



Non-Traumatic Coma in Children- A Review

**Manda Anusha^{a*}, Avirendla Sony^a,
Syeda Mohammadi Begum^a, Velupula Neelima^a
and Amatul Haseeb Maseera^a**

^a Department of Pharmacy Practice, St. Pauls College of Pharmacy, Hyderabad, Telangana, India.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/AJPR/2023/v13i2266

Open Peer Review History:

This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: <https://www.sdiarticle5.com/review-history/102616>

Review Article

Received: 25/05/2023

Accepted: 28/07/2023

Published: 03/08/2023

ABSTRACT

Coma is defined as the unintentional failure of the patient to open their eyes spontaneously or in response to noise and inability to open for commands or localised painful stimuli with or without the ability to express comprehensible words or language response, whereas the Non traumatic coma is without any injury to the brain, and the Glasgow coma scale below 12.

The Non traumatic coma causes due to the infections(tubercular meningitis, bacterial meningitis, encephalitis) and other metabolic conditions ,status epilepticus intracranial bleed, miscellaneous, hypoxic ischemic insults, Electrical shock, shunt dysfunction, Inborn error of metabolism.

The monitoring parameters are Heart rate, Respiratory rate and pattern, Blood pressure, Temperature, coma severity using GCS, pupillary size, extra ocular movements, corneal reflex, posture, and motor pattern. The main symptoms are ataxia or weakness, isolated cranial nerve palsy, mild alteration of tone, poor pulse volume, apnea, abnormal pupillary size and reaction, dizziness, Headache, abnormal extra ocular movements absent corneal reflex.

Management of non-traumatic coma patients at hospital includes Monitoring of Intracranial pressure, Maintenance of an adequate cerebral perfusion pressure, Management of persistent intracranial hypertension and Monitoring of Electroencephalographic Seizure activity.

*Corresponding author: Email: mandaanusha961@gmail.com;

Infections are the most common cause for the non-traumatic coma in several cases. The pattern of infectious caused are CNS infections > metabolic toxic exposure > encephalitis > Intoxication > hypoxic ischemic insults > Intracranial haemorrhages>miscellaneous>unknown cases. 40% of children are prone to Non traumatic coma per 10000 general population per year.

Keywords: Non traumatic coma; etiology; symptoms; children.

1. INTRODUCTION

“Non-traumatic coma (NTC) is a common cause of morbidity and mortality in children. Episodes were defined on the basis of a Glasgow Coma Score (GCS) of less than 12 for more than six hours” [1,2]. “Many acutely ill children are not fully conscious because pathologic processes may affect the parts of the central nervous system that mediate consciousness; alteration in the state of consciousness is a common feature of many different conditions” [1]. “An acquired brain injury (ABI) is damage to the brain occurring after birth caused either by trauma (traumatic brain injury; TBI) or through a medical problem or disease process such as neoplasm, vascular causes, inflammation or metabolic toxicity (non-traumatic brain injury; NTBI)” [2,3]. “Coma is defined as the unintentional failure of the patient to open their eyes spontaneously or in response to noise and inability to open for commands or localize painful stimulus with or without the ability to express comprehensible words or language response” [4].

2. ETIOLOGY

“Etiologically it can be divided into three broad categories: those without focal neurologic signs (e.g., metabolic encephalopathies) meningitis syndromes, characterized by fever or stiff neck and an excess of cells in the spinal fluid (e.g., bacterial meningitis, subarachnoid haemorrhage) and conditions associated with prominent focal signs (e.g., stroke, cerebral haemorrhage). In most instances non traumatic coma is part of an obvious medical problem such as drug ingestion, hypoxia, stroke, trauma, or liver or kidney failure. Conditions that cause sudden non traumatic include drug ingestion, cerebral haemorrhage, trauma, cardiac arrest, epilepsy or basilar artery embolism” [5].

3. SYMPTOMS

1. “**Normal:**Normal or no change from premonitory neurological examination.

