ORIGINAL ARTICLE

AN AETIOLOGICAL AND CLINICAL STUDY OF 150 CASES OF COMA

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ABSTRACT: INTRODUCTION: Coma is a common medical problem. The importance of this class of neurological disorders points to the necessity of a systematic approach to their diagnosis and management. In present study we evaluate 150 cases of coma, their management and clinical outcome. **AIM AND OBJECTIVES:** A majority of admissions in intensive care unit are presenting with impairment of consciousness. It is felt that a more detailed study of these cases is needed for better management so an effort is made to study 150 consecutive cases admitted with coma of medical aetiology to determine the aetiological profile, clinical localization of lesions and to predict the prognosis. Simultaneously it has been tried to standardize the initial protocols in the management of patients admitted with coma. **CONCLUSION:** In the present study the most common cause of coma is metabolic and infective 42%, among these, infective are common 21% and metabolic are next common 20%. In suprtentorial causes ischaemic strokes are more common 53% than haemorrhagic strokes. Among infective causes tuberculous meningitis, cerebral malaria are common causes. Cortical venous thrombosis is common in females. In drug poisoning organo phosphorus poisoning is common.
KEYWORDS: Coma, Supratentorial, Infratentorial.

INTRODUCTION: Since the days of the Greeks men have known that normal conscious behavior depends on intact brain function. However the range of awake and intelligent behavior is rich and variable. The clinical abnormalities are difficult to recognize, unless there are substantial deviation from the normal. The brain can tolerate only a limited amount of physical injury without suffering irreparable harm.[1]

Consciousness is the state of awareness of the self and the environment and coma is the total absence of awareness of self and environment, even when the subject is externally stimulated. Between the extreme states of consciousness and coma stand variety of altered states of consciousness.[2-7]

Two physiologic components govern conscious behavior namely content and arousal. The content of consciousness represents the sum of cognitive and affective mental functions. Any lesion, which interferes with full cognitive function, diminishes the content of consciousness. Arousal is the aspect of consciousness and at least behaviorally is closely linked to the appearance of wakefulness.[8-10]

Coma is a common medical problem. The importance of this class of neurological disorders points to the necessity of a systematic approach to their diagnosis and management.

MATERIALS AND METHODS: The study was conducted on 150 patients of medical coma, who were admitted in acute medical care unit, in Government General Hospital, Kurnool during the years 2003 August to 2005 May.

Patients below 12 years old are excluded, patients on treatment with loss of consciousness were also excluded, and whose coma caused by trauma was excluded.

Once the vital functions have been protected history was obtained from the patient attendants. General physical examination and careful neurological examination was done. All data relevant for coma was noted and entered on preformed Performa of neurological examination of coma. Relevant investigations were done and treatment was started.

Examination of the patient was conducted twice a day and when the patients deteriorated. All the patients were followed continuously till the patient fully recovered from the unconsciousness or death or discharge. The methods of eliciting neurological signs relevant to coma and biochemical investigations, which were done, are mentioned below.

DISCUSSION: The study of 150 patients of non-traumatic coma admitted in GGH, Kurnool in AMC was studied for various etiological causes, the symptomatology, signs and follow up during their stay in the hospital. Several prospective as well as retrospective studies have been undertaken to study the causes of coma and the value of physical signs in diagnosing the causes of coma.

Present work includes study of 150 cases of coma and a comparison of results with the works of BATES, PLUM and POSNER done at New York hospital.

The various conditions that produce coma in the various studies are divided into 3 groups.[11]

1. Supratentorial lesion.
2. Sub tentorial lesion.
3. Diffuse metabolic and infective brain diseases.

1. **ETIOLOGIES:** In comparison with the study of PLUM and POSNER the present study shows the different etiologies and percentage of causes of coma show in table 1.

In PLUM series the most common causes of coma was diffuse and metabolic brain dysfunction in 65.2%. Patient in the present study it was seen only in 42%. In PLUM series metabolic are commonest cause, next is drug poisoning. But in this series commonest is infective next is metabolic next one is drug poisoning.

Psychiatric causes of coma were 1.6% in PLUM series, but in this study no cases is recorded.

2. **CAUSES OF COMA:** In present study the different causes of coma are sub grouped as shown in the table-2. Commonest cause of supratentorial lesions producing coma in PLUM series was cerebral hemorrhage (15.2%). In the present study also it has got a high incidence of (13.3%). But the highest incidence was cerebral ischaemia in this study that is 17.3% as compared with plum series of only 1.8%. Though unilateral cerebral infarction rarely produces coma. In the present study supratentorial lesions causing coma may be

due to massive cerebral infarction (or) bilateral cerebral infarction may be the cause of coma.

The sub tentorial lesions causing coma in this study was compared with PLUM series. Brain stem infarction (or) haemorrhage was the commonest cause of coma. In PLUM series it was 10.63% as compared with our study 5.3%.

In the present study the diffuse metabolic and infective cause of coma is 52%. In PLUM series also it was the common cause for coma. Among infective, metabolic and drugs the infective cases 21% (32 cases), the next common is metabolic i.e., 20% (31 cases), next one is drug poisoning 10% (15 cases). Among infective causes tuberculous meningitis, and cerebral malaria are the commonest causes of coma i.e., 7.3% and 12% respectively. But in PLUM and POSNER series they have not recorded any cases, probably the incidence of tuberculosis and malaria was not present in New York.

Among the drug poisoning OP compound is commonest one 8%, because easily availability and most of the people are formers. But in PLUM and POSNER study the common cause was anti-psychotic drugs, phenytoin, and barbiturates.

