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Assesment of Outcome in COMA Patients in Relation to Etiology

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ABSTRACT

BACKGROUND:- *The comatose patient is one of the common neurological problems that an emergency physician encounters in the emergency room. Due to the heterogeneity of the causes of non-traumatic coma1,2, prediction of outcome in these patients is difficult and unfortunately no clinical, laboratory or electro-physiological parameter exists to determine their outcome. Early recognition of a comatose patient3,4 ,followed by timely and appropriate treatment strategies have been shown to significantly reduce mortality . .*

OBJECTIVES: - *1.to study the clinical profile of patients with non traumatic coma (NTC)*

2 To know the outcome of these patients with respect to etiology and GCS score

.**STUDY DESIGN**: *prospective cross sectional observational study.*

MATERIALS & METHODS:

100 patients admitted in the emergency department of Gandhi hospital during October 2014 to October 2015 ,who were diagnosed as nontraumatic coma and evaluated by detailed history and clinical examination were considered in this study.

Statistical methods: *The observations and data were analyzed in the statistical package social sciences (SPSS) trial version 11 by Chi-square and Fisher Exact test has been used to find the significance of proportions of outcome with various demographic, etiological and neurological factors.The level of significance was set at P<0.05.*

Results: *Intracranial causes (52%) formed the majority of cases followed by metabolic causes (32%) and drug/poisoning induced (12%). 14 different etiologies were observed causing non-traumatic coma in the present study.*

Mortality was highest in intracranial causes group, whereas 4 cases survived among 5 cases of coma due to drug overdose or poisoning. There were 18 cases of neuro-infection leading to coma out of which 4 cases were diagnosed to have tubercular meningitis, of which two were co-infected with HIV virus

Conclusions: *1. Results of this study indicate that an early indication of prognosis can be made from the combination of the GCS and etiologic subtype of coma. Knowing the predicted probability of outcome may assist in clinical therapeutic decisions.*

KEYWORDS : GCS-glasgow coma score, NTC-non-traumatic coma ICH-intracerebral haemorrhage ,SAH- subarchnoid haemorrhage

Introduction:

As coma can originate from diverse etiologies and is life threatening, it represents a challenge for emergency or critical care physicians. Coma also is a common cause for subsequent admission to the emergency department. Although there is no consensus on the precise cut-off point to define coma, in general a GCS lower than 8 points is used .Usually, as a first diagnostic step a differentiation is made between traumatic and non-traumatic coma (NTC)[1,2,3,4]. However, the determination of the cause of NTC is a challenge for the physician. In order to make adequate treatment decisions for patients suffering from NTC, it is vitally important to differentiate its etiology, such as severe sepsis, poisoning and hepatic encephalopathy. These conditions can be fatal if they are not detected or treated adequately.

An important first discrimination should be made between structural causes and non-structural causes of NTC by means of a computer tomography (CT) scan5. Structural coma can be due to cerebral infarction, intracranial hemorrhage, intracranial malignancy and central nervous system infection[6] (e.g. encephalitis or abscess). Non-structural coma include coma as a result of poisoning[7], epilepsy, extra cranial infections, circulatory shock, post-anoxic, cardiac arrest, respiratory failure, metabolic problems (such as hypoglycemia, ionic and acid–base disorders, hypothermia), hepatic encephalopathy[8] and uremic encephalopathy.

In comatose patients, evidence of widespread damage of brain stem or cerebral hemispheres at onset usually predicts death or severe disability. Therefore clinical signs that reflect extent and severity of dysfunction of cerebral hemispheres and/or brain stem were studied. Glasgow Coma Scale (GCS) Score reflects the integrity of cerebral functions[9]. Although there is a good correlation between early GCS scores and outcome it is important to consider other predictive features like Eye movements (in response to vestibular stimulation), pupillary responses[10], and corneal responses[11] primarily express functions regulated by the brain stem. 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.

MATERIALS AND METHODES:

This study is a prospective study of 100 cases of coma of non traumatic etiology, undertaken at Gandhi Hospital, secunderabad. All the cases were studied in detail with respect to history and clinical examination with a follow up daily till discharge or death to determine the outcome. The profile included assessment of severity of coma by GCS score. The outcomes were graded between death and good recovery.

Inclusion criteria:
- Patients presenting in comatose state(GCS score less than 8)
- Patients above the age 12 years

Exclusion criteria
- Patients having traumatic cause of coma

RESULTS AND DISCUSSION:
Analysis of age group revealed that majority of the cases appeared in the fifth to seventh decade. There were 32 patients in the age group of 51-60 years. There was no correlation between age of the patient and mortality. However, patients with age more than 50 were more likely to have had a bad outcome compared to the other groups.

