

**ABSTRACT**

Identification and management of acute coma or loss of consciousness is a clinical challenge. Most of the time, the patients are brought by unknown people, or with relatives with little details. Assessing the management, depends on the etiology, which has wide varieties. Hence it is mandatory that etiology of the coma, has to be determined, before managing the case. Many causes right from head injury, cerebro vascular accidents, infections of CNS, and metabolic causes result in coma. Wide tracking of causes, by repeated patient questioning to the relatives, at the same time, carrying out the emergency measures to recover the patient's health is imperative. The concept of "Time is Brain" has to be borne in mind, and a work up quick, investigations for the cause of coma, will make a physician's effort highly successful one.

**INTRODUCTION**

A patient brought to your office, in an unconscious state, is a challenge to the Physician, as there are numerous causes for the loss of consciousness, right from common hypoglycemia to a dangerous situation like subarachnoid hemorrhage. The relatives will be highly apprehended, to know the fact regarding recovery, prognosis, even doubts about our capability, approach, and efficiency. Mostly some will be urging to take the patient to a corporate hospital. We have to face lots of problem, apart from looking into the patient's welfare, the other issues also. Mostly, they want an answer immediately, about the survival and prognosis of the patient. A calm, quiet, confident, and efficient approach will win all the issues.

**Definition**

LOC or coma, is a clinical condition of prolonged

unconsciousness, caused by varied etiology, for example severe stroke syndromes, brain tumor going in for conning, head injury, encephalitis, other CNS infections, alcohol or toxic drug intoxications, so on so forth. The conscious level is maintained by the ascending reticular activating system in the brain stem.<sup>1</sup> And it is maintained by volleys of projections to the cortical system, by it's lots of inputs from various sensory, motor projections. The cortical alertness is maintained by such projections from ARAS to the cortex. Its functions are compromised either mechanically, or by toxins and toxic metabolites.

**SYMPTOMS****LOC or Coma, has fairly definite, constant symptoms**

1. The eyes are closed.
2. Non responding pupil, some times asymmetric or pin point and nonresponsive as in subarachnoid hemorrhage.
3. Depressed brain stem reflexes, as evidenced by depressed ocular movements.
4. No movement in the limbs, and occasionally some reflex movements.
5. Ataxic breathing, cold clammy skin, and in metabolic coma, special odors like uremic odor or fruit odor of DKA.
6. Seizures may manifest if the cause is cerebrally oriented, like inflammation or ICH.

**Bed side approach to an unconscious patient**

The following points are necessarily to be examined. First

**Table 1: Evaluation of coma from pseudocomma**

Method of examination		Functional (Pseudo coma)	Organic
1	Plantar response	Withdrawal or flexor	Extensor
2	Pupil	Normal and reactive	Abnormal, non reactive
3	Tone of muscles and limbs	Variable	Either flaccid or spastic consistently
4	Bladder status	Never incontinent	Mostly incontinent
5.	Blood pressure	Mostly normal	Variable
6	Respiration	Hyperventilation	Ataxic or shallow
7	Neck stiffness, classical	variable	Consistent
8	Forcible eye opening by the examiner	Resistance observed	No Resistance
9	Reflex eye movements	Invariably present	May or may not be present depending on the brain stem integrity

**Table 2: Common Etiological issues in COMA**

- **Traumatic brain injuries.**  
Trauma due to road traffic, fall, violence, blasts injuries.
- **Stroke.** Subarachoid Hemorrhage, intracerebral massive bleed, or major vessel occlusion as in carotid massive occlusion, along with compromised brain stem circulation or due or massive brain oedema and coning.
- **Diabetes Mellitus.**  
Hypoglycemia (acute onset coma) and hyperglycemia. (subacute onset coma)
- **Uremia** – Sub acute onset of loss of consciousness, with obvious history.
- **Lack of oxygen.**  
Drowning of post CPR.
- **Infections.**  
Encephalitis and meningitis. Toxemia, Septicemia.
- **Seizures.** Status epilepticus, post ictal coma,
- **Toxins.**  
Carbon monoxide, organophosphorous compounds or lead,
- **Tumors of the brain,**  
Sudden bleeding inside the tumor causing acute swelling, massive brain edema as seen in high malignant tumors, edema and coning of the brain stem.
- **Alcohol** –  
Acute alcoholic intoxication results in coma.
- **Drugs** - Marjuvana, opioids

thing to rule out is the functional causes of the so called LOC (Table 1).

Approach To The Etiology (Table 2). One has to do Police Man's Job, in detecting the etiology.