3. **Age incidence:** Cerebral vascular accidents are more common in the age group of 45-55 except cortical venous thrombosis, which is more common in younger age group. Metabolic coma is more common in the age group of 36-45 tuberculoses meningitis is more common in 26-35. Malarial fever is also more common in younger age group.

4. **Sex incidence:** The incidence of coma is more common in males in this study, because cerebral ischemia, cerebral haemorrhage is more common in males. All cases of cortical venous thrombosis causing coma are females. So any female patient during post-partum period the cause of unconsciousness with focal neurology deficit may be due to venous thrombosis and it must be excluded by neurological examination. Diffuse, metabolic and infective brain dysfunction is more common in males. Except sub arachnoid haemorrhage which is more common in female in this study.

5. **Mode of onset:** Cerebrovascular accidents,i.e cerebral ischaemia cerebral haemorrhage and sub arachnoid haemorrhage are having acute (or) indistinct onset.(TABLE - 3)

Acute onset in the present study is 36% and gradual onset is 43% and indistinct is 21%. In acute onset supratentorial causes are common. In gradual onset metabolic and infections are common causes. In indistinct again metabolic and infections are common causes for coma.

6. **Preceding symptoms:** (TABLE 4)- Before the patients developed coma, 23 cases of cerebral ischaemia 20 case of cerebral haemorrhage preceded by loss of power Diplopia is a preceding symptom in 3 cases of infarct and 4 cases of haemorrhage.

Fever is preceding complaint in 4 cases of ischemia and 1 case of haemorrhage and fever is preceding symptoms in 4 cases of DKA and 6 cases of hepatic encephalopathy. All cases of malaria and TBM are preceded by fever. Vomiting are preceding complaints in 10 cases ischemia,

15 cases of haemorrhage, 3 cases of uremic encephalopathy, 6 cases of DKA and 5 HEP and 14 cases of malaria, 3 cases of TBM.

Diplopia is preceding complaint in 3 case of ischemic stroke, 4 cases of hemorrhagic stroke. 4 cases of hypoglycemia has given history of anti-diabetic drugs at night and skipped dinner.

All case of OP compound poisoning has given history of OP compound consumption and 3 case of diazepam has given history of consumption of diazepam. Jaundice was preceding symptoms in all hepatic comas.

7. **Past illness:** Table-5 shows the past illness in coma patients. 12 patients of ischaemia strokes have given history of transient ischaemic attack and 6 cases have given past history of strokes. One cases of haemorrhagic stroke has given history of transient ischaemic attack and 3 cases have given history of strokes. Past History of ischaemic heart disease was present in 5 cases of ischaemic stroke, 6 cases of hemorrhagic stroke, 6 cases of diabetic keto acidosis, one case of hypoglycemia and one case of uremic encephalopathy. Two cases supratentorial lesion, 3 cases of sub tentorial has given history of Vertibro basilar insufficiency. 2 cases of hypoglycemia, 4 cases of diabetic keto acidosis and one undiagnosed case have given history of transient ischaemic attack.

One case of ICSOL has given history of ear discharge. 6 cases of hepatic encephalopathy have given history jaundice. 6 cases of tuberculous meningitis, 4 cases of malaria, one case of encephalitis has given history of pulmonary tuberculosis.

8. **Risk factor:** Table 6 shows risk factors in comatose patients. Out of 26 cases of ischaemic strokes 17(11.2%) cases were diabetic, 10(10.6%) cases were hypertensive, 16 cases were smokers as well as alcoholic. It was observed that in hemorrhagic cases 16(80%) cases were hypertensive, 9 cases were smokers, 7 cases were alcoholic. All cases of hypoglycemic were diabetics and hypertensives. Out of 12 cases of diabetic keto acidosis, 8cases were hypertensive.

In all cases of uremic coma it is observed that they are diabetics as well as hypertensives. On general examination they were found to be anaemic. Among all cases 62 patients were smokers and 49 cases were alcoholic. Smoking and alcohol are risk factors for ischaemic strokes and ischaemic heart disease.

9. **General examination:** Table No 7: Among all the cases 32 cases are anaemic. Almost all cases of diabetic keto acidosis were dehydrated. 4 cases of diabetic keto acidosis have got septic skin lesions and 1 case of hypoglycemia has septic skin lesions. Almost all cases of hepatic encephalopathy have got pedal edema. 5 cases of malaria have shown anaemia, 6 cases have showed jaundice, 3 cases have showed purpuric spots and 6 cases were found to be dehydrated. 2 cases of tuberculous meningitis have shown enlarged cervical lymph nodes. All most all cases of organo phosphorous poisoning have showed fasciculation.

10. **Respiratory pattern:** Table 8 shows respiratory pattern at admission. In the present study many of supratentorial cases has shown cheyne stroke breathing. NORTH and GENNETT emphasized that cheyne stroke breathing can accompany bilateral damage of the descending pathways anywhere from forebrain to upper pons. 23 patients showed rapid deep respiration in metabolic deep coma. Most of infective cases showed normal respiration. In all hepatic coma patients rapid deep respiration has been observed. Cases of diazepam have showed slow shallow breathing. As coma progressed most of cases showed ataxic breathing.

11. **Eye Position:** Eye position and movement. It is very important sign indicating localization of lesion. Structural lesion shows either conjugate deviation or dysconjugate eye position. In the present study supra tentorial lesions showed either conjugate deviation or mid position or conjugate roving with slight divergence indicating intact pathways. It is observed that in supratentorial lesion the conjugate deviation of eyes is towards the side of lesion and opposite to the side of hemiplegia. Though fishner described conjugate deviation towards paralyzed side, in supratentorial lesions, we did not observe this finding. The present study showed a high incidence of conjugate roving movements. 32 cases of supratentorial lesions showed conjugate roving movements indicating that brain stem's intact.

