

Post-Convulsive Coma

ABSTRACT: A post-convulsive coma case, with a detailed explanation as to how a coma case should be handled taking into account the examinations, investigations, diet, etc.

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A 21 yrs male, a mechanic by profession, was brought to our hospital on 30/9/2006 at about 5am in an unconscious state since 20 minutes. K/C/O Grand Mal Epilepsy since 8 years; he had suddenly stopped anticonvulsants (Eptoin) since 6 months. He had a history of generalized tonic, clonic convulsions before the state of unconsciousness. His elder brother – a witness to the episode, reported that there was frothing and soiling of clothes with urine.

No history of fever, headache or trauma. O/E – Patient was well built, slightly obese young gentleman with dark complexion. Vitals were stable except that BP was 90/64 mm Hg. Profuse sweating on chest, forehead and back-non offensive. Halitosis + CNS- Unconscious, Not responding to verbal commands but response to deep pain ++. Pupils dilated, reacting to light. DTR: Planters equivocal. HS pure. Chest clear.

MANAGEMENT

30/9/06: 5.15am	Hospitalized	IV NS @ 20 drops / min Oral suction done Carbo-veg 0/1, 5drops/10 mins Omit Carbo-veg Bufo 0/1, 5 drops / 10 mins
6.10 am	Stuporous. Unresponsive to verbal commands / superficial pain. Vitals stable. BP 100/70 mm Blood sent for investigations – [S electrolytes, RBS]	Bufo 0/1, 5 drops / 2 hourly
7.30 am	Started moving extremities. Drowsiness++. Vitals stable.	Bufo 0/1, 5 drops/ 3 hourly
9.30 am	Conscious. RBS – 96 mg/dl, Na: 141. K: 4.2. Ca: 9.9 mEq/L.	Omit Bufo
5.10 pm	Pt Awake. Talking. Asked for Tea. Vitals stable.	
1/10/06 8.30 am	Pt slept well. No complaints. Sent for EEG – Generalized Seizure disorder. CT Head – N Detailed History taken	

Patient was the second son. His father was a mechanic and elder brother, aged 23 was an LIC agent. Pampered child. He could complete basic education upto 9th Std only and joined his father as a mechanic. He hates music. Has an inferiority complex and therefore reserved, kept to himself with tendency to masturbate 3 times /day. Stammering+. His first convulsion was at the age of 14 in sleep and was put on Tablet Dilantin Sodium by a local physician. Seizures occurred with a frequency of once in two years and had not occurred for the past 3 years; assuming that he was cured, he stopped medications.

Hot patient, had a voracious appetite and desired sweets+++ . Thirst was normal but sweating was profuse over entire body, offensive and stained clothes yellow. Urine, stool and sleep were normal.

PAST HISTORY: Vaccinated with no reactions. No H/o of skin eruptions, wormy diathesis, tendency to catch cold, etc. Had never been hospitalized.

FAMILY HISTORY: Both GP had asthma (died). Fa: HT and Left Renal calculi and 1 Maternal Uncle had Grand Mal Epilepsy.

1/10/06: *Bufo* 0/2, 1 dose. SL x 7 days. Discharged.

26/12/06: No complaints. *Bufo* 0/3, 1 dose
Has not had seizure, since then .

DISCUSSION

1. This case belongs to Type I ie natural case
2. A patient's level of consciousness can be described in terms of the three A's of consciousness: awake, alert, and aware.

Awake: to be fully roused and not asleep.

Alert means able to pay attention to environment. **Aware** means to have an understanding of oneself and one's environment.

Being oriented is a manifestation of being

aware of one's environment. A person who is awake, alert, and aware may be said to be fully conscious. One who is awake and alert but has lost awareness is severely demented. Someone who is merely awake and not alert or aware is delirious. A person who is not awake, alert, or aware either is asleep (is arousable) or is somewhere along the spectrum of coma.

3. In the setting of coma management, there are only three possible meanings. First, the term unresponsive may be applied to a patient who is actually fully conscious but is unable or unwilling to respond verbally. For example, an aphasic patient may be awake, alert and aware, yet unable to speak. Second, the term unresponsive may be applied to a patient who has a waxing-and-waning level of wakefulness ie delirium. Third, the term unresponsive may be applied to a patient who truly is comatose.
4. Patients who are not aware, alert or even awake (except for those who are asleep) fall somewhere along the spectrum of coma. This spectrum reflects varying levels of impairment of the response to stimuli. On the high end of the spectrum, is the patient who can be aroused (albeit perhaps only temporarily) by the sound of a voice. Somewhat farther down is the patient who can be temporarily aroused by a painful stimulus. Farther down still is the patient who cannot be aroused by a painful stimulus but at least exhibits some motor response to it. On the low end of the spectrum is the patient who has no motor response to a painful stimulus; this is the generally accepted definition of coma.
5. The terms lethargy, stupor, and obtundation, though widely used, have

come to signify different things to different people. It is perhaps advisable to employ clear clinical patient descriptions rather than rely on these particular terms.