1. The following questions may give pathway to examination in a case of coma.
  - Had the LOC started suddenly or gradually?
  - Whether it was accompanied with problems in the vision, such as loss of vision, diplopia, vertigo, fainting spells, or numbness of the limbs etc ?
  - Is the patient a known case of diabetes, or patient on dialysis, seizures or old stroke ?
  - Had the patient severe headache prior to the LOC ?
  - Any mental changes observed, prior to LOC, like confusion, frequent falls etc ?
  - Did the affected person use any medications with or without prescription ?

A very detailed history from the accompanying person, if available, regarding the situation of the patient, is mandatory. Invariably, various histories

will be given. We have to analyze the correct pathway, of identifying the cause, is necessary towards the management of the patient. To achieve that, one should examine the patient in depth, to a possible extent, regarding the etiological discoveries, and apply the same towards eliciting the history. Based on the history elicited, further examination is mandatory. It is highly pertinent to remember, that what ever history is given should correlate with our clinical findings, otherwise we may be dragged to different etiology, missing the proper diagnosis.

2. In comatose patient's examination, there will be no resistance for examination but we should not take it to our advantage. A patient with head injury, and coma, may have dislocated cervical injury, and examination for neck stiffness should be extremely careful, as there may not be any resistance. Partial dislocation of spinal discs, will become complete on extreme cervical movements, on examination.
3. Look for more injuries like abdominal injuries, spleen, liver and any other hollow viscous rupture, even before attending to the patient with head injury and coma, as subsequent examination will be targeted only towards head injury, and death of patient may be due to other concomitant causes. It is always better to examine in a routine way from top to toe. Look for any cause that may be responsible for the coma. This procedure is essential to identify the injuries which may become life or limb threatening. Any negligence to identify the other lesions, may be left untreated till the patient recovers from the coma. The common issues are hip dislocations, spinal fractures, and fractures of metacarpals. It is essential that every joint should be put into full range of movements, to assess the integrity of the long bones. Where ever, suspicion results X ray should be done in that area. Injuries in the back, and medical conditions like pneumothorax, should not be missed. Palpation of the peripheral pulse is mandatory.
4. History of injury immediately or in recent past, however trivial it may be, is very important. A subdural may be "quiet" for some time, and suddenly it may enlarge and cone the brain stem. Many of the patients who had mild subdural due to trivial head injury, may land in enlargement of the subdural, if they were on medications with antiplatelet drugs for some other ailment. This may cause coning, which will become a practical problem.
5. Non surgical causes of loss of consciousness are many, which are tabulated (Table 1). Look for evidence for such etiology in case, if you could exclude surgical causes. But never compromise one for other. For example, simple alcoholic coma, may be associated with subdural, and a careful watch for both, is mandatory. Vice versa, a case of head

Table 3: Elements of the scale

Glasgow Coma Scale						
	1	2	3	4	5	6
Eye	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Utters inappropriate words	Confused, disoriented	Oriented, converses normally	N/A
Motor	Makes no movements	Extension to painful stimuli (decerebrate response)	Abnormal flexion to painful stimuli (decorticate response)	Flexion / Withdrawal to painful stimuli	Localizes painful stimuli	Obeys commands

Note that a motor response in any limb is acceptable.<sup>2</sup> The scale is composed of three tests :eye, verbal and motor responses. The three values separately as well as their sum are considered. The lowest possible GCS (the sum) is 3 (deep coma or death), while the highest is 15 (fully awake person). Interpretation of Glasgow Coma Scale; Individual elements as well as the sum of the score are important. Hence, the score is expressed in the form "GCS 9 = E2 V4 M3 at 07:35 minutes; Generally, brain injury is classified as: Severe, with GCS < 8–9; Moderate, GCS 8 or 9–12 (controversial)<sup>4</sup>; Minor, GCS ≥ 13. Ref: "The Glasgow Coma Scale: clinical application in Emergency Departments". Emergency Nurse. 14 (8): 30–5. 2006. doi:10.7748/en2006.12.14.8.30.c4221.

- injury may be accompanied with metabolic or alcoholic coma.
- One of the common causes of LOC is cerebrovascular accident. The coma results mostly in massive intracranial bleed, due to hypertension, aneurysm rupture, leading to acute subarachnoid hemorrhage. Acute intracerebral bleed, if massive, or massive carotid infarcts, do result in LOC.
  - Pressing the nasal ridge at the root in between the eyes, and look for wincing.
  - Eliciting bone pain, by pressuring the shin of tibia and the response of the patient.