In subtentorial lesion patients had mid eye position on admission and dysconjugate movements. 29 cases of metabolic and diffuse brain dysfunction have showed conjugate roving movements. And conjugate movements are also observed in tuberculous meningitis patients due to cranial nerve irritation. Conjugate deviation to the opposite side is seen due to irritative lesion. PANT et al also observed this finding. In drug toxicity the eyes are mid position indicating external Ophthalmoplegia, ORTH, ALMEDIA described these finding in their study. In deep coma all patients showed fixed mid position of eyes.

12. **Pupillary Reflex:** The importance of pupillary reflex lies in the locating the level of lesion. In supratentorial lesions small but reactive pupil observed in 15 cases. The similar findings were observed by FISHNER in his series. The observations of small reactive pupils are due to involvement of diencephalan. FISHNER et al. observe this finding in his study. Unilateral pupillary constriction and ptosis (HORNERS Syndrome) was observed in 9 cases of suprtatentorial lesion. This finding was recorded by CRILL in his patients. Lateral due of time in deep coma the supratentorial lesion showed pupillary abnormalities like dilated and fixed pupil due to transtentorial lesion. Unilateral dilatation is observed first in supratentorial lesion, may be due to uncal herniation causing midbrain ischaemia. This is the crucial period where treatment helps in arresting the progression of transtentorial herniation. These features were observed in PLUM and POSNER series. All cases of subtentorial lesion showed either unilateral (or) bilateral pupillary change. In this present study 3 cases showed bilateral pinpoint pupils indicating pontine haemorrhage. Of the subtentorial lesion unilateral dilated pupil has seen in 4 cases. In all cases of space

occupying lesion it was observed as a fixed dilated pupil. In metabolic coma small reactive pupil was observed in 18 cases. This implies that focal changes of pupil indicates structural lesion as described by Plummer and Posner. Pupillary pathways relatively resistant to metabolic insult. The presence or absence of light reflex is the single most important physical sign distinguishing structural from metabolic coma.

All cases of organo phosphorous compound poisoning has showed pinpoint pupil. The pupillary changes in tuberculous meningitis showed all varieties indicating cranial nerve involvement due to raised intra cranial tension.

13. **Oculocephalic reflexes:** Table 11 Shows oculocephalic reflex is absent in only 3 cases of supratentorial lesions. It was present in 46 cases out of 49 cases. In subtentorial lesions oculocephalic reflex are absent in 8 cases and in one case it was present. In metabolic and infective coma oculocephalic reflex were retained in 90.4% cases were it was absent in 9.6% cases. In all cases of OP poisoning it was present but in all cases of diazepam poisoning it was absent. In all cases of anoxic encephalopathy it was absent.

14. **OCULOVESTIBULAR REFLEX:** Table 12 shows oculovestibular reflex. It was present in 46 cases out of 49 cases. This indicates that the site of lesion is above tentorium and intact pathways though impairment was observed in 3 cases of supratentorial lesion, due to conjugate deviation of eyes. This same findings was observed by BIEL SCHOW sky et al.

In subtentorial lesion 8 cases showed absent oculovestibular reflex and 1 case showed presence of oculovestibular relex.

In metabolic coma oculovestibular reflex is absent only in Anoxia. In drugs toxicity oculocephalic reflexes was absent in diazepam poisoning only.

15. **Meningeal signs:** Meningeal signs are elicited in all cases of tuberculus meningitis. 7 cases of cerebral haemorrhage and 3 cases of subtentorial lesion showed meningeal signs. It may be due to meningeal irritation with blood. 6 cases of malarial fever have shown neck rigitidy.

16. **Fundus examination:** Neurological examination is not complete without fundus examination. It gives valuable information. In present study 20 patients showed raised intracranial tension changes and giving caution to avoid lumbar puncture. Haemorrhage and exudates were observed 21 of cases. One case of subarachnoid haemorrhage showed sub hyloid haemorrhage. 8 cases of Tuberculus meningitis showed papilledema.

17. **MOTOR SYSTEM EXAMINATION:** Examination of the motor system gives either focal or diffuse brain dysfunction. In the present study the supra tentorial lesions showed high

incidence of neurological deficit. 22 cases of ischaemic stroke showed hypertonia remaining 4 cases showed hypotonia, 26 cases showed neurological deficit in the form of hemiplegia and decarticate rigidity in 6 cases. 16 cases of haemorrhage showed hypertonia and all cases of neurological stroke showed neurological deficit in the form of hemiplegia. 10 cases of malaria showed hypertonia and 6 cases of hepatic encephalopathy has showed plantar extensor.

18. **DURATION AND PROGNOSIS:** Table – 16 shows the duration and prognosis. In the present study the prognosis was good for metabolic causes expect uremia. Encephalitis and undiagnosed cases showed a bad prognosis. Hypoglycemia patient showed improvement within 2 - 4 hours most of patient regained consciousness in causality itself. All diabetic keto asidosis patients showed recovery. Hepatic encephalopathy shows 66.7% improvement 33.3% mortality.

Malarial fever patient showed good improved, out of 20 case 2 patients expired on 1st day. All patient of tuberculous meningitis were recovered and 2 patients has suffered with vascular insult and discharged with residual heriperesis.

Most of supratentorial cases expired within 24 hours, of all cases 11 cases are improved with residual neurological deficit. 6 cases of organo phosphorous poisoning and 1 case of diazepam poisoning have taken ventilator assistance.

