233842	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	139	136	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1YR	M	12/18/2015	12/24/2015	235075	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	138	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1MNTH	M	178-12-15	12/19/2015	235054	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	139	139	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
5.5M	F	12/19/2015	12/20/2015	235129	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	135	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1.3Y	M	12/21/2015	12/22/2015	236010	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	131	141	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
2MONTH	M	12/22/2015	12/24/2015	236375	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	137	137	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1MNTH	F	12/23/2015	12/25/2015	236472	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	118	141	Y	BAD	NO	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
10M	F	12/23/2015	12/26/2015	236904	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	135	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
3M	M	12/24/2015	12/28/2015	237270	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	124	137	NO	GOOD	NO	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
2MONTH	F	12/24/2001	12/28/2015	237258	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA	138	141	NO	GOOD	Y	BRONCHOPNEUMONIA	NO	NO
1.5YEAR	F	12/24/2015	12/26/2015	237311	N	N	N	N	N	N	N	N	N	AB N	N	N	N	N	SEPSIS WITH ACUTE GE	118	135	NO	GOOD	NO	SEPSIS WITH ACUTE GE	NO	NO
1.6Y	F	12/26/2015	12/27/2015	237958	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	139	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
6Y	F	12/26/2015	12/28/2015	238005	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	128	137	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
11M	M	12/27/2015	12/29/2015	238020	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	128	138	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1MNTH	M	12/27/2015	12/31/2015	222075	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	130	139	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
3M	M	12/27/2015	12/29/2015	238079	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	139	143	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1.9M	F	12/27/2015	1/2/2016	238071	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	136	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
3M	M	12/29/2015	12/31/2015	238796	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	137	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
2MONTH	M	12/29/2015	12/29/2015	238982	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	140	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1.5YEAR	M	12/31/2015	1/2/2016	238997	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	140	NO	N	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
10M	M	12/30/2015	1/2/2016	239383	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	137	139	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
8MNTH	F	12/30/2015	1/2/2016	239406	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	130	139	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
2YRS	M	1/1/2016	1/4/2016	240022	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	137	137	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
5.6Y	M	1/1/2016	1/4/2016	240057	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	136	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
6Y	M	1/1/2016	1/2/2016	240065	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	137	136	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
9M	M	1/2/2016	1/5/2016	240446	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	138	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
2YRS	M	1/3/2016	1/4/2016	240556	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	139	136	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
2YRS	F	1/3/2016	1/5/2016	215090	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	135	140	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
8MNTH	F	1/3/2016	1/4/2016	240567	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	137	138	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
12YEARS	M	1/3/2016	1/5/2016	240570	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	DENGUE FEVER WITH WARNING SIGNS	131	136	NO	GOOD	NO	DENGUE FEVER WITH WARNING SIGNS	NO	NO
3M	M	1/3/2016	1/5/2016	238796	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	137	136	Y	BAD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO

9MONTHS	F	1/4/2016	1/16/2016	240998	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	143	135	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
1 MONTH	F	1/5/2016	1/7/2016	241055	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	138	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
7YEARS	M	1/6/2016	1/7/2016	240177	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	139	139	NO	BAD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
6 YEARS	F	1/7/2016	1/13/2016	241860	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	DENGUE FEVER WITH WARNING SIGNS	128	135	NO	GOOD	Y	DENGUE FEVER WITH WARNING SIGNS	NO	NO
4 YEARS	M	1/7/2016	1/8/2016	242231	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	137	137	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
3 MONTHS	F	1/7/2016	1/10/2016	242241	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	138	136	NO	GOOD	NO	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
7 MONTHS	M	1/8/2016	1/10/2016	242651	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	139	138	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
3YEARS MALE	F	1/10/2016	1/11/2016	244234	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	ABN	SEVERE DENGUE FEVER	138	136	NO	GOOD	NO	SEVERE DENGUE FEVER	NO	NO
1MNTH	M	1/11/2016	1/17/2016	243515	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	136	140	NO	GOOD	NO	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
9MONTHS	F	1/14/2016	1/16/2016	242568	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	135	138	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
9 YEARS	M	1/15/2016	1/20/2016	244963	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	138	136	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	Y
4MONTHS	M	1/16/2016	1/17/2016	244339	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	138	136	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
9MONTHS	M	1/19/2016		246210	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	138	135	NO	GOOD	Y	ACUTE ENCEPHALITIS	NO	Y
10 YEARS	F	1/13/2016	1/13/2016	243948	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	139	138	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
10 MONTHS	M	1/14/2016	1/16/2016	239383	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	140	139	Y	BAD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
3 YEARS	M	1/16/2016	1/17/2016	245378	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	123	135	NO	GOOD	Y	BRONCHOPNEUMONIA WITH RESPIRATORY DISTRESS	NO	NO
2 YEARS	M	1/15/2016	1/20/2016	245768	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	133	136	NO	GOOD	Y	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
3 YEARS	F	12/14/2015	12/21/2015	235637	N	N	N	N	N	N	N	N	N	AB N	N	ABN	N	N	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	136	137	NO	GOOD	Y	BRONCHIOLITIS WITH RESPIRATORY DISTRESS	NO	NO
4 YEARS	M	6/12/2015	6/20/2015	234543	N	N	N	N	N	N	N	N	N	AB N	ABN	N	N	N	ACUTE ENCEPHALITIS	138	138	NO	GOOD	Y	ACUTE ENCEPHALITIS	Y	NO