Common Neurologic Emergency Cases

Vicki L. Campbell, DVM, DACVA, DACVECC
Assistant Professor of Emergency and Critical Care
CSU Veterinary Medical Center

Neurologic Emergencies

- Commonly present on emergency
- One of the four major ER systems
- The ER doctor needs to feel comfortable diagnosing, stabilizing, treating, and prognosticating neurologic emergencies

Objectives

- Head trauma
- Select neurologic toxins

Heat Trauma Objectives

- Pathophysiology
- Approach to treatment
- Future directions for treatment
- Prognosis

Head Trauma Case

Triage Examination

- Young adult DSH, MC
- MM: pale pink
- CRT: 3 sec
- HR: 220 bpm w/ weak pulses
- RR: 40 bpm with normal to slightly increased breath sounds all fields
- Abdominal palpation – WNL

Triage Neurological Examination

- Depressed mentation
  - Capable of responding, but response is decreased
- Non-ambulatory
  - Recumbent with intermittent extensor rigidity

Triage Neurological Examination

- Cranial nerves:
  - Anisocoria (L < R)
  - Direct and indirect PLR's present, but slow
  - Difficult to assess menace but appears to be visual bilaterally
  - All other CN's WNL

Other Obvious Trauma

- Palpable skull fractures
- Mandibular symphyseal fracture

What are the life-threatening problems?

- Traumatic shock
- Head trauma

Pathophysiology: Primary Brain Injury

- Occurs at the time of trauma
- Direct injury to parenchyma and blood vessels
- Damage is already done
- Time will tell reversibility

Pathophysiology: Secondary Brain Injury

- Results from secondary intracranial processes
  - Inflammation
  - Edema
  - Vasculitis
  - Increased intracranial pressure

Specific Causes of Secondary Brain Injury

- Hypoxemia and hypovolemia contribute to ATP depletion in the brain
- Release of excitatory neurotransmitters (esp. glutamate) increase cerebral metabolic oxygen demand
- Oxygen free radical formation from ischemia damages brain cells

Goals of Head Trauma Treatment

- Target reduction of secondary brain injury!
- Restore oxygen delivery to the brain
- Decrease cerebral edema and intracranial pressure

Case: Immediate Diagnostics and Treatment

- **Blood pressure:**
  86/40 (60)
- **Blood glucose:**
  312 mg/dL
- **Flow-by oxygen**
- **IV fluids:**
  5 ml/kg IV Hetastarch over 15 min

Maximizing Oxygen Delivery

- Administer supplemental oxygen as soon as possible
  - Maximizes oxygen in blood (and hopefully the brain)
  - Can reduce ICP by decreasing cerebral vasodilation
- Maximize cerebral perfusion by treating shock and hypotension

Understanding Cerebral Perfusion

- CPP = MAP – ICP
- Treat hypotension before targeting intracranial pressure
- Normalizing BP allows cerebral perfusion to be maintained and helps decrease ICP by reducing cerebral vasodilation
  - Target a MAP of 80 mmHg

Fluid Resuscitation in Head Trauma

- Need fluids that quickly raise blood pressure to normal, but do not contribute to cerebral edema
- Concept is to reduce the amount of crystalloids that are given
- The addition of a colloid, regardless of how much crystalloid has been given has been shown to be beneficial in head trauma

Fluid Choices in Head Trauma

- Hetastarch alone
  - 5-10 mls/kg IV over 10-15 min
- Hetastarch/hypertonic saline
  - 2/3 hetastarch w/ 1/3 7% hypertonic saline combination
  - 3-5 mls/kg IV over 10-15 min
- Dextrans/hypertonic saline
- Avoid hypertonic saline alone

Fluid Choices in Head Trauma

- Judicious use of crystalloids is okay
  - Titrate to effect
  - ¼ shock dose over 10-15 minutes
    - 22 ml/kg in the dog
    - 15 ml/kg in the cat
- Lactated ringers (LRS) is a great crystalloid choice in neonates

Controversies in Fluid Choices

- Concern about colloids and hypertonic saline with massive intracranial bleeds
- Do crystalloids truly contribute to cerebral edema?

Reducing Intracranial Pressure

- The cranial vault is a confined space and does not have much room to expand without dangerously increasing intracranial pressure
  - Blood vessels
  - Parenchyma
  - CSF

Reducing Intracranial Pressure

- Reduce excessive cerebral vasodilation
  - Restore blood pressure to normal
  - Prevent hypoxemia to brain
  - Prevent hypoventilation
    - Avoid respiratory depressant drugs (general anesthetics, heavy sedation, pure opioids?)