Supratentorial Space occupying lesion showed 100% mortality. Sub tentorial cases showed good prognosis with neuro surgery intervention.

SUMMARY AND CONCLUSIONS:

1. In the present study the most common cause of coma is metabolic and infective 42%, among these, infective are common 21% and metabolic are next common 20%. In suprtentorial causes ischaemic strokes are more common 53% than haemorrhagic strokes. Among infective causes tuberculous meningitis, cerebral malaria are common causes. Cortical venous thrombosis is common in females. In drug poisoning organo phosphorus poisoning is common compared with PLUM and POSNER study. We come cross three undiagnosed cases but not in PLUM and POSNER study because availability of sophisticated investigations and autopsy facilities to them.
2. Cerebro vascular accidents have acute onset. Most of metabolic and infective coma have gradual onset. Drug poisoning showed gradual onset.
3. The presence of reactionary hypertension in few cases of supra and subtentorial lesions, who were not a known hypertensives cautioned to start and hypertensive therapy until other evidence of hypertensive end organ changes are excluded with investigations. Conjugate deviation to opposite side of hemiplegia and conjugate roving movements are more in supra tentorial lesions, dysconjugate or mid position is more common in sub tentorial lesions. Conjugate roving movement were commonly seen in all metabolic and drug toxicity except in diazepam toxicity.

4. Use of multiple signs greatly strengthened the accuracy of diagnosis and prognosis. Prognosis was very bad in all sub tentorial lesions, uremic encephalopathy, encephalitis, cerebral haemorrhage and undiagnosed patient. Prognosis is satisfactory in supra tentorial lesions if the management arrest transtentorial herniation. Chances of recovery are 66.4% in hepatic coma in present study. Good prognosis is observed in cases of hypoglycemia diabetic keto acidosis, anoxia, drug toxicity and tuberculous meningitis if treatment is started early.

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TABLE – I
COMPARISION OF THE AETIOLOGIES OF PLUM AND POSNER STUDY WITH THE PRESENT STUDY

	PLUM SERIES			PRESENT STUDY		
	Total	Number of cases	Per%	Total	Number of cases	Per%
I. SUPRA TENTORIAL	500	(101)	20.2%	150	(49)	
Cerebral Haemorrhage		76	15.2%		20	13.3%
Infarction		9	1.8%		26	17.3%
Abscess		6	1.2%		2	1.33%
Tumors		7	1.4%		1	0.66%
II. SUB TENTORIAL		(65)	13.0%		(9)	
Infarction & Haemorrhage		53	10.6%		8	5.33%
Compressive Lesion		12	2.4%		1	0.66%
III. DIFFUSE AND METABOLIC BRAIN DYSFUNCTION	300		65.2%			
Sub Arachnoid Haemorrhage		13	2.6%		3	2%
Meningo-Encephalitis		14	2.8%		3	2%
Tuberculous Meningitis		-	-		11	7.33%
Malarial Fever					18	12%
Extrinsic Metabolic disorders						
Anoxia		10	2%		3	2%
Hypoglycemia		16	3.2%		7	4.66%
Uremia		8	1.6%		3	8%
Diabetic Ketosis		12	2.4%		12	2%
Hepatic Encephalopathy			29.8%		6	4%
Drug Poisoning		149			15	10%
IV. PSYCHIATRIC COMA			(8)	1.6%		
V. UNDIAGNOSES			(-)	-	3	2%

TABLE NO – II

DIFFERENT CAUSES OF COMA IN GOVERNMENT GENERAL HOSPITAL, KURNOOL

I. NEUROLOGICAL		
1) SUPRA TENTORIAL LESIONS		
1.	Hemorrhage	20 (13.3%)
2.	Infarction (Arterial thrombosis, embolism and venous occlusions)	26 (17.3%)
3.	Cerebral Abscess	2 (1.33%)
4.	Tumors	1 (0.66%)
2) INFRA TENTORIAL LESIONS		
1.	Infarction (or) Hemorrhage	8 (5.33%)
2.	Tumors	1 (0.66%)
3) DIFFUSE INFECTIVE BRAIN DISEASES AND OTHERS		
1.	TUBERCULUS MENINGITIS	11 (7.33%)
2.	MALARIA	18 (12%)
3.	ENCEPHAILITIS	3 (2%)
4.	EPILEPSY (8)	8 (5.3%)
5.	SUB ARACHANOID HAEMORRHAGE	3 (2%)
II. METBOLIC CAUSES & INFECTIVE CAUSES		
1)	ANOXIA	3 (2%)
2)	HYPOGLESEMIA	7 (4.66%)
3)	DKA	12 (8%)
4)	UREMIC ENCEPHALOPATHY	3 (2%)
5)	HEPATIC ENCEPHALOPATHY	6 (4%)
III. DRUGS		
1)	OP COMPOUNDS	12 (8%)
2)	DIAZEPAM	3 (2%)
3)	OTHERS	
IV. PSYCHIATRIC CAUSES		
V. UNDIAGNOSED		3 (2%)