2. **Mild:**Disability: Mild (grade 4) weakness or ataxia, isolated cranial nerve palsy, mild alteration of tone, power or deep tendon reflexes.
3. **Moderate disability:** Moderate weakness (grade 3) or ataxia, multiple cranial nerve involvement.
4. **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 three age groups: less than 2 years; 2-6years; 6-14 years of age” [6]. “Headache, dizziness, palpation, poor pulse volume, abnormal respiratory pattern, apnea, abnormal pupillary size and reaction, abnormal extra ocular movements, absent corneal reflex, abnormal motor muscle tone are the symptoms observed” [7].

4. MONITORING PARAMETERS

“To determine the clinical signs of the non-traumatic coma the vitals that are to be monitored are temperature, pulse rate, blood pressure, respiratory rate, GCS, pupillary reflex, tone and fundus to corneal reflex, extra ocular movements, motor pattern, seizure type, and fundus picture” [8].

5. MANAGEMENT

5.1 Monitoring of Intracranial Pressure

After admission to the hospital, the first thing that is to be monitored is blood pressure. Intra cranial pressure (ICP) monitoring should be considered, as it is very useful in irreversible brain stem damage, bleeding diathesis and should be carefully monitored. “Patients with liver diseases, associated with prolonged prothrombin time, platelet count below 50×10^9 is an absolute contraindication with extradural ICP monitoring. Bleeding may be more in patients with meningitis, in whom the vessels may be monitored, for ICP monitoring, the widely used method is Camino system in non-traumatic coma” [9].

5.2 Maintenance of an Adequate Cerebral Perfusion Pressure

“There is Evidence that in non-traumatic coma, outcome is related to minimum CPP than to maximum ICP” [10]. “CPP depends on the age of the child. In children outcome is poor if the CPP is consistently lower than 65-70mmhg” [11]. “ICP spikes can be prevented by maintaining systemic circulation, with plasma if the circulation is under filled and otherwise with Inotropic support” [12].

5.3 Management of Persistent Intracranial Hypertension

“Firstly, it is possible to reduce cerebral blood flow, below the ischemic threshold” [13]. “The current recommendation, is to ventilate to normocapnia, the patients can then by hyperventilated or bagged during ICP spikes. Fluid management is very difficult and harmful in patients with subarachnoid haemorrhages and meningitis” [14-16]. “To manage patients properly, it is essential to monitor BP, cerebral venous pressure, urine output, weight, core and peripheral temperature, plasma and electrolytes and osmolality at least six hourly, mannitol may reduce spikes of ICP very rapidly, there is vogue that anaesthetic agents reduce ICP. Barbiturates and sedatives, there is no evidence on decreasing ICP level. Reducing the body temperature by 1 degree centigrade can reduce cerebral metabolic rate, evidence for an addition beneficial effect on Ischemic brain tissue, one advantage that hypothermia may easily reversible. CSF drainage in ICP persistence, an intraventricular cannula may be placed, it is difficult, if the ventricles cannot be seen on CT scan. It is beneficial to treat the patient by knowing the cause of the non-traumatic coma” [17].

5.4 Monitoring of Electroencephalographic Seizure Activity

“Once an unconscious patient is ventilated, it is usually impossible to detect clinical evidence of seizures, but a substantial proportion have ongoing status epilepticus” [18]. “A 1–4 channel cerebral function monitor can be used to detect the majority of electrical discharges, but requires considerable neurophysiological back up, including regular full 16-channel EEG” [19-21]. “So that the data is interpreted correctly and focal discharges are not missed. The pathophysiology of epilepsy occurring in the context of coma is

poorly understood. There are a number of reasons for seizures to occur, for example, fever, ischemia in the anterior and posterior cortical border zones, release of excitotoxic neurotransmitters after ischemia, particularly in the hippocampal region of the temporal lobe, direct cortical invasion and thrombophlebitis in meningitis, small vessel vasculitis in conditions such as haemolytic uremic syndrome, vasospasm, steno occlusion of the basal cerebral vessels in meningitis, and perhaps venous thrombosis in conditions such as cerebral malaria where the venous system is the site of parasitic invasion. One benefit of neurophysiological monitoring is to give early warning of potentially treatable complications, presenting either as deterioration in the background pattern or as seizure discharges. It has been argued that prolonged seizures and status epilepticus in unconscious patients simply reflect the degree of brain damage already sustained, but there is evidence that poor outcome is associated with the presence of encephalopathies” [22-27]. In a study using CFAM monitoring, outcome (but not mortality) was related to number and duration of electroencephalographic seizures and to the duration of the longest seizure [27].

