NON-INDUCED TRAUMATIC COMA IN CHILDREN IN NORTHERN REGION OF YEMEN
Causes and Mortality

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ABSTRACT ● Objective : To describe the causes of non-induced traumatic coma (CNIT) in children in Yemen and to report the morbidity and mortality of such presentations. **Methods** : Cross-sectional study over a period of three years. Files of 150 children aged between one month and 14 years admitted with CNIT to the pediatric unit of Al-Salam Hospital, Saddah, Yemen, from July 2012 to July 2015, were reviewed. History, presenting symptoms, clinical and laboratory data were collected. Etiology of coma was determined on the basis of clinical history and relevant investigations. The outcome was recorded as death or neurological condition at discharge defined as normal, mild or sever disability. Chi-square test was used to test differences in categorical variables. **Results** : Out of 8967 children admitted during the study period, 150 presented with coma giving an incidence rate of 1.67%. There were 87 (58%) boys and 63 (42%) girls. The mean ± SD age of patients was 2.7 ± 2.35 years. Systemic presentations including nausea, vomiting, fever, lethargy and poor feeding were more prominent in children < 2 years. Etiology of coma in 60% cases was CNS infection (viral encephalitis : 30, bacterial meningitis : 24, cerebral malaria : 18) ; other causes were : toxic-metabolic conditions (16%), status epilepticus (10%), congenital brain malformation, hydrocephalus with V-P shunt (3.4%), and unknown (8%). Ninety-nine children survived, 39 were normal, 14 had mild disability, 32 had moderate disability and 14 were severely disabled and dependent. Survival was significantly better in patients with CNS infection (73.4%) as compared to those with toxic-metabolic causes (37%) and poisoning/intoxication (25%). **Conclusion** : CNS infections and toxic metabolic origins were the most common causes of coma non-induced trauma (CNIT) in childhood.

Keywords : non-induced traumatic coma ; children ; infections

RÉSUMÉ ● Objectif : Déterminer l'incidence, l'étiologie et les résultats du coma non traumatique chez les enfants observés à l'hôpital Al-Salam, Saddah, Yémen. **Méthodes** : Étude transversale sur une période de trois ans. Les dossiers de 150 enfants âgés de 1 mois à 14 ans admis avec coma non traumatique à l'unité pédiatrique de l'hôpital Al-Salam, Saddah, Yémen, de juillet 2012 à Juillet 2015, ont été examinés. L'historique, les symptômes présentés, les données cliniques et de laboratoire ont été recueillis. L'étiologie du coma a été déterminée sur base des antécédents cliniques et de recherches pertinentes. L'évolution du coma a été enregistrée comme mort ou un état neurologique défini comme un handicap normal, léger ou grave. Le χ² test a été utilisé pour tester les différences dans les variables catégorielles. **Résultats** : Sur 8967 enfants admis pendant la période d'étude, 150 ont présenté un coma, soit un taux d'incidence de 1,67%. Il y avait 87 (58%) garçons et 63 (42%) filles. L'âge moyen ± SD des patients était de 2,7 ± 2,35 ans. Nausées, vomissements, fièvre, léthargie et mauvaise alimentation étaient plus importants chez les enfants < 2 ans. L'étiologie du coma dans 60% des cas était des infections du SNC (encéphalite virale : 30, méningite bactérienne : 24, paludisme cérébral : 18); ou d'autres causes telles que troubles métaboliques toxiques (16%), état de mal épileptique (10%), malformation congénitale du cerveau; hydrocéphalie avec shunt ventriculo-péritonéal (3,4%), et inconnus (8%). Quatre-vingt-dix-neuf enfants ont survécu: 39 étaient normaux, 14 avaient un handicap léger, 32 un handicap modéré et 14 étaient sévèrement handicapés et dépendants. La survie était significativement meilleure chez les patients atteints d'une infection du SNC (73,4%) par rapport aux causes toxico-métaboliques (37%) et empoisonnement/intoxication (25%). **Conclusion** : Les infections du système nerveux central et le métabolisme toxique étaient les causes les plus fréquentes de coma non traumatique pendant l'enfance.

Mots-clés : coma non traumatique ; enfants ; infections

INTRODUCTION

Coma is a common childhood neurological emergency that can have a devastating outcome [1]. In children, it can be classified into traumatic (surgical emergency) and non-induced traumatic coma (CNIT) which is usually managed by physicians [2].