TABLE - 3

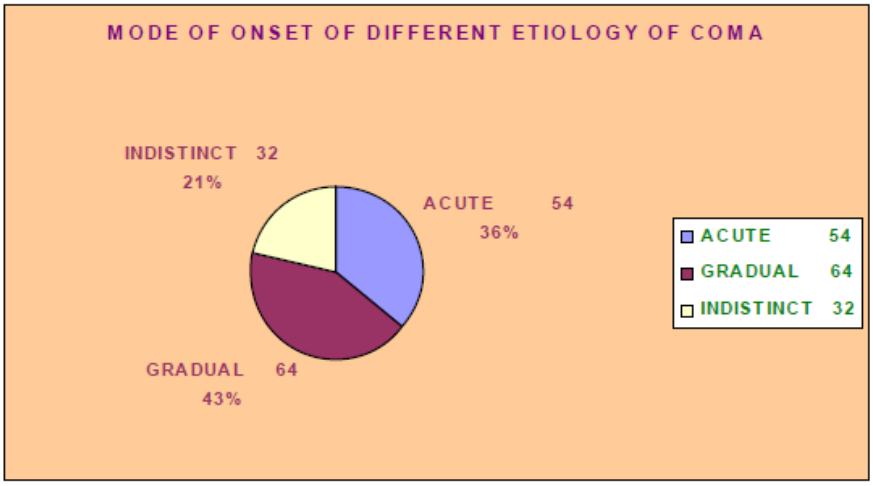


TABLE NO :4
PRECEEDING SYMPTOMS BEFORE UNCONSCIOUSNESS DIRECTLY FELATED TO COMA (COMPLAINTS)

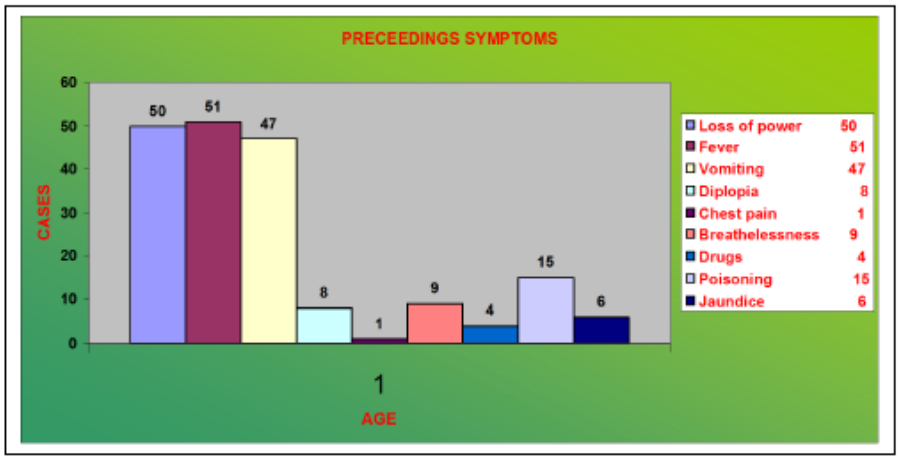


TABLE NO :5
PAST ILLNESS

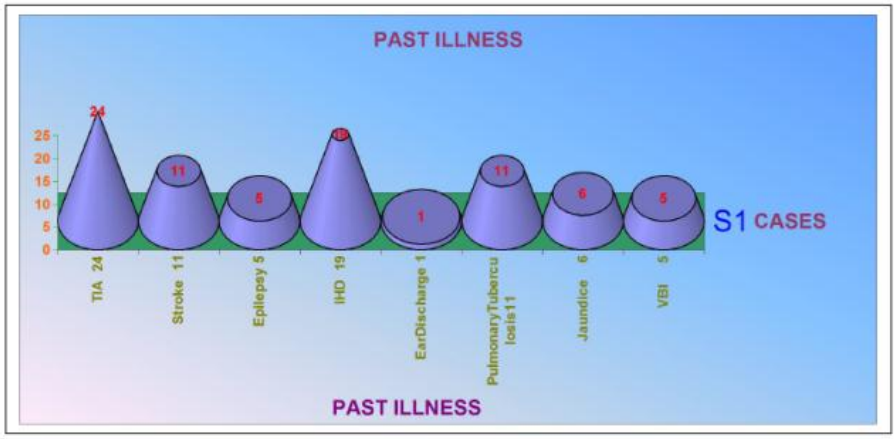


TABLE NO :6
ASSOCIATED ILLNESSES AND PERSONAL HISTORY (RISK FACTORS)