6. DISCUSSION

We took few of the research articles from that we would like to see what is the outcome status of Non traumatic coma in children. In this research article there attempt is to guide the worried in casualty or on the ward faced with a child in non-traumatic coma who may need intensive care. In 117 children, 72 boys and 44girls in that 80 cases were due to infection, 15 cases are due to toxic metabolic causes, 6 cases are due to hypoxic ischemic insults, 4 cases due to intracranial haemorrhages, 9 cases due to miscellaneous causes and 2 cases are unknown. In that children 39 patients died, 32 patients developed permanent neurological defects and 38 were discharged well. So infections account for 74% of non-traumatic coma [28].

“Etiology and clinical profile of non traumatic coma in children and to determine the clinical signs and predictive outcomes, for the population was 100 between 2 months and 6 years of age for this clinical study the data collected are Temp, PR,HR,BP,GCS,RR, respiratory pattern, pupillary and corneal reflex, extra ocular movements, motor patterns, seizure types, fundus picture recorded during the hospital stay,

the outcome are the survived (mild, moderate ,severe, disability) and died, the outcome are 60% patients due to infection (tubercular meningitis-19, encephalitis-18,bacterial meningitis-16 and others are 7).metabolic conditions-19%,status epilepticus 10%, intracranial bleed-7%, miscellaneous-7%.Out of 100 sample size 65 children are normal, 14 mild,21 moderate and 14 patients are severe disability. Survival was significantly better in patients with CNS infection compared to metabolic causes” [29].

The children with acute non traumatic coma, the sample size was 270 and age of children between 2months and 6 years, the data are MGCS and brain stem reflexes assed 6hrs interval on time of admission, the analysis are lowest MGCS and worst brain stem reflex, The method used for this was spearman rank correlation coefficient=0.577, they found the adverse effects, are improper ocular response, motor response, verbal response, brain stem response [30].

The non traumatic coma in children, the prospective study the sample size was 100 in mostly in paediatrics between Months to years. In this 50% patients died, the causes are due to metabolic (33%) CNS infection (28%) and intra cranial haemorrhage's (13%) The outcome was the Metabolic causes are the most common etiology in paediatrics non traumatic coma [31].

A prospective cross sectional study over a year of 5 years, the sample size was 150 children from 1 month to 14 years of age, the data collected are persisting symptoms and clinical laboratory Data, mostly under 2 years of age children are systemic presentation, nausea, vomiting, poor feeding, and lethargy. Out of 150 children 63 boys and 87 girls, and 49 patients are due to infections (meningitis, Encephalitis, respiratory system) 44 patients are due to epilepticus, metabolic 11 patients, that is (diabetic ketoacidosis, inborn error of metabolism) 10 patients due to Intoxication, (electrical shock, suffocation, shunt, dysfunction) 11 patients are unknown cause, 25 children are died, and 16 are severe disabled here there is one evidence that accidents and infections had higher mortality [32]. The coma defined as GCs below 12 for more than 6 hours, 14% of patients cause was unknown and the remain mostly due to infections [6].

The sample size are 61 from the age 6 months to 12 years here 25 cases are due to infections, 16

toxic metabolic, 10 status epilepticus, 10 encephalitis, mostly mortality is due to toxic metabolic coma, than the infections. Simple clinical signs are responsible for non traumatic coma [33]. The data collected from the patients are temperature, pulse rate, blood pressure, respiratory rate, Glasgow coma scale, pupillary reflex. The morbidity and mortality are due to 58% due to infection, 50% status encephalitis, 10%metabolic, 14%intoxication, Intracranial bleed 2%, and Glassgow coma scale below 8 [34].

7. PREVENTION AND SUMMARY

“Many infective causes of NTC are preventable with vaccines, and controlled or prevented by eliminating malnutrition/anemia. Other essential public health measures include safe water supply, sewage disposal, mosquito control, education on personal hygiene, and timely access to child health care, both preventive and illness related. Education may also minimize poisoning and NAHI, as it has the incidence of pediatric head injuries in Western countries. Other steps include being aware of and identifying populations at high risk for IEM, strokes, and certain encephalopathies” [34].

