E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.01.27

Research Article

Clinical Profile, Etiology and Outcome of Acute Encephalopathy among Children at Pediatric Intensive Care Unit in a Tertiary Care Hospital

Dr. A. Monisha[1*], Dr Kovendan[2], Dr. J Hemachitra
[1*]Assistant Professor, Department of Pediatrics, Institute of Social Obstetrics and Kasturba Gandhi Hospital, Madras Medical College.
[2]Assistant professor, Department of Paediatrics, Thanjavur Medical College.
[3]Professor of Pediatrics, Department of Pediatrics, Institute of Child Health and Hospital for Children, Madras Medical College.
Email: [1*]monishashok3@gmail.com, [2]drkovendhan@gmail.com, [3]hemachitramkumar@gmail.com
Corresponding Author: Dr. A. Monisha
Received: 04.11.25, Revised: 10.12.25, Accepted: 24.01.26

INTRODUCTION

Encephalopathy is clinically defined as diffuse disorder of brain with at least two of the following [1] altered sensorium, altered cognition or personality, seizures. Acute encephalopathy is a pediatric emergency with high morbidity and mortality, however early identification and timely intervention can influence the outcome.

Acute encephalopathy in children occurs due to various etiology which can pose a great challenge in diagnosing promptly for timely intervention. There is a broad classification of etiology as traumatic and non-traumatic. The non-traumatic encephalopathy in children is caused by infections, auto immune diseases, seizures, metabolic disorders, hypertensive, toxins, hypoxic-ischemic insult, malignancy, hemorrhage and idiopathic.

This study was done to identify the clinco-etiological profile and the risk factors associated with mortality and morbidity of acute encephalopathy in children at a tertiary care level over a period of 1 year.

Materials and Methods:

Study setting: This was a cross sectional analytical study conducted in Pediatric Intensive Care unit in Chengalpattu Government Medical College.Ethical committee approval was obtained from the institution. (ECR/774/INST/TN/2016)

Duration of the Study: The study was conducted for a period of one year from 2017- 2018.

Study Population: All children of age 1 month to 12 years with acute encephalopathy admitted at the Pediatric intensive care unit.

Inclusion Criteria: All children of age 1 month to 12 years with clinical evidence of acute encephalopathy at admission were included in the study.

Exclusion criteria: Children with encephalopathy less than 6 hours. (In order to prevent over estimation of brain injury present at acute phases due to transient issues. [2])

METHODOLOGY

The sample size calculated with precision 10.5% and 95% confidence interval was 73, considering 10% non-responsive rate the sample size was increased to 80. According to inclusion criteria 100 cases were selected during the study period. All eligible children with acute encephalopathy who were consecutively admitted in our PICU were included in the study after obtaining an informed consent from the care giver. A detailed history and demographic data were recorded. An elaborate physical and neurological examination of the child which included the GCS was conducted at the time of admission and during the course of illness in the hospital. The etiology was classified according to the predefined definitions (Table 1). The children included in the study were treated according to protocol of the unit. These children were followed up till GCS become 15. The outcome was categorized as recovery to discharge or death. The survived children were assessed neurologically and also have undergone ophthalmic and auditory evaluation to detect any sequelae as per the existing protocol.