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Understanding the etiology and predictors of outcome of CNIT can influence the improved management strategies, especially in resource-poor countries where supportive diagnostic facilities are rarely available [3,4]. There is scant information on the etiology and outcome of CNIT to children in developing countries [2-6]. Neurologic outcome is often of foremost concern to parents and physicians, It may range from absence of impairment to severe disability or death [3, 4,7]. Etiology of coma and clinical status at the time

of presentation are likely predictors of outcome [6]. A better understanding of causes and outcome is essential to help improve the approach and to plan rational management of CNIT [2-6,8].

Literature on pediatric coma of non-induced trauma is rather inconclusive, as there are few systematic studies, and most of these are retrospective. There is no available information regarding CNIT in children in Yemen and very little information from some developing countries, such as India [3,8,9].

In that Indian prospective study, the authors had therefore examined the etiology, clinical signs and severity of non-traumatic coma in children, with a view to define predictors of outcome. On the basis of the Glasgow coma score (GCS), episodes in some studies were defined as less than 12 for more than six hours [10,11], but in most studies, many ill children are not fully conscious due to pathologic processes that may affect the parts of the central nervous system (CNS) that mediate consciousness [1], and some of these children make a full neurological recovery. However, depending on the underlying etiology, CNIT may cause considerable mortality and morbidity in pediatric age groups [8,9,12].

Taking into consideration the fact that acute CNIT is a common problem in pediatric practice accounting for 10-15% of all hospital admissions [8,13], it makes heavy demands on intensive care units [7].

In this retrospective cross-sectional study we reviewed the etiology, clinical signs and outcome of CNIT in a pediatric unit.

SUBJECTS AND METHODS

This is a cross-sectional study. Files of 150 children aged between 1 month and 14 years admitted with CNIT to the pediatric unit of Al-Salam Hospital, Saddah, Yemen, over a period of 3 years, from July 2012 to July 2015, were reviewed.

Coma was defined as significant depression of consciousness level as a GCS of less than 12 for more than 6 hours. In patients less than 5 years of age, modified GCS was used [10,11] as shown in Table I. GCS is checked routinely by our physicians and nursing staff in every child with altered level of consciousness.

Inclusion criteria: All children aged between 1 month and 14 years with non-induced traumatic coma i.e. with coma caused by medical illness admitted to the Pediatric Unit of Al-Salam Hospital, from July 2012 to July 2015, were included.

Exclusion criteria: All children who presented with coma as a final stage of malignant diseases and coma due to head trauma were excluded.

Data were collected from presenting symptoms, medical history, clinical and laboratory investigations (lumbar puncture, neuroimaging, metabolic work-up). Presenting manifestations were classified as CNS (central nervous system)-related, non CNS and organ specific.

Etiology was classified into infective and noninfective, epileptic, accidental and unknown causes.

Outcome was determined by patient's death or neurological condition at the time of discharge.

TABLE I	GLASGOW COMA SCALE (MODIFIED)		
SCORING	REACTING STIMULUS	REACTING STIMULUS	REACTING STIMULUS
Eye opening	**> 1 year**	**0-1 year**	
4	Opens spontaneously	Opens spontaneously	
3	Opens to a verbal command	Opens to a shout	
2	Opens in response to pain	Opens in response to pain	
1	No response	No response	
Best motor response	**> 5 years**	**2-5 years**	**0-23 months**
5	Oriented and able to converse	Uses appropriate words	Cries approprietly
4	Disoriented and able to converse	Uses inappropriate words	Cries
3	Uses inappropriate words	Cries and/or screams	Cries and/or screams inapproprietly
2	Makes incomprehensible sounds	Grunts	Grunts
1	No response	No response	No response
Best verbal response	**> 1 year**	**0-1 year**	
6	Obeys command	Spontaneous	
5	Localizes pain	Localizes pain	
4	Flexion withdrawal	Flexion withdrawal	
3	Flexion abnormal (decorticate)	Flexion abnormal (decorticate)	
2	Extension (decerebrate)	Extension (decerebrate)	
1	No response	No response	

Assessment of clinical neurological status was performed for all children. It included cranial and peripheral motor and sensory neurological examination, including cerebellar function.