All the above methods may give a fair response to the examiner, regarding the level of loss of consciousness, which is very much essential to fix the prognosis.

### EVALUATION OF COMATOSE PATIENT

Assessment of level of consciousness is assessed by Glasgow Coma Scale, especially for comatose patients with head injury. GCS can be used for other causes of coma also.

### GLASGOW COMA SCALE (TABLE 3)<sup>2,3</sup>

- A practical hint to evaluate the prognosis, and severity of the loss of consciousness. Predominantly applied for head injury and CNS causes, and usually not applied for metabolic causes. But for practical purposes one can apply.
- Bed side approach.

Assessing the sensory status and response:

- Level of consciousness, is assessed by the sensory stimuli applied to the patient. By calling the patient by his name, either by the examiner or by a close relative of the patient, and seeing the response.
- Pinching, the skin of the limbs, clenching the Achilles tendon for deep pressure pain response.
- According to some Senior authors, squeezing the nipple is another method, which is not practiced now on ethical issues.
- Assessing the corneal reflex, carefully with a sterile cotton wisp and looking for the response.

### OBJECTIVE CLINICAL SENSORY RESPONSES

- Oculo-cephalic movement reflex. This tests certifies the brain stem integrity. If present give a good prognosis, compared to no response.
- Assure that there is no cervical bone injury, when this examination is performed.
- Pupillary response, to light, is a mandatory examination. A dilated non responding pupil is a poor prognostic sign.
- Cilio spinal reflex can be observed if the papillary response is normal.
- Syringing the ear with sterile water, and trying to elicit nystagmus, assessing the integrity of brain stem, connections with the higher brain centers.
- Plantar response, is highly dubious, and depends on careful observations of the response, and method of elicitation. Bing's response is more reliable than Babinski's. (Bing sign - sharp pressure pain over the dorsum of foot )
- Tone of the muscle, is an index of severity or organicity of the LOC. A variable tone is a sign of non organic LOC, where as, a constant tone, as increased or decreased, could be marked as an organic cause of LOC. A continuous flaccid tone is associated with poor prognosis.

**Table 4: Essential Investigations**

- Blood sugar, blood urea estimation
- Basal blood tests, like ESR, CBC, Hb
- Parasites in the blood, (malaria)
- CT Scan/ Followed by MRI
- Electro encephalogram.
- Electrolytes/anion gap, pH determination
- Renal function tests, urea, creatinine, GFR
- X-rays, urine basic tests
- Acetylsalicylic acid/acetaminophen blood level
- Ethanol/osmolality
- Arterial blood gas/carbon monoxide levels
- Lumbar puncture if necessary, and to be avoided as much as possible

8. Persistent tachy, bradycardia or rather arrhythmias is a sign of organic coma, with poor outcome.

### INVESTIGATIONS (TABLE 4)

Investigations have to be performed in a war foot method. Any delay in the investigations will cause more devastating results in the recovery. Mind "Time is Brain".

### THERAPY

The algorithm for management of coma is given in Figure 1. The first and foremost advise to the management of comatose patient, is application of 50% dextrose intravenous infusion as hypoglycemia is one of the most common cause of coma, and the patient's response is dramatic.<sup>4</sup>

1. In patients with prolonged hypoglycemia, for more than an hour, or in patients with associated motor seizures, recovery response may be delayed.
  - It is observed, that patients with ischemic stroke, if IV infusion of 50% dextrose may enhance the anaerobic glycolysis in infarcted area, to induce production of free radicals', which are harmful to the dying neurons. But it is also observed that most of the ischemic stroke, do not cause comatose state, and coma is uncommon in ischemic stroke, unless it is gross, hemispherical with progressive edema. Hence application of the dextrose need no worry.<sup>5</sup> It is pertinent to observe, that rapid chemstrips may fail to endorse the clinical hypoglycemia in the presence of numerical normoglycemia.<sup>5</sup> By giving dextrose to an already hyperglycemic patient is dangerous in a subtle way, than withholding it from a hypoglycemic patient.

If one is afraid a trial dose of 50 ml of 50% dextrose may be infused for elective result, and recovery from coma.<sup>6</sup>

2. Prevention of hypercapnea: Neuronal death is rapid in the presence of hypercapnea. A comatose patient has mostly has a compromised ventilator drive, which is deleterious. Immediate

supplementation of oxygen is mandatory, by all means. It is preferable, over a period of 15 mts, if there are signs of compromised ventilation and oxygenation, detected by  $PO_2$  less than 70%, one should immediately switch over to the ventilator drive. It is preferable to have 100% oxygen, to obtain saturation of 98% in the blood. This procedure is highly important to save the neurons, and the remaining dying neurons.