Table 1: Predefined Criteria for Etiological Classification

Sr. no.	Etiology	Criteria
1.	CNS infections a. Bacterial meningitis [3] b.tuberculous meningitis [4] c. Encephalitis [3] d. HSV encephalitis[5] e. JE encephalitis[6]	a. Acute febrile encephalopathy with identification of microorganism from the CSF culture or presence of 3 more of these criteria- • Polymorphonuclear leukocytosis ≥ 100 cells/mm3 • Glucose – 40 mg/dl or 50% of blood sugar • Elevated proteins > 40 mg/dl • Microorganism seen by gram staining b. family history of tuberculous, positive Mantoux test, CSF showing pleocytosis with leucocyte count 10- 250 cells/mm3, lymphocyte predominance, protein > 100-3000mg/dl, decrease glucose < 50 mg/dl and /or CT / MRI showing basilar enchancement or communicating hydrocephalus. c. acute febrile encephalopathy with CSF pleocytosis with lymphocyte predominance, elevated proteins (50 – 200 mg/dl) and absence of demonstrable bacteria on microscopy or staining where no other cause identifiable. d. focal seizures with CSF PCR positive and neuroimaging findings suggestive of HSV encephalitis. e. Acute onset of fever and change in mental status and behavior and/or new onset seizures with CSF PCR suggestive of JE
2.	Metabolic encephalopathy[7]	Metabolic derangement corresponding to the proportion of clinical encephalopathy.
3.	Toxic encephalopathy[8]	Encephalopathy with strong history of ingestion of toxins.
4.	Status epilepticus[9]	Continuous seizure activity or recurrent seizures without regaining consciousness in between lasting more than 5 min.
5.	Hypoxic ischemic encephalopathy [10]	Encephalopathy following hypoxic cerebral injury such as cardiopulmonary arrest, near drowning, shock or hanging.
6.	Traumatic	Nondegenerative or noncongenitally insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognition, physical and psychosocial functions, with an associated diminished or altered state of consciousness.
7.	Hypertensive encephalopathy [3]	Acute onset encephalopathy associated with blood pressure more than 95% percentile for age and sex with or without retinal changes.
8.	Vascular	Encephalopathy with evidence of bleed on neuroimaging.
9.	Idiopathic	Encephalopathy with absence of clinical clue and a negative investigation

Statistical Analysis

The study was approved by the institution ethical committee. Data was entered in excel spread sheet and analysed by SPSS software. The continuous data were expressed by means of mean ± standard deviation and ordinal variables by median and range. A p value ≤0.05 is considered to be statistically significant.

Results

Th study included 100 children with acute encephalopathy. The study population were categorized into 2 groups according to the outcome. Group 1 refers to survivors and group 2 expired. (Figure 1)

=

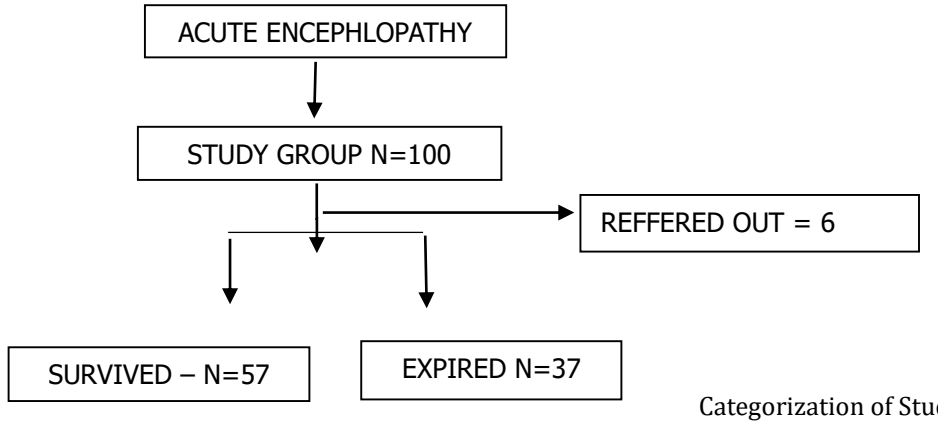

Figure 1: Categorization of Study Population

In this study 59 male child (59%) and 41 female child (41%) were studied. Male were affected more than female with the ratio 1.4:1. The study included children aged 1month – 12 years. Children below 1year were 15%, 1-3years were 46%, 3-6 years (26%) and 13% of the children were aged > 6 years (Table 2).