6. A seizure disorder can cause coma in two different ways. First, the coma may be the initial manifestation of the immediate postictal state after a generalized seizure. In such cases, the coma resolves early. The duration of the postictal coma may be directly correlated with patient's age and inversely correlated with baseline functional status. Second, coma may develop when multiple generalized seizures occur in succession and there is not enough time between seizures to allow patients to recover. The resulting state is status epilepticus.
7. In Coma the history is usually straightforward, the neurologic examination is focused and the differential diagnosis is limited. A good neurologic examination, in combination with a thoughtful battery of tests, will invariably achieve the correct diagnosis. Once initial stabilization of the patient has been achieved, management of coma is determined by the specific causative conditions present.
8. The first step stabilize the patient by addressing the ABCs of resuscitation (Airway, Breathing, and Circulation). The patient's airway must be cleared of all foreign material, and the patency must be verified. The spontaneous rate and rhythm of respiration should be noted. Endotracheal intubation is indicated in patients who are dyspneic, hypoventilating, or vomiting uncontrollably. If a patient is to be intubated, however, it is extremely helpful first to obtain a focused neurologic examination (which can be done in 60 seconds) because the information that can be gained from this examination will be lost when the patient is paralyzed for the intubation. Hyperventilation with a bag and mask and 100% oxygen should be performed before intubation to ensure adequate oxygenation during the procedure. If there is any possibility of cervical spine injury, intubation should be delayed, if possible, until fracture can be ruled out radiographically.
9. Circulation must be vigorously supported, especially in the setting of brain injury or hypoxia. This is accomplished by inserting a large-bore IV or central venous catheter and infusing isotonic fluids or volume expanders. The use of solutions containing free water (eg 5% dextrose in water) should be avoided, especially in the setting of brain injury or stroke, because such solutions have the potential to increase cerebral edema.
10. As the patient is being stabilized, evaluation should be initiated. A witness or someone else capable of providing a history should be sought. The differential diagnosis of coma is wide but limited. Clues from the history, the focused neurologic examination, and the general physical examination are often helpful and sometimes diagnostic.
11. Optimally, if the coma was of sudden onset, a witness was present who can describe what occurred. A history of trauma, drug use, medications, recent febrile illness, heart disease, organ failure, or seizures often rapidly leads to the correct diagnosis.
12. A complete and exhaustive neurologic examination is totally unnecessary. For the purposes of evaluating coma, a focused neurologic examination is preferable, being both valuable and rapid (~ 60 seconds). This examination addresses a number of

- key findings (1) spontaneous movements, (2) pupillary response, (3) ocular motility and (4) motor response. A reflex hammer is not needed.
13. Spontaneous movements should be observed over a period of 10 to 20 seconds.
 14. Pupillary responses are important in that their presence or absence distinguishes structural from metabolic coma. The pupils are generally resistant to metabolic insult. They also indicate the integrity of the brain stem. The pathways that control ocular motility also lie adjacent to the Reticular Activating System. Roving eye movements usually indicate that the brain stem is intact and that a metabolic problem is affecting the brain. Minimal or absent eye movement in conjunction with reactive pupils also signifies a metabolic process.
 15. Blood pressure, heart rate, and cardiac rhythm are keys to the diagnosis of the various cardiovascular and hemodynamic causes of coma.
 16. As emergency management is being provided, laboratory studies should be obtained, including serum electrolytes, calcium, magnesium, phosphorus, blood urea nitrogen (BUN), and creatinine levels, as well as liver function tests. A complete blood count (CBC), a urinalysis and a urine toxicity screen should also be obtained. A lumbar puncture should be performed only if meningitis is suspected on the basis of clinical examination.
 17. Diet: A high-fat, low-protein, low-carbohydrate (ketogenic) diet may help control the frequency of seizures. Ask the patient to avoid alcohol and caffeine. Taurine, an amino acid that has been shown in studies to inhibit seizures. Folic acid is grossly depleted during seizures. Magnesium is essential for normal muscle and nervous system function. ○

NJH's Comprehensive Calendar of the year 2006-2007

Date	Event	Venue
16 th , 17 th , 18 th February 2007	<p>" HORIZON 2007" – Fourth Annual National Post – Graduate Workshop and Seminar TOPIC: Study of Homoeopathic Repertory and its Clinical Applications SPEAKER: Dr. Sohan Singh , Dr. Parinaz Humranwala CONTACT : Dr Sarita Shinde 9869304147, 27565439 FEES: Rs 700 till 1st Feb 2007</p>	<p>Yerala Medical Trust Homoeopathic Medical College and Hospital, PG Institute, Kharghar, Navi Mumbai</p>

The trouble with being punctual is that nobody's there to appreciate it.

- Franklin P. Jones

We must believe in luck. For how else can we explain the success of those we don't like?

- Jean Cocturan