



www.emergencymedicinescases.com

## **Episode 13 – Killer Coma Cases**

*Prepared by Dr. Lucas Chartier*

### **General approach to the comatose patient in ED**

1. Structural (i.e. reticular activating system dysfunction in brainstem) vs. metabolic (bilateral cerebral hemisphere insult)
2. An approach to coma based on priorities: 3 causes of coma that we need to think about for every patient who presents with altered LOC and treat immediately are **hypoglycemia, hypoxia and opiate overdose** as they have simple rapid treatments
3. **“AEIOU TIPS”** – **A**lcohol (incl. toxic alcohols), **E**lectrolytes (incl. endocrinopathies), **I**nsulin (i.e. glucose), **O**verdose (or withdrawal from drug), **U**remia, **T**rauma (eg, intracranial bleeds, incl. spontaneous), **I**nfection (eg, sepsis, meningitis), **P**sychiatric, **S**eizure (incl. non-convulsive status epilepticus)

Neurological exam in comatose patient should include GCS with focus on motor exam, eyes – pupils, reactivity, deviation and movement, fundi (consider bedside U/S for detection of papilledema; complete vitals with focus on RR (high RR may be sign of acidosis), signs of meningismus (helpful only if present), upper motor neuron signs (Babinski, Hoffman’s) and ankle clonus, and brainstem reflexes (doll’s eyes, cold caloric)

### **Non-convulsive status epilepticus (NCSE)**

- Defined as convulsions for at least 30min, with change in cognition or level of consciousness (LOC); probably less damaging to neurons than convulsive status epilepticus, although they may progress to it, and patients at high risk of aspiration due to failure of protecting their airway
- When to consider NCSE: any patient with altered mental status, especially those patients with known seizure disorders, a history of recent seizures, or patients who are comatose with no readily identifiable structural, metabolic, or traumatic causes; it sometimes follows generalized convulsive status epilepticus (up to 25%), so if the patient’s not back to baseline after an hour or 2 after a prolonged seizure, you should think about NCSE
- Often presents as altered LOC plus subtle automatisms such as blinking, twitching, grabbing at things, facial grimacing or clenching of the teeth, or simply change in behavior or psychosis – perform a thorough neuro exam before intubation and paralysis!
- EEG is diagnostic if available (or once in the ICU), or a diagnostic and therapeutic trial of benzodiazepine may be attempted if EEG is not immediately available

### **Six pearls for ‘found down’ patients**

1. Don’t assume it’s (only) ethanol
2. Thorough examination, full neurologic exam
3. Consider C-spine injury in unclear mechanism
4. Aggressive airway management, avoid sux
5. Don’t delay advanced neuro-imaging (CT/MR-A)
6. Thorough approach to toxidromes (pupils, skin)

### Pearls from our experts

- Some **causes of seizures that require additional specific treatment besides supportive therapy and anti-seizure meds**: carbon monoxide poisoning (clue: multiple people from the same location are involved, Tx 100% O<sub>2</sub>), INH toxicity (Tx: pyridoxine), and pre/eclampsia in pregnant and post-partum patients (Tx: magnesium and labetalol); Do not use **dilantin** in alcoholics or toxicologic cases as it is pro-arrhythmic
- How to confirm **pseudo-coma** (i.e. psychiatric): patients will roll their eyes up when opened to avoid eye contact with the provider; lift their arm above their face and let it drop, which the conscious patient will redirect to avoid hitting themselves in the face; use cold caloric test as a last resort
- Suspect **pseudo-seizures** when 'gross theatrics' are involved, there is no post-ictal state, or the response to benzodiazepine is physiologically too quick (i.e. wake up within seconds)
- **Naloxone**: Usual starting dose of naloxone is 0.4mg, then increase to 1mg, then 2mg until desired effect; patients in coma often do not respond to doses <2mg; however, there may be another toxin on board, and so reversing them fully may bring out undesired effects of the other toxin; also, you can precipitate narcotic withdrawal in chronic narcotic users causing severe myalgias, diarrhea and agitation; avoid repeat boluses of naloxone since its effect wears off in 30-60mins – rather, consider starting an infusion at 2/3<sup>rd</sup> the converting dose as the hourly infusion rate

### Differential diagnosis of altered LOC and elevated temperature

Infection, sympathomimetic (incl. amphetamines) and anticholinergic toxidromes, neuroleptic malignant syndrome (NMS), serotonin syndrome, malignant hyperthermia, endocrinopathies (eg, thyroid storm), withdrawal syndromes (eg: GHB, alcohol) heat exhaustion/stroke (esp. in summer and in psychiatric patients as antipsychotics effect thermoregulation)

- Altered LOC and *hypothermia* should prompt a differential of infection, environmental exposure, hypothyroidism or hypoadrenalism