Table 2. Demographic Characteristics of Study Population

Sr. no.	Parameter	Male	Female
1.	Gender	59(59%)	41(41%)
2	Age	< 1 year 1-3 year 3-6 year >6 year	15(15%) 46(46%) 26(26%) 13(13%)

In our study, convulsions was the most common presenting symptoms (61%) followed by vomiting (52%) and fever (40%) (Figure 2).Headache (p =0.02) was significantly associated with mortality (Table.3). Pupillary reflex (p=0.001) was significantly associated with mortality among clinical signs (Table.4)

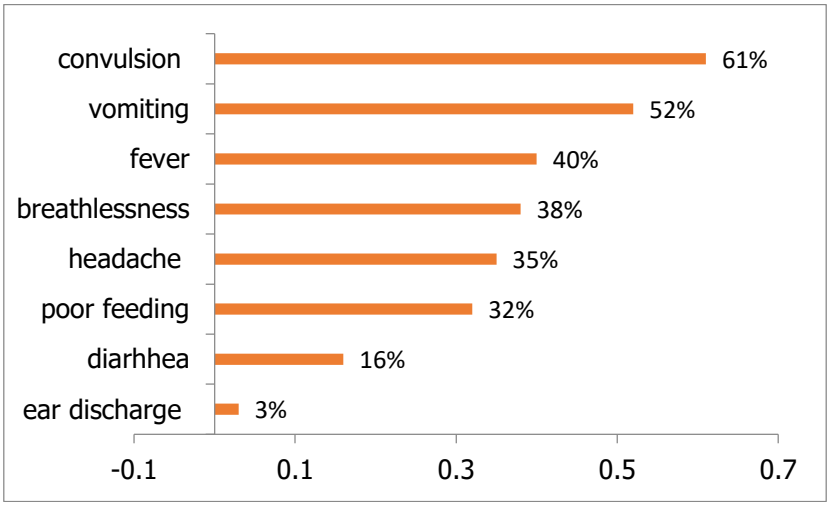

Figure 2. Clinical Presentation of Study Population

Table 3. Headache among Survived and Expired Children

Headache	Survived n=57(n %)	Expired n=37(n %)	Total	P value

Present	25(43.9%)	8(21.6%)	33	
Absent	32(56.1%)	29(78.4%)	61	0.002

Table 4. Pupillary reflex among survived and expired children

Pupillary reflex	Survived n=57(n %)	Expired n=37(n %)	Total	P value
Present	56(98.2%)	6(16.21%)	62	
Absent	1(1.7%)	32(8.64%)	34	0.001

All the 100 children underwent investigation relevant to their history and clinical examination to arrive at an etiology. Poor GCS (p=0.001), abnormal Capillary blood glucose (p=0.001), arterial blood gas pH (p=0.020), deranged coagulation profile (p=0.018) and low serum sodium (p=0.015) at arrival were significantly associated with mortality. Neuro-imaging was abnormal in 57% children. The complications that were encountered among the admitted children included hepatic failure, renal failure, VAP, UTI, ARDS, bed sores, shock and refractory seizures. Refractory seizures (p=0.001) and shock (p=0.001) were associated significantly with mortality among complications.

The etiological classification of the study population with acute encephalopathy showed CNS infection as the most common cause (40.4%) (Figure 3).

