NON-TRAUMATIC COMA IN CHIDREN

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,extraocular 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 extraocular 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.

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

INTRODUCTION:

Non- traumatic coma (NTC) is a common cause of morbidityand 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]

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]

SYMPTOMS

1. Normal: Normal or no change from premorbid 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 reflux, abnormal motor muscle tone are the symptoms observed.[7]

MONITORING PARAMETRS

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, papillary reflux ,tone and fundus to corneal reflux, extra ocular movements, motor pattern, seizure type, and fundus picture.[8]

MANAGEMENT

1) MONITORING OF INTRACRANIAL PRESSURE

After admission to the hospital, the first thing that is to be monitored isblood 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 x10^9 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]

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.[11] CPP depends on the age of the child. In children outcome is poor if the CPP is consistently lower than 65-70mmhg.[10] ICP spikes can be prevented by maintaining systemic circulation, with plasma if the circulation is underfilled and otherwise with Ionotropic support.[12]

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.[14,15,16] 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]

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 haveongoing 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.[19,20,21] 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 prolonged seizures in a number of encephalopathies.[22,23,24,25,26,27] 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]

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 thisresearch 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 reflux, extra occular 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 refluxes assed 6hrs interval on time of admission, the analysis are lowest MGCS and worst brain stem reflux , The method used for this was spearman rank co-relation coefficient=0.577, they found the adverse effects, are improper occular 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. The coma defined as GCs below 12 for more than 6 hours, 14% of patients cause was unknown and the remainmostly 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 reflux. 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]

PREVENTION AND SUMMARY

Many infective causes of NTC are preventable with vaccines, and controlled or prevented by eliminatingmalnutrition/anemia. Other essential public healthmeasures 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]

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.

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