### Meningo-encephalitis

- Although most don't present classically (esp. immunocompromised & elderly patients), consider the diagnosis of bacterial meningitis in the face of fever, nuchal rigidity, headache or altered LOC (incl. lethargy or behaviour and personality changes as per family)
- **Herpes encephalitis** often presents with pronounced alteration in LOC, associated with psychiatric symptoms, seizures or dysarthria; LP may appear traumatic due to hemorrhage in the temporal lobes
- **Physical exam**: Jolt accentuation sign (turning head rapidly left-right several times) has the highest PPV of any maneuver for meningitis, while Brudzinski and Kernig signs have very poor sensitivity
- Textbook **indications for CT scan before LP** to identify patients with raised ICP who theoretically might herniate from LP are: focal neurological sign, papilledema, seizure, immunocompromised, malignancy; however this is controversial and some experts believe that herniation following LP has never been proven to be caused by the LP and that a normal scan does not exclude raised ICP
- **When to start antibiotics**: after an immediately performed LP, or if doing CT first, do pan-cultures (which often will yield the offending bacteria) and start antibiotics *before* the LP, while doing

everything you can to minimize the time to LP; note - PCR on CSF is very good at identifying organism despite antibiotics

- **Textbook CSF findings** (interpret with caution!):
  - Bacterial meningitis: opening pressure >300 (in recumbent position), WBC >1,000, neutrophils >80%, protein >200
  - Viral meningitis: WBC <300, neutrophils <20%, normal protein and glucose levels
  - HSV encephalitis: viral findings as above + RBCs (due to temporal lobe hemorrhage)
- EMC experts recommend using **dexamethasone** 10mg IV within 15min of 1<sup>st</sup> antibiotic dose when bacterial meningitis is suspected esp. with a GCS of <12, as it may lead to decreased mortality, decreased hearing loss, improved cognitive outcome, no adverse events demonstrated in meta-analyses and as recommended by Cochrane review (NNT=9)
- **Antibiotic regimen:**
  - Ceftriaxone 2g IV for all, plus vancomycin 2g IV if MRSA is suspected, plus ampicillin 2g IV if Listeria is suspected (immunocompromised patients, incl. AIDS and alcoholism), plus acyclovir 1g IV if herpes encephalitis is suspected (altered, seizure, neuropsych symptoms, RBCs in CSF)
  - Post-exposure prophylaxis, only for those in close contact with affected patients (eg, sharing saliva, bodily fluids), is Ciprofloxacin 500mg PO x1 dose

### **Methanol Poisoning**

**When to suspect toxic alcohol ingestion** in the face of an intoxicated patient: **Kussmaul breathing** (i.e. metabolic acidosis), abdominal pain or ocular complaints, if the patient appears drunk with low blood levels of ethanol, or isn't clinically improving

**Causes of increased osmolar gap** in a patient with coma: "**ME DIE**": Methanol, Ethylene Glycol, Diuretics (osmotic ones like mannitol), Isopropyl Alcohol ('rubbing alcohol'), Ethanol; also consider propylene glycol, glycerol and ketones

Methanol (in windshield washer, solvents and fuels) kills retinal and optic nerve cells, leading to classic 'snowstorm' vision, flashes, and eventually blindness if not treated; lethal dose is 1g/kg (as little as 30cc of methanol or 100c of windshield washer fluid)

Osmolar (usually between -2 and +6) and anion (usually <12) gaps:

- As toxic alcohols are ingested, the compounds are only osmolally active at first (i.e. high osmolar gap, no anion gap), until they are degraded in their metabolites which create the anion-gap acidosis (i.e. both osmolar and anion gap); late in the course, when all the parent compounds have been degraded, there is only an anion gap with no osmolar gap; therefore *early presenters may have normal AG and late presenters may have normal Osmolar Gap*
- **Treatment** should be started **empirically** before toxicologic confirmation occurs with **fomepizole** (expensive) or ethanol (IV or oral), bicarb for pH<7.3 (add 150mEq of sodium bicarb to 1L of D5W and infuse at 150-200cL/h), and dialysis in severe metabolic acidosis, ocular findings or >30cc of methanol ingestion (get your nephrologist involved early!)

### **Serotonin syndrome (SS)**

Often rapid onset (vs. more prolonged in NMS) as a result of overdose or, most commonly, combination of prescription (eg, SSRI, MAOIs) and non-prescription drugs (recreational such as 'ecstasy'/methamphetamine, or OTC)

Mnemonic "**SHIVERS**": **S**hivering, **H**yper-reflexia (myoclonus vs. "lead-pipe" rigidity in NMS), **I**ncreased temperature, **V**ital signs instability (high BP, HR, RR), **E**ncephalopathy (or any altered LOC), **R**estlessness, **S**weating

Clonus in the setting of 'Ecstasy'/methamphetamine use should prompt the diagnosis of SS!

'Ecstasy' is now the most common trigger of SS in Ontario, Canada

Also beware of **dilutional hyponatremia** in people participating in 'Rave Parties', where they drink LOTS of water

**Drugs associated with SS:** anti-depressants – SSRIs, SNRIs, MAOIs, TCAs; pain medications – Demerol, fentanyl, tramadol; headache medications – maxeran, triptans; weight loss drugs; drugs of abuse – amphetamines; as well as dextromethorphan (in Tylenol Cold©), linezolid, ondansetron and granisetron

**Treatment:** mainly supportive with activated charcoal as necessary, external and/or internal cooling, benzodiazepines, and consider olanzapine, chlorpromazine (DO NOT use if NMS is considered), and the antidote **cyproheptadine** in conjunction with a toxicology consultation (do not give both charcoal and cyproheptadine as it is an oral medication!)