3. In most of the patients who are comatose, the retina runs to ischemia, and also if it is associated with acute hypotension, and low  $O_2$  saturation.  $O_2$  supplementation becomes more mandatory to prevent retinal dysfunction and death of neuronal layer. It is pertinent to observe that hypercapnea is far less likely to render irreparable brain damage than hypoxia. More so the retinal damage is more with hypercapnea, especially in  $CO_2$  poisoning, severe macular damage is a usual sequele.
4. Apart from this, infective causes also route to LOC. Meningitis, especially pyogenic meningitis, should be treated with appropriate antibiotic.

One of the rare cause of LOC, of nutritional origin is thiamine deficiency, may be associated with situations where the alcoholism is highly prevalent. In alcoholic coma, even though the LOC is due to toxic causes, an associated thiamine deficiency is invariable associate. Administration of the thiamine is mandatory in such situation.<sup>6</sup>

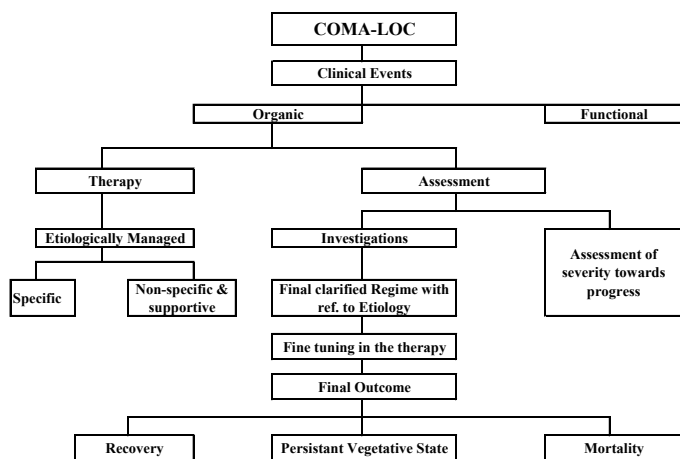
5. Cerebral malaria should be treated with anti parasiticidal drugs, if MP is identified in the peripheral smear. CSF, may be positive for MP, in rare cases apart from the positive peripheral smear.

### Management of cerebral Malaria

Management is multifocal, right from all parameters of management of coma, cardiac monitoring etc, appropriate drugs are as follows. The drug of choice is quinine and artesunate.

Artesunate has a limited shelf life. The dose is  $2.4 \text{ mg kg}^{-1}$  given intravenously, followed by the same dose at 12 and 24 h, then once daily until the patient is able to take artesunate ( $2 \text{ mg/kg}$  per day) by mouth to complete 7 days, then Doxycycline 200 mg or Clindamycin daily by mouth for 7 days. There are many complications of artesunate, which has to be balanced between the recovery and side effects of the drug.

6. Metabolic causes like diabetes mellitus with acute hypoglycemia, requires immediate infusion of 50% glucose, 100 ml followed by 5% dextrose saline. Appropriate correction of acidosis in hyper osmolar ketotic coma, with Ringer solution is mandatory.
7. In renal failure, care should be taken to reduce the BUN, and patient should be dialyzed, appropriately, with the correction of renal acidosis, electrolyte abnormalities.



**Fig. 1: Management of Coma - An Algorithm**

8. Naloxone (0.4 to 2 mg intravenous ) is a tangible remedy in coma due to opioid abuse and intoxication. Even though this situation is common to some local areas, especially in the high northern side, where - in access to the opioids is slightly common.<sup>7</sup>

The dose of naloxone should be titrated to make the patient breathe without reversing the entire opioid load. This will avoid precipitating the acute opioid withdrawal symptoms, more so in absconding cases. It is pertinent to mention the effect of Naloxone wears off before the opioid, and hence opioid withdrawal symptoms may get precipitated.

### COMPLICATIONS

Although many people gradually recover from coma, others enter a vegetative state or even die. Some people who recover from coma may have major or minor disabilities. Complications may develop during coma, including pressure sores, bladder infections, leg vein thrombosis and other problems.

### CONCLUSION

COMA or LOC, is a condition met with by every practitioner and consultant, which is a challenge to decide, treat, and to give a good recovery. Early and swift actions of the consultant, practitioner, and good co operation of the relatives, accompanying persons, to accept the management, give funds for the tests, is a mandatory issue. Since "Time is Brain", an earliest intervention will save the patient's brain and life, to a great extent.

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