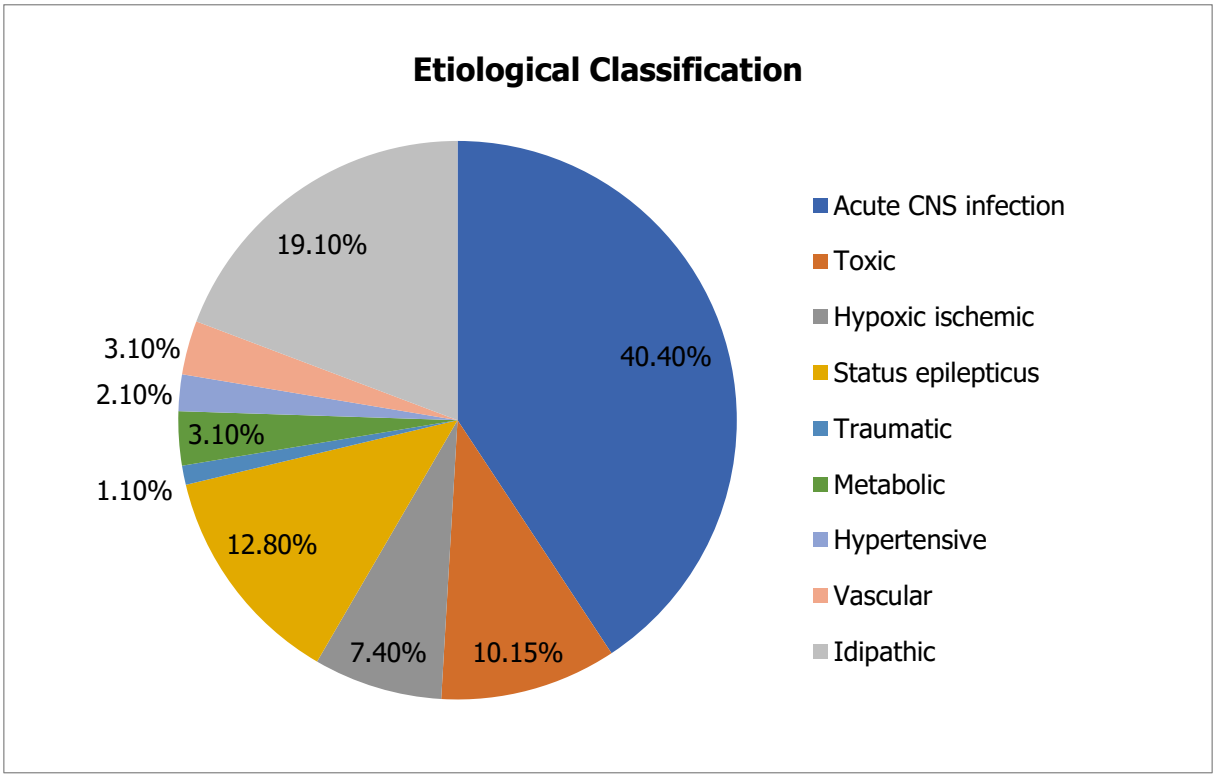

Figure 3. Etiological Profile Children With Acute Encephalopathy.