Out of 100 cases studied 62 were males and 38 were females. The difference in mortality rates among males and females were not statistically significant. (p>0.05)

From history, onset of coma was divided into abrupt and gradual onset. The temporal profile of the onset of coma provided a clue to reach the probable diagnosis. Cerebrovascular causes, status epilepticus, hypoglycemia, drug or poison induced comas formed majority of the abrupt onset of coma. In this study however there was no statistical significance between the onset of coma and mortality **(P value is 0.844**.). Two Out of four TBM cases presented with abrupt onset of coma. Both also had history of seizures prior to onset of coma.

From the history of(figure-1) preceding complaints, headache and vomiting, suggestive of symptoms of raised intra-cranial pressure, were the more common complaints in this analysis. Among the risk factors in CVA group diabetes and hypertension were the major risk factors, and hypertension was more associated with intracerebral hemorrhage. There were two cases of subarachnoid hemorrhage and both were associated with hypertension. Patients presenting(table-2) with Hypertension had bad outcome as shown by the **p-value:0.024 (p value <0.05)** which is statistically significant. Hypertension was present more commonly in patients with cerebrovascular disease and also more commonly associated with mortality.

Among the patients with papilloedema, **p-value was 0.01** which is **statistically significant.** .However there was no statistical significance in patients with diabetic retinopathy(p-value:0.22)and hypertensive retinopathy(p-value:0.632).

While analyzing the etiology(table-1) of coma seemed to influence the outcome as present study indicated that the intracranial causes (34%) were the commonest cause of medical coma followed by metabolic coma (32%) ,neuroinfections (18%) , Drug and poisoning induced coma comprised 6% of cases in each sub group and status epilepticus (2%). **There was higher mortality and morbidity (48.2%) in patients with coma due to intracranial causes** Among the etiologies**, CVA** had statistical significance **(p-value:0.004)**

In the present study Out of 34 cases of cerebro vascular accidents ,there were 16 cases of cerebral infarctions followed by 12 cases of ICH, 2 cases of SAH, , and 4 cases of CSVT[12], one of them being in postpartum. Out of 16 cases of cerebral infarction 3 cases were known cases of rheumatic heart disease.Among cerebral infarcts and hemorrhages, hemorrhages carried the worse prognosis.

There were 18 cases of neuro-infection leading to coma out of which 4 cases were diagnosed to have tubercular meningitis[13] one among them having tuberculoma on CT brain. One of them was co infected with HIV virus, one of them developed hydrocephalus in the course of hospital stay. She was taken up for shunt surgery, but died post operatively. Thus HIV co-infection is a major risk factor, among neuro-infections, leading to mortality**.**

Also there were four cases of cerebral malaria presenting as coma, reflecting increased incidence of malaria in our area.

There were 32 cases of (Figure1-2)metabolic cause of coma out of which 8 were in hepatic coma .All these patients of hepatic coma were alcoholics and four patients presented with upper GI bleeding. There were 3 cases of uremic encephalopathy and all had chronic renal failure . One patient expired soon after he was brought to emergency department. The other two cases were taken up for emergency dialysis and gradually recovered from coma.Two cases of hypoglycemic coma and one case of diabetic ketoacidosis showed good recovery. The preservation of pupillary light reflexes in the presence of deep coma should alert the clinician to a possible metabolic coma. Among 16 cases of metabolic cause of coma there were 7 survivals**.** Metabolic coma carried better prognosis in our study in comparison with intracranial causes.

Six cases of drug overdose were present, out of which two had consumed Alprazolam tablets, one had come with consumption of phenytoin,one with nitrobenzene ingestion who was treated with methylene blue and the patient regained consciousness . Drug induced coma showed the best recovery. So drug induced comas can be taken as independent predictors of outcome.

In this study the severity of coma at the time of presentation has been assessed by Glasgow coma scale. The GCS score at the time of presentation was between 3 and 8 in this study. There were total 54 cases with scores between 3-5 had 35 mortality and 19 survivors. Among the patients with GCS score 3-5 that made a good recovery were drug overdose and OP poisoning patients. In the group of GCS score 6-8 there were 46 cases with 7 mortality and 39 recovered . Most of the cases with scores of 3 had intracerebral hemorrhage and.The group of patients who had GCS score between 3-5 at the time of admission had the maximum mortality, in compared to the group of patients with GCS score between 6-8 with a **p -value:0.000001** which is **statistically significant**.. Thus there was an inverse relation of mortality to GCS score.. None in the CVA group could achieve recovery with low GCS.