Neurological outcome was divided into four groups :
- *Normal*: no change from premorbid neurological examination.
- *Mild disability*: (Grade 4) weakness or ataxia, isolated cranial nerve palsy, mild alteration of tone, power or deep tendon reflexes.
- *Moderate disability:* moderate weakness (Grade 3) or ataxia, multiple cranial nerve involvement.
- *Severe disability:* severe weakness (< Grade 3) or ataxia, tetraplegia, vegetative state.

In assessing the *effect of age on etiology and mortality*, children were divided into four age groups: • less than 1 month (neonate) • toddler (1-3 years) • pre-school (3-6 years) and • school age (6-14 years).

The *etiology of coma* was determined on the basis of history, clinical examination and relevant laboratory investigations. Investigations, such as lumbar puncture, CT scan and metabolic work-up, depended on the clinical presentation and were determined by the consultant in charge.

Bacterial meningitis was defined as acute febrile encephalopathy with microorganism identification from the cerebrospinal fluid (CSF) culture or latex agglutinins, or presence of 3 or more of the following abnormalities in CSF: (i) polymorphonuclear leukocytosis > 100 cells/mm; (ii) glucose ≈ 40 mg/dl or 50% of blood sugar; (iii) elevated proteins > 40 mg/dl; (iv) micro-organisms seen by Gram staining. Diagnosis of tuberculous meningitis was based on the criteria by Ahuja *et al*. [14]. Encephalitis was defined as acute febrile encephalopathy with CSF pleocytosis with lymphocyte predominance (> 5 cells/mm) and absence of bacteria on direct microscopy, culture or latex agglutination and where no other alternative diagnosis was identifiable [1,14,15].

When a metabolic disorder suitable with the clinical picture or toxic ingestion was confirmed, a label of toxic-metabolic coma was used. Coma following hypoxic cerebral injury such as after cardio-respiratory compromise or shock was considered to be hypoxic-ischemic.

Statistical analysis

Statistical analysis was conducted using Statistical Package for Social Sciences (SPSS) version 11. Descriptive statistics were expressed as mean and standard deviation. χ^2 test analysis was performed to test differences in proportions of categorical variables between two or more groups. The level of $p < 0.05$ was considered as being significant.

Ethical consideration

Ethical approval was obtained from hospital director and ethical committee of the Sana'a University Faculty

Age (months)	Sex				Total	
	Male		Female			
	n	(%)	n	(%)	n	(%)
< 1	12	54.5	10	45.4	22	14.7
12-36	20	52.64	18	47.36	38	25.3
36-72	30	58.82	21	41.17	51	34
> 72	25	64.10	14	35.89	39	26
Total	87	58	63	42	150	100

TABLEAU II
DISTRIBUTION OF ADMITTED COMATOSE PATIENTS
ACCORDING TO THEIR AGE GROUP

of Medicine & Health Sciences; confidentiality of gathered data and/or information was assured using passworded computer and anonymity was secured.

RESULTS

During the period under review, 8967 children were admitted to the children ward, of which 150 (1.67%) had non-accidental traumatic coma. Table II recapitulates the admission of comatose patients according to their age group • neonatal group: 22 (14.7%) • toddlers [1-3 years]: 38 (25.7%) • preschoolers [3-6 years]: 51 (34%) and • > 6 years: 39 (26%).

In this study, CNS infections accounted for 60% of the cases (Table III). Among the 24 children classified as toxic-metabolic, 6 had hepatic coma, 5 gave history of toxic ingestion and 6 had hypoxic ischemic encephalopathy (HIE) secondary to shock caused by sepsis (n = 3) or acute diarrhea (n = 3). Four had diabetic ketoacidosis (DKA).

Outcome

Fifty-one patients died and 99 survived. Of the 99 survivors, 7 left against medical advice before recovering from primary illness, 36 were normal (without any deficit), 13 had mild disability, 31 were moderately disabled and 14 were severely disabled and dependent (Table III).

Mortality rate was 50% (11 of 22) among neonates, 39.5% (15 of 38) among toddlers, 22% (11 of 51) among preschool children, and 20.5% (8 of 39) among children 6 to 14 years old. None of the neonates had a normal outcome, whereas 10% of toddlers, 20.5% of preschool and 16% of school age children had no deficits at discharge. Mortality was similar between the two sexes. Severe disability was higher in male children (12 of 40, 30%) compared to female (7 of 39, 18%) whereas intermediate level of disability was higher in female children.