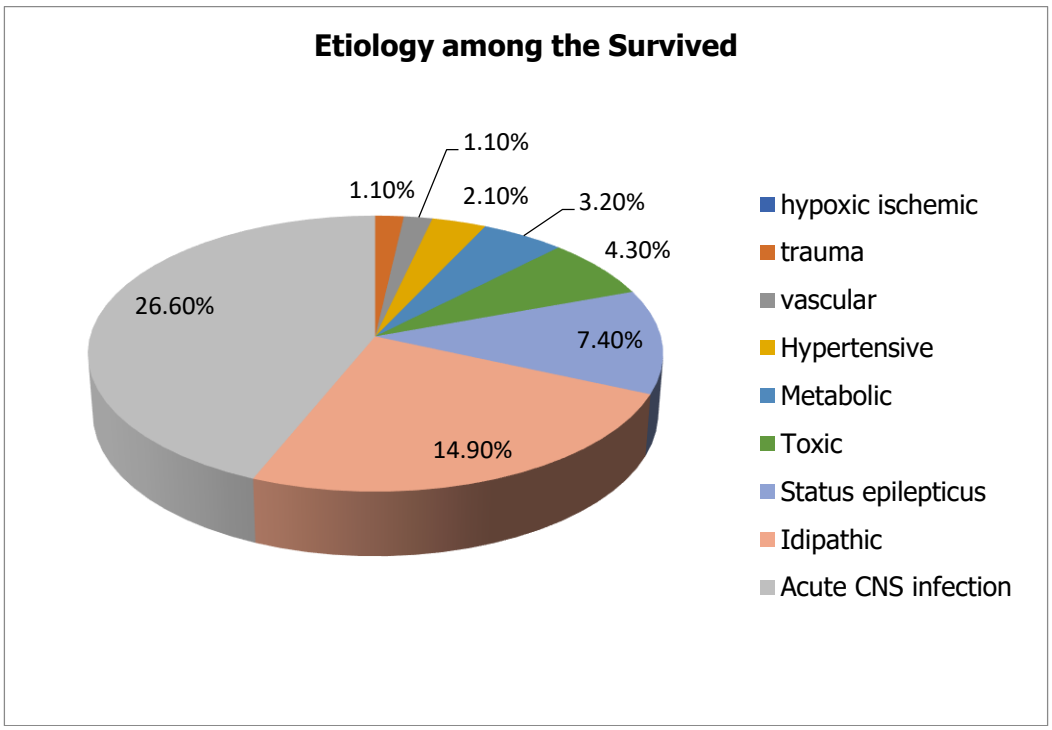

Figure 4. Etiological Profile of Children Who Survived

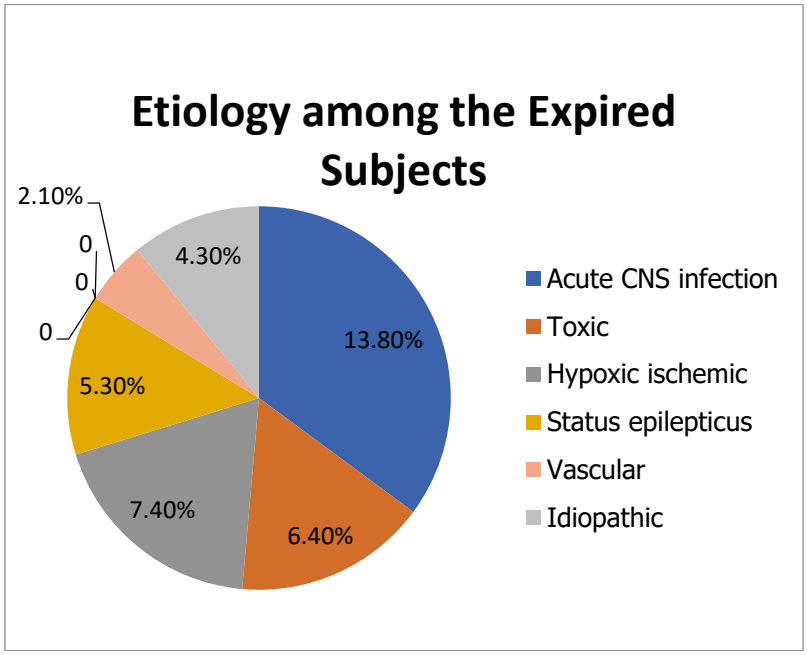

Figure 5. Etiological Profile of Children Who Expired

Among the 57 children who survived, sequelae were seen in 13 children in form of motor disability.

DISCUSSION

In this study the etiology, clinical profile and outcome of acute encephalopathy has been discussed. Most of the studies were conducted on acute febrile encephalopathy, studying the profile of infectious etiologies. Our study included a wide

range of etiologies and their clinical profile and outcome.

Among the 100 children recruited in the study, proportion of male was higher with 59% and females (41%). Arun bansal et al [3] also had a higher proportion of male (65%)[3]. The male: female ratio was 1.4:1 in this study as in Saba Ahmed et al [11]. The mortality rate was higher among the male (26.60%) compared to female (12.80%). However, gender was not significantly associated with outcome. This was also stated in Fariba Khodapanahandeh et al study [2].

The majority of children belonged to the age group 1- 3 years. Ali kajeh et al [12] and Fariba Khodapanahandeh et al [2] study also showed majority of children with acute encephalopathy were < 5years in their study whereassome studies [13][3] showed that infants were more vulnerable.

Among the clinical symptoms at the time of admission, convulsions (61%) and vomiting (52%) were the predominant among these children followed by breathlessness and headache. This was compatible with Fariba Khodapanahandeh et al [2] study.