8. CONCLUSION

Infections are most common cause for the non-traumatic coma in several case the CNS infections > Toxic metabolic > Status Epilepticus > Encephalitis > Intoxication > Hypoxia Ischemic insults > Intracranial haemorrhages > Miscellaneous > unknown cause.

Infections were the leading cause of non-traumatic coma as well as the leading cause of mortality in the study. Hypothermia, hypotension, altered breathing pattern, Non-reactive pupils, low GCS, hypotonic, hyporeflexic and low muscle power score were significantly associated with mortality in children presenting with non-traumatic coma.

CONSENT AND ETHICAL APPROVAL

It is not applicable

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Frigerio S, Molteni E, Colombo K, Pastore V, Fedeli C, Galbiati S, Strazzer S. Neuropsychological assessment through Coma Recovery Scale-Revised and Coma/Near Coma Scale in a sample of pediatric patients with disorder of consciousness. *Journal of Neurology*. 2023;270(2):1019-29.
2. Wong CP, Forsyth RJ, Kelly TP, Eyre JA. Incidence, aetiology, and outcome of non-traumatic coma: A population based study. *Archives of disease in childhood*. 2001;84(3):193-9.
3. Canadian Institute for Health Information. Inpatient rehabilitation in Canada, 2003–2004. Ottawa: CIHI; 2005. [cited 2008 Jan 15].
4. Bajaj J, Yadav Y, Sharma D. Modifications of glasgow coma scale—A systematic review. *Indian Journal of Surgery*. 2023;27:1-2.
5. Ahmad I et al, Non traumatic coma in children, *International Journal of Contemporary Pediatrics*. 2015;2(2).
6. Khoda PF, Ghasemi NN. Etiology and outcome of non-traumatic coma in children admitted to pediatric intensive care unit. 2009;393-398.
7. Arun Bansal, Sunit C, Singh S. Ramesh, Non traumatic coma, *The Indian Journal of Paediatrics*. 2005;72:467-473.
8. Schoeggl A, Reddy M, Bavinzski G. A lateral mass fracture of C1 associated with left vertebral artery and mid-basilar artery occlusion. *Journal of Neurotrauma*. 2001;18(7):737-41.
9. Kirkham FJ. Non-traumatic coma in children. *Archives of disease in childhood*. 2001;85(4):303-12.
10. Fouad H, Haron M, Halawa EF, Nada M. Nontraumatic coma in a tertiary pediatric emergency department in Egypt: etiology and outcome. *Journal of child neurology*. 2011;26(2):136-41.
11. Nag DS, Sahu S, Swain A, Kant S. Intracranial pressure monitoring: Gold standard and recent innovations. *World Journal of Clinical Cases*. 2019;7(13):1535.
12. Chambers IR, Kirkham FJ. What is the optimal cerebral perfusion pressure in children suffering from traumatic coma?. *Neurosurgical Focus*. 2003;15(6):1-8.
13. Kumar R, Singhi S, Singhi P, Jayashree M, Bansal A, Bhatti A. Randomized controlled trial comparing cerebral perfusion pressure–Targeted therapy versus intracranial pressure–targeted therapy for raised intracranial pressure due to acute CNS infections in children. *Critical care medicine*. 2014;42(8):1775-87.
14. Maconochie IK, Bhaumik S. Fluid therapy for acute bacterial meningitis. *Cochrane Database of Systematic Reviews*. 2014(5).
15. Yu PL, Jin LM, Seaman H, Yang YJ, Tong HX. Fluid therapy of acute brain edema in children. *Pediatric Neurology*. 2000;22(4):298-301.
16. Alamarat Z, Hasbun R. Management of acute bacterial meningitis in children. *Infection and drug resistance*. 2020:4077-89.
17. Kochanek PM, Tasker RC, Carney N, Totten AM, Adelson PD, Selden NR, Davis-O'Reilly C, Hart EL, Bell MJ, Bratton SL, Grant GA. Guidelines for the management of pediatric severe traumatic brain injury: Update of the brain trauma foundation guidelines. *Pediatric Critical Care Medicine*. 2019;20(3S):S1-82.
18. Towne AR, Waterhouse EJ, Boggs JG, Garnett LK, Brown AJ, Smith JR, DeLorenzo RJ. Prevalence of nonconvulsive status epilepticus in comatose patients. *Neurology*. 2000;54(2):340.
19. Kirkham FJ, Wade AM, McElduff F, Boyd SG, Tasker RC, Edwards M, Neville BG, Peshu N, Newton CR. Seizures in 204 comatose children: Incidence and outcome. *Intensive Care Medicine*. 2012;38:853-62.
20. Kirkham FJ, Ashwal S. Coma and brain death. *Handbook of Clinical Neurology*. 2013;111:43-61
21. Topjian AA, De Caen A, Wainwright MS, Abella BS, Abend NS, Atkins DL, Bembea MM, Fink EL, Guerguerian AM, Haskell SE, Kilgannon JH. Pediatric post–cardiac arrest care: A scientific statement from the American Heart Association. *Circulation*. 2019;140(6):e194-233.
22. Ramantani G, Holthausen H. Epilepsy after cerebral infection: Review of the literature and the potential for surgery. *Epileptic Disorders*. 2017;19(2):117-36.
23. Bartoloni A, Zammarchi L. Clinical aspects of uncomplicated and severe malaria. *Mediterranean Journal of Hematology and Infectious Diseases*. 2012;4(1).
24. Gladstone M. A review of the incidence and prevalence, types and aetiology of