Mortality with various CNS infections (Table III) was similar, but as a group CNS infections had significantly better survival rate compared to the toxic-metabolic group (RR = 0.5; 95% CI: 0.3 to 0.9; *p* = 0.04).

	Frequency (n = 150)		No. Died n = 51 (34%)		No. Survived n = 99 (66%)		Disability			
							Normal	Mild	Moderate	Severe
CNS Infections	90	(60%)	24	(26.6%)	66	(73.4%)				
Viral encephalitis	30	(20%)	8	(26.7%)	22	(73.3%)	4	4	11	3
Bacterial meningitis	24	(13.4%)	6	(25%)	18	(75%)	2	3	10	3
Cerebral malaria	18	(12%)	2	(11.2%)	16	(88.8%)	16	0	0	0
Sepsis	7	(4.7%)	2	(28.6%)	5	(71.4%)	3	1	1	0
Herpes encephalitis	4	(2.7%)	2	(50%)	2	(50%)	-	-	-	2
Rabies	4	(2.7%)	3	(75%)	1	(25%)	-	1	-	-
Tubercular meningitis	3	(2%)	1	(33.3%)	2	(66.7%)	-	1	1	-
Toxic-metabolic	24	(16%)	15	(63%)	9	(37%)	-	-	-	-
Hepatic coma	6	(4%)	4	(66.6%)	2	(33.4%)	2	-	-	-
Hypoxic encephalopathy (Secondary to shock)	6	(4%)	3	(50%)	3	(50%)	1	-	2	-
Poisoning/intoxication	5	(3.4%)	4	(75%)	1	(25%)	-	-	-	1
Diabetic ketoacidosis	4	(2.7%)	1	(25%)	3	(75%)	2	-	1	-
Snake bite	3	(2%)	3	(100%)	-		-	-	-	-
Post status epilepticus	15	(10%)	4	(26.7%)	11	(73.3%)	5	2	2	2
Congenital brain malformation	5	(3.4%)	2	(40%)	3	(60%)	-	-	1	2
Accident	4	(2.7%)	2	(50%)	2	(50%)	1	-	1	-
Unknown	12	(8%)	4	(33.4%)	8	(66.6%)	3	2	2	1

p value by chi-square test among the patients who died = 0.025
p value by Fisher's Exact test among the main etiological causes leading to disability among survivors = 0.100
p value by Fisher's Exact among the various causes of infections leading to disability among survivors = 0.511
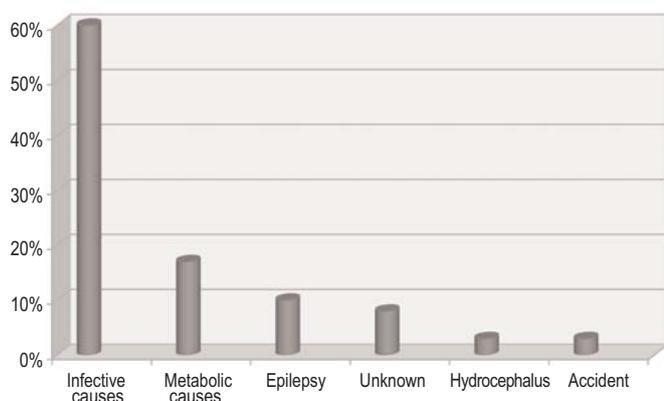

Figure 1. Frequency of causes at presentation of children with CNIT in study patients (n = 150)

A total of 150 patients were included in the study. There were 63 (42%) girls and 87 (58%) boys. The mean age of patients was 2.47 years (range: 1 month - 14 years ± 2.35). In the present study the modified GCS recorded at admission had significant association with outcome; mortality rates progressively increased with decreasing GCS.

Systemic presentations were particularly evident in children less than 3 years of age ($p = 0.009$) compared to other age groups. Infections (CNS infections with systematic infections), toxic-metabolic conditions and epilepsy were the commonest causes of non-accidental traumatic coma accounting for 129 (86%) cases.

The pattern of infection varies in different regions. In our study the most frequent infections were viral encephalitis 30 (20%), bacterial meningitis 24 (13.4%), cerebral malaria 18 (12%), and among noninfective causes: hepatic coma 6 (4%), hepatic encephalopathy 6 (4%) and poisoning/intoxication 5 (3.4%), followed by post status epilepticus 15 (10%) and unknown 12 (8%) (Figure 1).