On logistic regression analysis, low GCS at admisssion had independently significant association with mortality. Ali kajeh et al [12] and CM Bokade et al [14] also state in their study that low GCS was associated with 4.32 times higher risk of mortality than those with GCS > 8. This was also statistically proved significant.

Pupillary reflex was another clinical sign that was associated with outcome significantly. Pupillary reflex was absent in 86.4% of those expired. Arun bansal et al [3] study quotes that papillary signs were very good predictors of survival and neurological outcome.

With regard to the laboratory investigation, serum sodium levels and abnormal CBG were parameters that showed significant association with the outcome. Diana Grace et al [15]and Bhutia et al [16] in their study state the hyperglycemia is commonly seen in critically ill children and is associated with mortality. This study showed that hyponatremia was more associated with mortality. Zakia Al Lamki et al [17] also states that hyponatremia in pediatric inpatients was associated with 20 times higher mortality.

Abnormal coagulation profile and arterial pH abnormality were also found to have significant association with outcome of acute encephalopathy in this study

The etiology of acute encephalopathy was arrived at after detailed history, examination and laboratory investigations. In this study acute CNS infections (40.4%) were the most common cause of acute encephalopathy. The organism that was tested positive in our study population were streptococci pneumoniae in 2 children,tuberculosis in 1 child, Japanese encephalitis in 2 children and herpes simplex in 1 child. This was followed by idiopathic (19.1%) and status epilepticus (12.8%). Toxic encephalopathy accounted for 10.1% and hypoxic ischemic encephalopathy (7.44%). The other causes contributed smaller proportion. Several studies state that CNS infection was the commonest cause of acute encephalopathy in children. However, Fouad et al [18] has found in their study that metabolic was the most common cause of encephalopathy in children in their population and Ali kajeh et al [12] states toxic encephalopathy was most common among their study population. In a study done in Saudi Arabia, Ali AM [19] states that trauma was the most common cause of encephalopathy followed by infections.

The variables that were significantly associated with mortality were onset of illness, headache, low GCS, absent pupillary reflex, tachycardia acidosis, abnormal CBG, refractory seizures, abnormal coagulation profile, sodium abnormality and shock.

The outcome of the study was categorized as death and discharged. The mortality rate in this study was 39.3% and the survival rate was 60.6%. This was higher than the Arun Bansal et al [3] study done in India which showed a mortality of 35 %. Mortality was higher among the age group 1-3 years as in Arun Bansal et al [3] study.

Among the expired children the most common etiology was acute CNS infection (13.8%) followed by hypoxic ischemic encephalopathy (7.4%) . This was comparable to a large number of published studies which also showed higher mortality due to infectious etiology[21] . However all children diagnosed with hypoxic ischemic encephalopathy expired accounting to case fatality rate of 100 % and none of those children diagnosed with metabolic , traumatic and hypertensive encephalopathy had expired .

Limitation

Availability of wider panel of investigations and bedside EEG would have thrown more light on the diagnosis of the etiology of encephalopathy.

CONCLUSION

Among the study population with acute encephalopathy the clinical profile, etiology and outcome were analysed. Acute CNS infections were the most common etiology followed by idiopathic and status epilepticus. The study showed that acute CNS infections had higher mortality rate while hypoxic ischemic encephalopathy had higher case fatality rate. Headache, pupillary reflex, low GCS, abnormal arterial pH, low serum sodium, deranged CBG and coagulation profile, refractory seizures and shock were associated with mortality and it was proved statistically by logistic regression analysis. Hence knowledge about the etiological profile and outcome predictors of acute encephalopathy among children is required for rapid assessment and plan prompt treatment by directing limited resources for maximal benefit and reduce further neurological impairment.