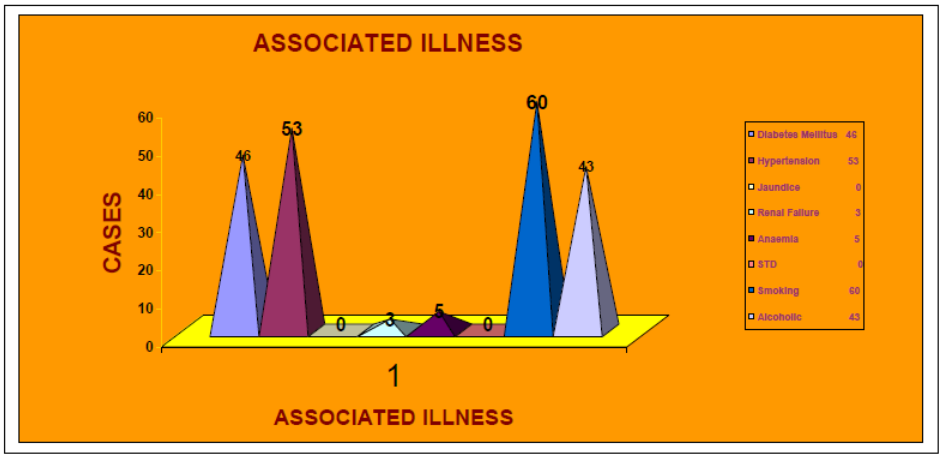


TABLE NO :7
GENERAL EXAMINATION

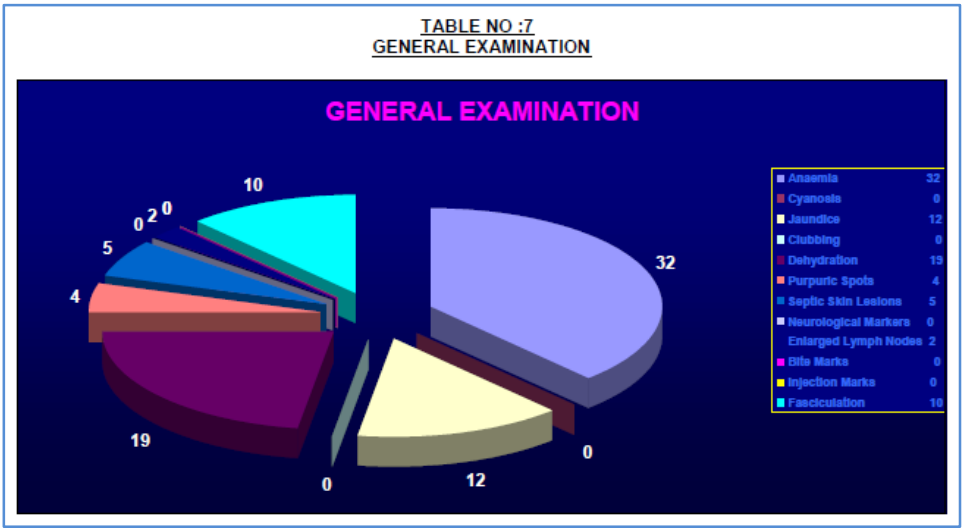


TABLE NO – 8
RESPIRATORY PATTERN AT ADMISSION

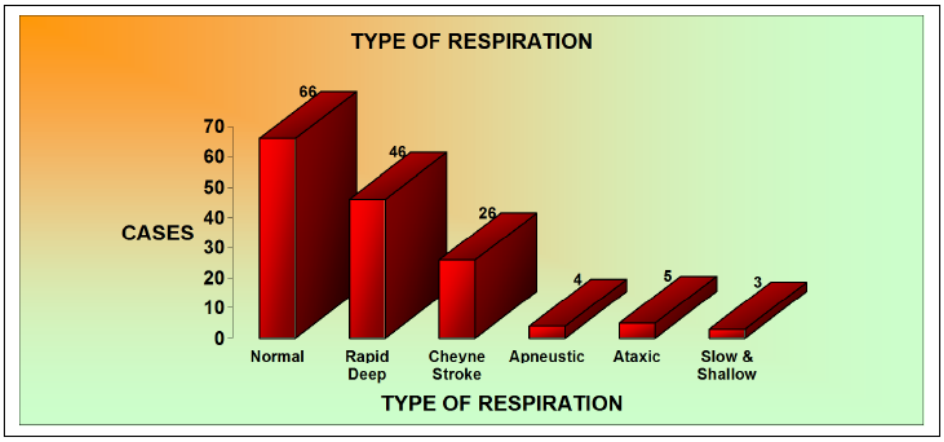


TABLE NO – 9
EYE POSITION AND EYE MOVEMENTS

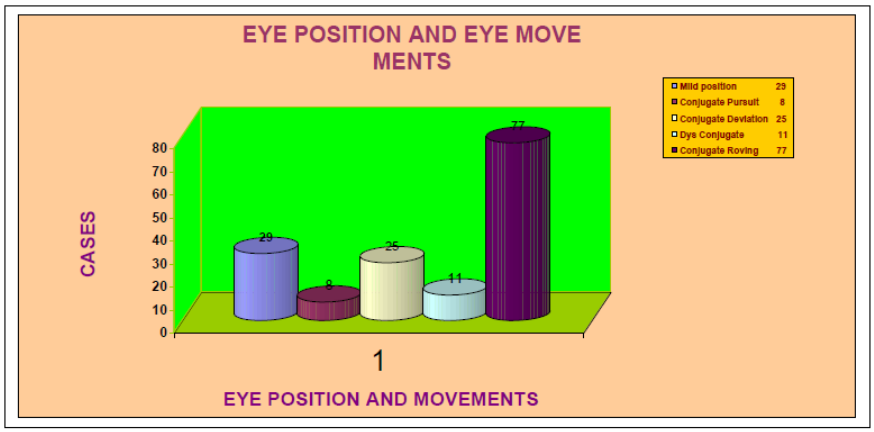


TABLE NO – 10
PUPILLARY CHAGNES OBSERVED AT ADMISSION