Infection and congenital etiology were significantly more common in children < 3 years of age ($p = 0.002$ and $p = 0.009$ respectively), whereas accidents and intoxications occurred more prominently in those between 3 and 6 years of age ($p = 0.004$).

Epilepsy causing prolonged seizure and altered level of consciousness was more common in children > 6 years ($p = 0.008$).

DISCUSSION

In the present study, the incidence rate of non-induced traumatic coma (CNIT) among children was 1.67%. It occurred in 58% boys and 42% girls. The main causes were CNS infection in 60% of cases, metabolic and toxic causes in 16%, status epilepticus in 10%. The mortality rate was 34%.

Pediatric coma of non-accidental trauma is an important health problem making significant demands on

TABLEAU IV	COMPARISON OF CNIT ETIOLOGY AND OUTCOME IN DIFFERENT STUDIES				
	Sofiah *et al.* [17] MALAYSIA n = 116	Seshia *et al.* [22] CANADA n = 75	Ogunmekan *et al.* [6] NIGERIA n = 225	Vijaykumar *et al.* [3] UK n = 328	This study YEMEN n = 150
CNS infection	80 (69)	26 (34.7)	94 (41.8)	164 (50)	90 (60)
Toxic and metabolic	15 (13)	11 (14.7)	43 (19.1)	39 (12)	24 (16)
Status epilepticus	-	11 (14.7)	64 (28.5)	88 (27)	15 (10)
Unknown	11 (9.5)	3 (4)	-	24 (7)	12 (8)
Septicemia	-	3 (4)	9 (4)	-	7 (4.7)
Hypoxia	6 (5)	18 (24)	9 (4)	13 (4)	6 (4)
Mortality	35.7%	26.7%	26.7%	12%	34%
CNIT: non-induced traumatic coma					

intensive and high dependency care resources. It can result from a wide variety of primary etiologies, and as such constitutes a diagnostic challenge for the medical staff. A better understanding of the causes and outcome of this heterogeneous group of children should contribute to designing protocols for their investigation and management.

Consciousness requires normal functioning of both hemispheres as well as the ascending reticular activating system (ARAS) [1]. ARAS is a diffused group of neurons in the reticular formation of the brain extending from the lower medulla to the midbrain and diencephalon.

The clinical signs that reflect dysfunction of cerebral hemispheres and/or brain stem were noted. The GCS score reflects the integrity of cerebral functions. Breathing pattern and signs of abnormal tone of limbs and posturing indicate the extent and severity of cerebral hemisphere as well as brain stem damage.

In this study, it was observed that CNS infections were the commonest cause of CNIT (Table III). This is

TABLEAU V
CNIT CLINICAL FEATURES AMONG OUR CHILDREN

MANIFESTATION	NUMBER	PERCENTAGE
CNS related		
Seizure	52	*34.8%*
Headache	6	*4%*
Irritability	7	*4.6%*
Behavioral change	3	*2%*
Systemic		
Nausea and vomiting	25	*16.4%*
Fever	21	*14.2%*
Lethargy	10	*6.6%*
Poor feeding	4	*2.6%*
Poor weight gain	3	*2%*
Organ specific		
Respiratory tract infection	9	*6%*
Gastrointestinal infection	6	*4%*
Rash	4	*2.6%*
Total	**150**	*100%*

also supported by other studies in India and Nigeria [9, 6], However, the type of infection seems to vary in different regions (Table IV).

Cerebral malaria causing childhood coma (CNIT) was common in Africa [5,16], whereas dengue hemorrhagic fever was an important cause of coma in South-East Asia [17]. while septicemia was prominent in Saudi Arabia [18]. Both individual components of the Glasgow coma scale and neurological outcome were not assessed in the present study, while Nayana Prabha *et al*. [10] stated that Glasgow coma scale does not have a long-term predictive value in acute non-traumatic coma; rather, it is the etiologic agents that are predictive. But in another study in Canada, both depth of coma and etiology were not predictive of outcome [19]. In our study, the infection is the main cause in the children, in sharp contrast to adult hospital-based studies done in the UK and USA where degenerative and cerebrovascular pathologies predominate [8, 15]. In our study, mortality was lower than in adults, their mortality rates being 60% and neurological intact survival rates 10% [20]. It was also noted that *fever at admission* or its continuance 48 hours after hospitalization was not associated with higher risk of mortality or poor neurological outcome, as did *hypothermia* associated with poor prognosis. The hypothermic patients number in our study was too small for any meaningful conclusion, compared with other studies by Johnson and Seshia [19] in which all 13 hypothermic children died. In fact hypothermia, regardless of etiology causes, can diminished cerebral metabolism and very low temperature may result in an isoelectric electroencephalogram [21].