- childhood cerebral palsy in resource-poor settings. *Annals of tropical paediatrics*. 2010;30(3):181-96.
25. Paganini H, Gonzalez F, Santander C, Casimir L, Berberian G, Rosanova MT. Tuberculous meningitis in children: clinical features and outcome in 40 cases. *Scandinavian Journal of Infectious Diseases*. 2000;32(1):41-5.
 26. Lan SY, Lin JJ, Hsia SH, Wang HS, Chiu CH, Lin KL, Cheese study group. Analysis of fulminant cerebral edema in acute pediatric encephalitis. *Pediatrics & Neonatology*. 2016;57(5):402-7.
 27. Kirkham FJ, Wairui C, Newton CR. Clinical and electroencephalographic seizures in coma: Relationship to outcome. *Arch Dis Child*. 2000;82(suppl 1):A59.
 28. Fouad H, Haron M, Halawa EF, Nada M. Nontraumatic coma in a tertiary pediatric emergency department in Egypt: etiology and outcome. *Journal of child neurology*. 2011;26(2):136-41.
 29. Arun Bansal, Sunit C Singhi, Pratibha D Singhi, Khandelwal N, Ramesh S, Non traumatic coma. *The Indian Journal of Paediatrics*. 2005;72,467-473.
 30. Prabha PN, Nalini P, Serane VT. Role of Glasgow Coma Scale in pediatric nontraumatic coma. *Indian Pediatrics*. 2003;40(7):620-5.
 31. Fouad H, Haron M, Halawa EF, Nada M. Nontraumatic coma in a tertiary pediatric emergency department in Egypt: Etiology and outcome. *Journal of Child Neurology*. 2011;26(2):136-41.
 32. Ikhlas ahmed,kaiser. Ahmed,Imran ahmed Gatto Mohammad Younus mir,Baba, Non traumatic coma in children, *International Journal of Paediatrics*. 2015;(2) 77-84.
 33. Suganthi V, Kumar MS, Kumar BR. Non-traumatic coma in children: Clinical profile and outcome. *Journal of Evolution of Medical and Dental Sciences*. 2016;5 (17):867-71.
 34. Ramakrishnan M, Ulland AJ, Steinhardt LC, Moisi JC, Were F, Levine OS. Sequelae due to bacterial meningitis among African children: A systematic literature review. *BMC Medicine*. 2009;7: 1-7.

© 2023 Anusha et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:

The peer review history for this paper can be accessed here:

<https://www.sdiarticle5.com/review-history/102616>