Another variate found useful in predicting outcome of non-traumatic coma was the presence or absence of (Figure1-2)brainstem reflexes. In this study there were 9 cases with absent brainstem reflexes and all of them expired. The presence of Brainstem reflexes in patients with coma at the time of admission predicts good outcome ,With brainstem reflexes intact there was 36.9% mortality. The chi-square statistics is 13.6578 and the **p-value is 0. 000219 (p<0.05)** which is **statistically significant**..

Abnormal respiratory pattern was seen in total 58 patients out of which 38 died.. Thus abnormal respiration was associated with significant mortality (chi-square=31.35261,**p value:0.00001**(P<0.05) which is **statistically significant**

Absence of pupillary and corneal reflexes 6 hours after the onset of coma has been shown with universal fatality.

Conclusions:
1) There was no significant relation between the age group,sex and the outcome.

2) Onset of coma is statistically not associated with outcome. However Mortality was more in patients who presented with abrupt onset of coma.

3) Mortality was highest in intracranial causes group,

4) Hypertension was present more commonly in patients with cerebrovascular disease and also more commonly associated with mortality.

5) Results of this study indicate that an early indication of prognosis can be made from the combination of the GCS score and etiology.

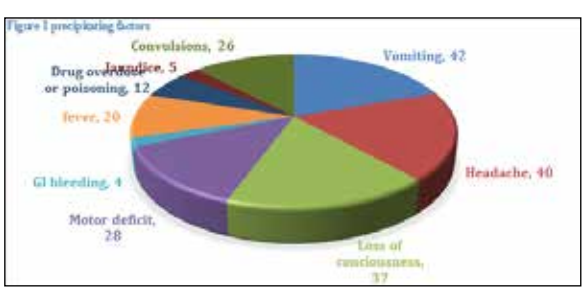
6) Absent brainstem reflexes, abnormalities of respiration, decerebrate posture were very much significantly associated with poor outcome.

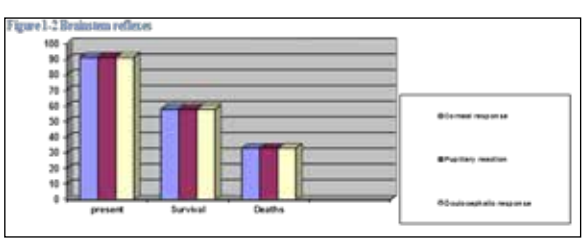
7) Drug induced coma carried better prognosis and thus was an independent predictor of outcome.

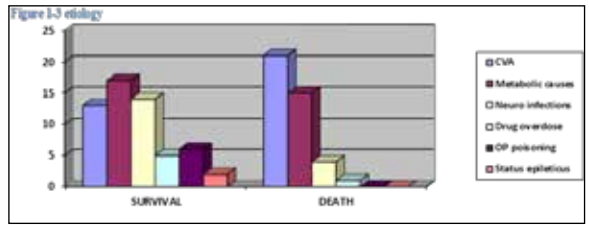
TABLE:-1 AETIOLOGY	NO
1) Cerebrovascular Accidents	34%
(a) Intracerebral Hemorrhage	12
(b) Infarctions	16
(c) CorticoVenous thrombosis	4
(d) Subarachnoid Hemorrhage	2
2) Metabolic causes	(32%)
(a) Hepatic Encephalopathy	8
(b) Uremic Encephalopathy	3
(c) Hypoglycemic coma	5
(d) Diabetic Ketoacidosis	3
(e)hyperglycaemic hyperosmolar state	5
(f) Hyponatremia	4
(g) Hypernatremia	2
(h) Myxedematous coma	2
3) Neuro Infection	(18%)
(a) Bacterial Meningitis	6
(b) Tubercular Meninigitis	4
(c) Cerebral Malaria	4
(d) viral meningoencephalitis	3
(e) fungal meningitis	1
4) Drug overdose	(6%)
5) Status Epilepticus	(2%)
6) Organo Phosphorus Poisoning	(6%)
7) cause not known	(2%)

Table -2 risk factors

Past history	Total	Death	Survival	P value
HTN	44	24	20	0.024
DM	30	14	16	0.535
Liver dis	16	6	10	0.69
TB	10	5	5	0.588
CKD	10	6	4	0.224
COPD	5	4	1	0.077
CVA	6	1	5	0.194
Epilepsy	4	1	3	0.482







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