**Encephalopathy**

**Confusion** – inability to maintain a coherent stream of thought or action

**Delirium** – confusional state with superimposed hyperactivity of sympathetic system causing tremor, tachycardia, diaphoresis, mydriasis

**Acute Toxic-metabolic Encephalopathy (TME**) – encompasses delirium and acute confusional state. Causes acute condition of global dysfunction in the absence of primary structural brain disease.

**Types of Etiologies:**

1. Septic encephalopathy
   1. Most common cause of TEM
   2. Multifactorial pathophys – microcirculatory abnormalities, altered blood-brain barrier permeability, inflammatory cytokines, reduction in monoamine neurotransmitters
   3. Lumbar puncture to exclude meningitis can be normal or show excess protein concentration
   4. Tx: control underlying infection
2. Hepatic encephalopathy (HE)
   1. Two types: Acute HE w/ cerebral edema (seen in acute onset of liver failure), Chronic HE due to chronic liver disease and protosystemic shunting of blood.
   2. Pathophys – increased ammonia, false neurotransmitters, endogenous Benzo-like substances, abnormal fatty acid metabolism, free radical damage, cerebral edema
   3. Dx made based on clinical ground. Normal arterial NH3 level does not exclude HE
   4. Get CT head to r/o ICH in the setting of coagulopathy
   5. Tx begins w/ correction of coagulation parameters, electrolyte abnormalities, volume depletion, hypoxemia, identification and tx of potential
3. Uremic encephalopathy
   1. Seen in advanced renal failure. Could also occur w/ dialysis disequilibrium syndrome
   2. Early signs include lethargy, irritability, disorientation, hallucinations, rambling speech.
   3. Acute uremic enceph reverses w/ dialysis, although a lag time of 1-2 days required for mental status to clear.
4. Hyponatremia
   1. Most commonly caused by SIADH or decrease in effective circulating blood volume
   2. Hyponatremia developing in less than 12-24 hours and Na <120 associated w/ more severe symptoms
   3. s/s – confusion, disorientation, agitation, delirium, lethargy, muscle cramps
5. Hypernatremia
   1. Commonly due to insensible water losses, decreased thirst or access to water, infusion of large volume saline/bicarb, DI
   2. Tx: treat underlying cause. Rate of correction of 1-2 mEq/L/hr recommended.
6. Hypoglycemia
   1. Usually due to use of insulin or hypoglycemia agents, Etoh, liver disease
   2. Presents w/ symptoms of increased epinephrine release (ex: tremor diaphoresis), followed by generalized seizures, coma, focal deficits
   3. Tx: bolus of 25-50 gras of dextrose IV. This can be followed by dextrose infusion w/ hourly BG measurement until resolution.
7. Hyperosmolar hyperglycemia (HHS) and DKA
   1. Progressive obtundation and ultimately coma likely 2/2 combination of metabolic acidosis and hyperosmolarity
   2. Tx underlying disease process
8. Wernicke’s encephalopathy
   1. 2/2 thiamine deficiency
   2. Occurs in alcoholics and nonalcoholic subjects. Patients who are fasting, receiving parenteral nutrition, recovering from GI surgery, being fed after period of starvation, undergoing HD, suffering from cancer are more susceptible
   3. s/s: triad of confusion, ataxia, ophthalmoplegia. Ocular signs are the hallmark of the disease (horizontal nystagmus, complete ophthalmoplegia, papillary abnormalities)
   4. tx: IV thiamine.
9. Hypoxic-ischemic encephalopathy
   1. Etio: cardiac arrest w/ prolonged resuscitation. Prolonged hypotension, hypoxemia
   2. In comatose patients, presence of papillary light reflex, flexor/extensor responses, conjugate or orienting eye movements on initial exam may be used to identify patients w/ better prognosis
10. Post-transplantation encephalopathy
    1. Etio: due to underlying conditions, operative procedures, immunosuppressive medications, cranial radiation, opportunistic infections.

**Diagnosis**

* All of the above are diagnosis of exclusion. Always r/o Alcohol withdrawal, meningitis, encephalitis, brain tumors, non-convulsive seizures, central venous thrombophlebitis, bacterial endocarditis, fat embolism, basilar artery thrombosis, traumatic brain injury, and right hemisphere stroke can present with an acute confusional state

**Lab studies/Imaging**

* CBC, coag studies, CMP, Mg, Phos, glucose, NH3, serum osmolality, ABG, tox screen, Thyroid panel, vit B12, serum cortisol
* CT or MRI head
* EEG
* Neuro consult