Among the non-infectious causes in our study, toxic-metabolic causes were comparable in frequency with other studies (Table IV).

In studies from U.K. [3] and Nigeria [6] status epilepticus was the second commonest cause of childhood coma (CNIT), respectively 27% and 28.5%, higher than our findings (10%).

In the course of this study, CNIT children presented with nausea and vomiting, fever, lethargy, poor feeding; poor weight gain was seen more in children < 3 years old

(Table V). This finding was compatible with studies done in Canada, India and Malaysia [8,9,17].

Preschool age was the most common age group that presented with CNIT (34%). This differs from the findings of Ali *et al*. [18] in Saudi Arabia who reported higher non-traumatic coma among infants age-group while traumatic coma was more common among the prechool age group.

Severe cases of malaria, like cerebral malaria, are uncommon in early infancy in this country, probably due to the maternally acquired partial protection in many hyper endemic areas [6,16,18] same to that in Yemen [23].

In the present study, the high mortality is likely due to late presentation, and could be a reflection of the declining investment in the health sector in the Yemeni government hospitals with a resultant inadequate man-power, diagnostic and resuscitation facilities. It is believed that prognosis in coma depends on its severity but there is rather inconclusive data on the use of GCS score and its predictive value in pediatric CNIT. We also noted that there is a relationship between the Glasgow coma scale score and mortality which also had been reported by studies in Nigeria and Saudi Arabia [4,5,18], While some had observed that it is the motor component that has a prognostic significance [10], others observed that it is the predictive value that determines the neurological outcome among survivors [18]. Moreover in this study, the incidence and the outcome of coma was not associated with gender differences which is similar to studies in Nigeria, Canada and Malaysia [6,8,17]. However, a study in Japan [13] had shown a greater mortality in male (42%) compared to female children (20%).

Mortality rate among children under 3 years was significantly higher in the present study similar to studies in Canada and India [8,9]. Post complications of rabies, poisoning, hepatic coma and accidents resulted in higher mortality rates compared to other etiology groups, while in a study in India by Arun Bansal *et al*. [9] infectious etiology resulted in highest death rate followed by toxic metabolic etiology. A study in Malaysia [17] also reported highest death rate in infectious group.

Among survivors of infectious diseases in our study about one third had normal outcome and the remainder left hospital with some degree of disability. This is compatible with Wong, Bansal and Sofiah studies [8,9,17]. Severe disability was also seen in about 23.33% of patients, in contrast to other published studies where 59% and 74% of the infectious group had a good outcome compared with 36% and 47% respectively in the metabolic group [13]. Some other studies in Tanzania and USA, revealed that 60% to 75% of the infectious group had a good outcome compared with 36% and 47% respectively in the metabolic group [24,25].

Due to the retrospective nature of the study, it was not possible to provide information on cognitive and adaptive outcome among survivors.

Our *overall mortality* of 34% was slightly higher as compared to other pediatric hospital-based series, 26.7% from Nigeria [6] and 26.7% in Canada [8]. The mortality rate among children under 3 years was significantly higher. It may be related to higher frequency of toxic-metabolic causes, congenital brain malformation (VP shunt to hydrocephalus) and higher mortality with CNS infections. Snake bite, poisoning, rabies and hepatic coma causes had the worst outcome in comparison with cerebral malaria and bacterial meningitis survival rates (Table III).

CONCLUSION

Non-traumatic comas in children in Al Salam Hospital, Saddah, Yemen, were mainly due to infections (viral encephalitis, bacterial meningitis, cerebral malaria), followed by toxic metabolic causes and epilepsy, the most predominant causes. Mortalities among these cases were high, and call for increased efforts to control infection so as to reduce the incidence of non-traumatic coma in this study site.

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