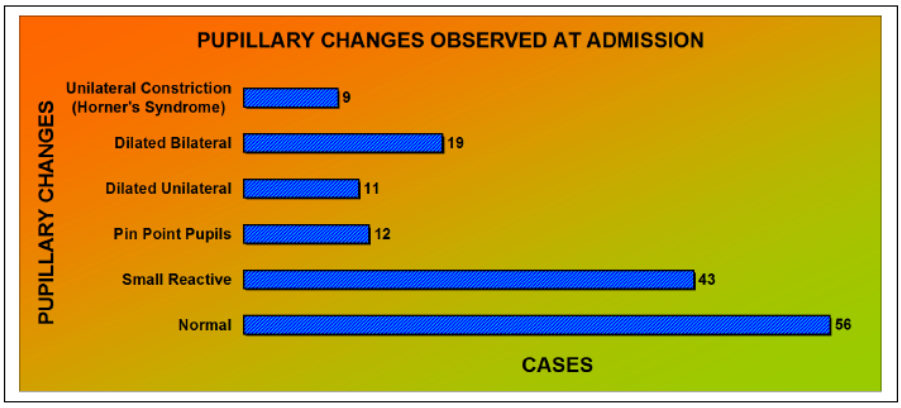


TABLE NO – 11
OCULO CEPHALIC REFLEX

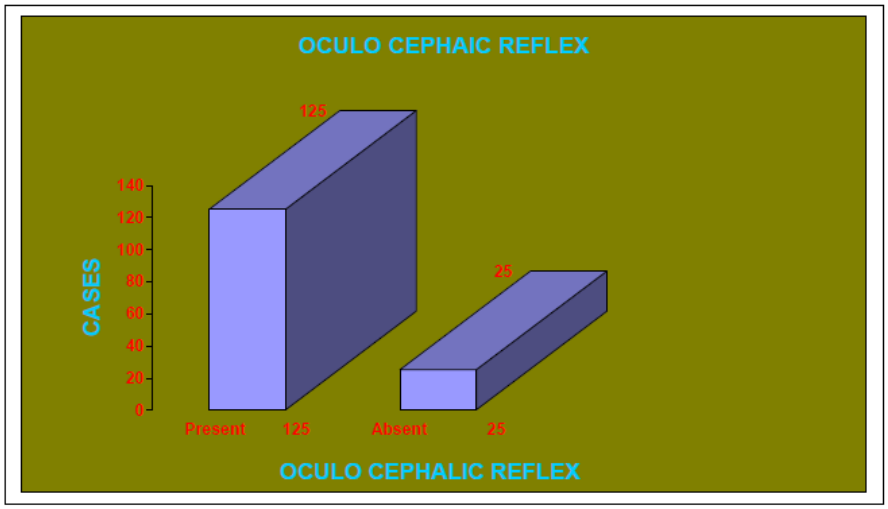


TABLE NO – 12
OCULO VESTIBULAR REFLEX

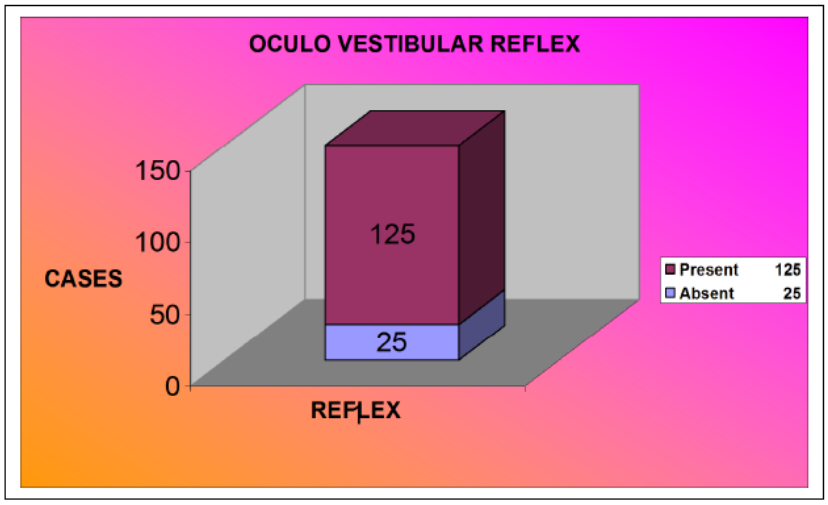


TABLE NO – 13
MENINGIAL SIGNS

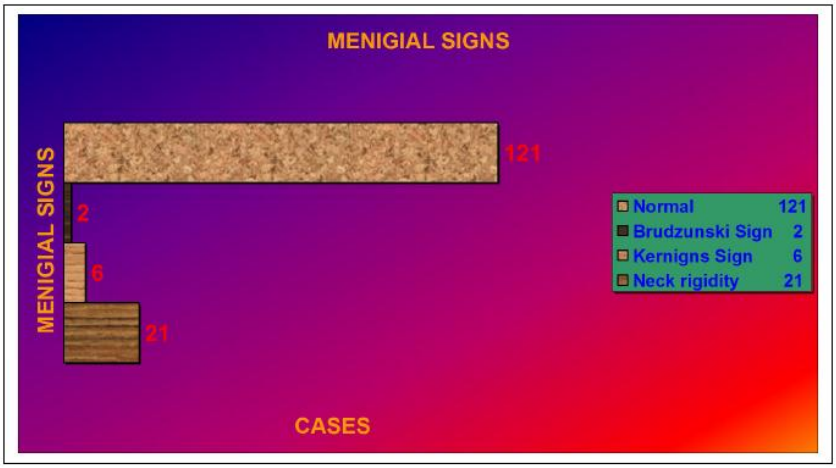


TABLE NO – 14
FUNDUS CHANGES (OPHTHALMOLOGICAL EXAMINATION)