REFERENCE

1. Altered states of consciousness.In:Gerald M. Fenichel. Clinical Pediatric Neurology 5thEd. Elsevier: Philadelphia; 2009:49.
2. Fariba Khodapanahandeh, MD, Najmeh Ghasemi Najarkalayee, MD . Etiology and Outcome of Non traumatic Coma in Children admitted to Pediatric Intensive Care Unit ,Iran. J PaediatrDec 2009; Vol 19 (No 4), Pp:393-398.
3. Arun Bansal, Sunit C. Singhi, Pratibha D. Singhi. Non Traumatic Coma. Indian Journal of Pediatrics.2005; 72(6):467-73.
4. Ahuja GK, Mohan KK, Prasad K, Behari M. diagnostic criteria for tuberculous meningitis and their validation. Tuber Lung Dis 1994;75;149-152.
5. P G E Kennedy, A chaudari. Herpes simplex encephalitis.J Neurol Neurosurg Psychiatry.10.1136/jnp.73.3.237.
6. T. Solomon, T.T. Thao, P. Lewthwaite. A cohort study to access the new WHO Japanese Encephalitis Surveillance standards," Bulletin of the World Health Organization ,2008; vol.86 no.3, pp-178-186.
7. G F Van Der Watt, FCpath (SA). Investigation of Metabolic encephalopathy.CME.2013vol.31. No.1.11-12.
8. Jacob Valk, M.S.Van der knap. Toxic Encephalopathy.AJNR1992;13:747-760.
9. Jagannathan H, Palanivelu S. Convulsive status epilepticus in children. Int J ContempPediatr 2017;4:1861-5.
10. Stephen Ashwal. Disorders of consciousness in children.In:Keneth F.Swaiman, Stephen Ashwal,Donna M.Ferriero, Nina .F.Schor, Richard S.Finkel, Andrea L.Gropman. Pediatric Neurology Principles and Practice 6th Ed. Elsevier: Philadelphia;2017:767.
11. Saba Ahmed, Kiran Ejaz, Muhammad Shahzad Shamim, Maimoona Azhar Salim, Muhammad Umer Rais Khan. Non-traumatic coma in paediatric patients: etiology and predictors of outcome :2011; JPMA 61:671.
12. Ali Khajeh, Ghasem Miri-Aliabad, Afshin Fayyazi.Non-traumatic Coma in Children in South-East of Iran: J ComprPed. 2015;6(1): e25049.
13. Wong CP, Forysyth RJ, Kelly TP. Incidence, Etiology and outcome of Non – Traumatic Coma: a population-based study. Arch Dis child 2001;84(3):193-9.
14. CM Bokade, RR Gulhane, AS Bagul, SB Thakre. Acute Febrile Encephalopathy in Children and Predictors of Mortality, Journal of Clinical and Diagnostic Research. 2014 Aug,Vol-8(8): PC09-PC11.
15. Grace RD, Poovazhagi V.Stress Hyperglycemia: clinical profile and outcome in critically ill children . Int.J contemp Pediatr 2017;4:2017-9.
16. Bhutia TD, Lodha R, Kabra SK.Abnormalities in Glucose Homeostasis in critically ill children. Pediatr Crit Care Med.2013;14:e16-25.
17. Zakia Al – Lamki, Maffooz A Farooqui , Saeed Ahmed. Incidence and outcome of severe hyponatremia in children and young adults. Sultan Qaboos University Medical Journal.2006;vol6:13-16.
18. Fouad H, Haron M, Halawa EF, Nada M. Nontraumatic coma in a tertiary pediatric emergency department in egypt: etiology and outcome. J Child Neurol. 2011;26(2):136–41.
19. Ali AM, Al-Abdulgader A, Kamal HM, Al-Wehedy A. Traumatic and non-traumatic

coma in children in the referral hospital, Al-Hasa, Saudi Arabia. East Mediterr Health J. 2007;13(3):608–14

20. Vijayakumar K, Knight R, Prabhakar P, Murphy PJ, Sharples PM. Neurological outcome in children with non-traumatic coma admitted to a regional pediatric intensive care unit. Arch Dis Child 2003; 88: A30-32..
21. Trubel HK, Norotny E, Lister G. Outcome of coma in children. Curr Opin Pediatr 2007; 15: 283-7.