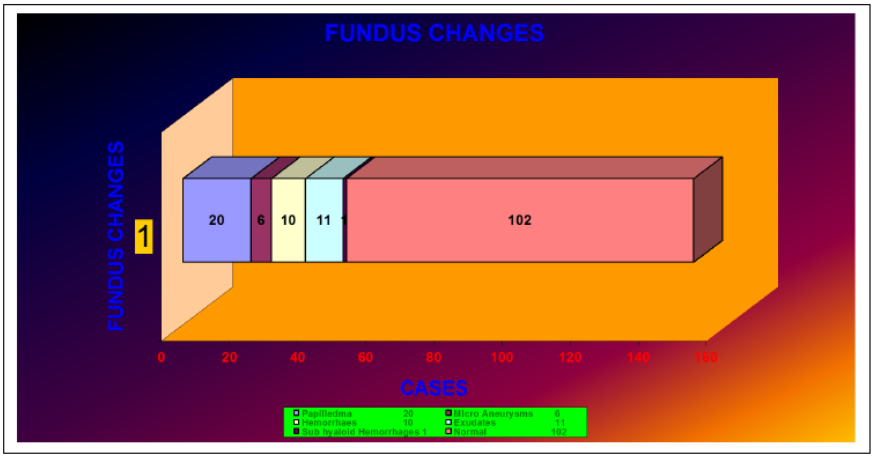


TABLE NO – 15
MOTOR SYSTEM EXAMINATION

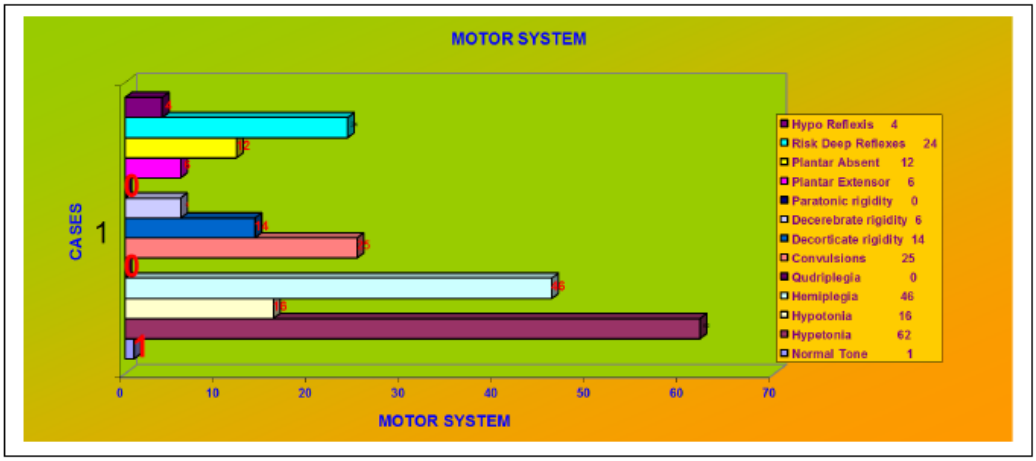
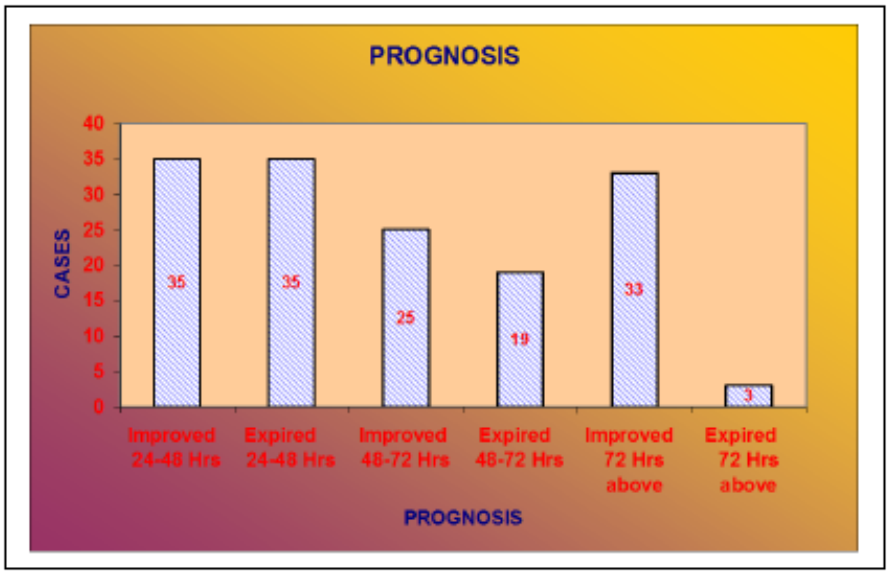


TABLE NO. 16

PROGNOSIS OF COMA IN PATIENTS

I. ETIOOLOGY OF COMA	TOTAL	24-48 HR		48-72 Hr		5th day		
		Improved	Expired	Improved	Expired	Improved	Expired	
I. NEUROLOGICAL CAUSES								
1) SUPRA TENTORIAL LESIONS			13		5	2		
1. Hemorrhage								
2. Infarction			9		8	9		
(Arterial thrombosis, embolism and venous oclusions)								
3. Cerebral Abscess			1			1		
4. Tumors					1			
2) INFRA TENTORIAL LESIONS								
1. Infarction (or) Hemorrhage			4		2	2		
2. Tumors						1		
3) DIFFUSE INFECTIVE BRAIN DISEASES AND OTHERS								
1. TUBERCULUS MENINGITIS			1	8		2		
2. MALARIA	12	2		3		1		
3. ENCEPHAILITIS						3		
4. EPILEPSY	6			2				
5. SUB ARCHANOID HAEMORRHAGE			2		1			
II. METBOLIC CAUSES & INFECTIVE CAUSES								
a. ANOXIA						3		
b. HYPOGLESEMIA	7							
c. DKA	6			3		3		
d. UREMIC ENCEPHALOPATHY			1		1		1	
e. APATIC ENCEPHALOPATHY			1	2	1	2		
III. DRUGS								
1. OP COMPOUNDS	2			6		4		
2. DIAZEPAM	2			1				
3. OTHERS								
IV. PSYCHIATRIC CAUSES								
V. UNDIAGNOSE			1			2		
		35	35	25	19	33	3	150



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