Reducing Intracranial Pressure

- Avoid excessive blood flow to brain
  - Raise head 15 to 30 degrees (make sure no evidence of neck injury first)
  - Avoid putting pressure on jugular veins, which will decrease venous return from the head
- Avoid temporary raises in ICP
  - Prevent coughing (especially if needing to intubate)

Reducing Intracranial Pressure

- Decrease cerebral edema
  - Mannitol administration
    - Osmotic diuretic
    - Free-radical scavenger
    - Decreases blood viscosity, which may improve microperfusion
  - Controversial use of mannitol
    - Intracranial hemorrhage
    - Rebound effect

Mannitol

- Despite controversy, the majority of head trauma animals seem to respond to mannitol administration
- 0.5 -1 g/kg over 20-30 minutes
- Can start with 0.5 g/kg and if there is improvement, repeat
- If decline with treatment, discontinue
- Do not exceed 3 g/kg in 24 hours

Case: Reducing ICP

- 15-30 degree head elevation
- Mannitol
  - 0.5 g/kg IV over 20-30 minutes
  - Observe for changes in mentation
  - Consider repeating dose if improvement or no change
- Maintain normal ventilation
  - Check arterial or venous CO_2

Other Treatments

- Lasix therapy
  - May enhance diuretic effect with mannitol and prevent rebound effect
  - Dose is usually 1-2 mg/kg IV
  - Controversy when to give in relation to mannitol (before or after)
  - Be cautious of causing volume depletion and return of hypotension
  - Used more commonly in brain tumor cases as opposed to head trauma cases

The Steroid Controversy

- Currently out of favor in ER/CC and neurology specialties
- No improvement in outcome shown
- Worsened outcome in some human studies
- May worsen hyperglycemia
- Has only been shown to be useful PRIOR to head trauma

Hyperglycemia

- In people the degree of hyperglycemia on presentation is correlated to the severity of head trauma and the outcome
- In dogs and cats, it is correlated to the severity of head trauma, but not outcome
- May be due to severity of catecholamine release

Hyperglycemia

- May be detrimental if brain hypoxemia
  - Brain lactate levels may increase
  - Cellular acidosis
  - Cell death
- Unknown if we should control hyperglycemia associated with head trauma

Future Considerations

- Control glutamate levels in brain
  - NMDA receptor antagonism?
    - Magnesium
    - +/- low dose ketamine
- Decrease calcium levels in brain
  - Calcium channel blockers?
- No studies to date on these theoretical treatments

Clues to Severity of Injury

- Progression of pupil size
  - Pinpoint
  - Midrange
  - Dilated
  - Fixed and dilated
- Initial blood glucose

Prognosis: Assessing Neurologic Status

- Ensure the animal is out of shock
- Give the animal time!
  - Minimum 24-48 hours
  - If comatose > 48 hours, prognosis is poor to grave
  - Some animals need weeks to months to recover
- Recovery should be considered successful if the animal can eat, drink, eliminate, sleep, and interact positively

Prognosis

- Many animals will continue to eat and drink despite being in an obtunded state
- Head trauma patients need a lot of energy to repair themselves
- Offer food/water early, as long as gag reflex is intact and the animal can be in a sternal position

Prognosis: Modified Glasgow Coma Scale

- Looks at 3 categories:
  - Motor activity
  - Brain stem reflexes
  - Level of consciousness
- Scoring:
  - 3-8 is considered grave
  - 9-14 is considered guarded
  - 15-18 is considered good

Prognosis In This Case

- Motor activity
  - Recumbent, intermittent extensor rigidity (4)
- Brain stem reflexes
  - Slow PLR's (5)
- Level of consciousness
  - Depression, but capable of responding (5)
- Total score = 14 = guarded prognosis
- Hyperglycemia is mild (for a cat)

Continued Supportive Care

- Oxygen
- Raise head 30°
- Fluid support
- Mannitol
- Nutrition
- Analgesics

Head Trauma Summary

- Treat shock and hypotension
- Reduce intracranial pressure
- Consider colloid/hypertonic saline fluid therapy for resuscitation
- Oxygen therapy and head elevation should be instituted on every case
- Consider mannitol administration
- Give 24-48 hours before determining ultimate prognosis

Neurologic Toxin Case

- 2 year FS Lab
- Presenting complaint:
  - Acute onset of ataxia and “drunk” appearance
  - Polydypsia
  - Vomiting

A Tale of Two Toxins

- Ethylene glycol toxicosis classically causes ataxia, a drunken appearance, polydypsia, and vomiting in the early stages
- Early intervention is the key to successful treatment
- Untreated ethylene glycol toxicosis will lead to irreversible and fatal kidney failure, therefore it is imperative that the veterinarian feels comfortable ruling it in or out

A Tale of Two Toxins

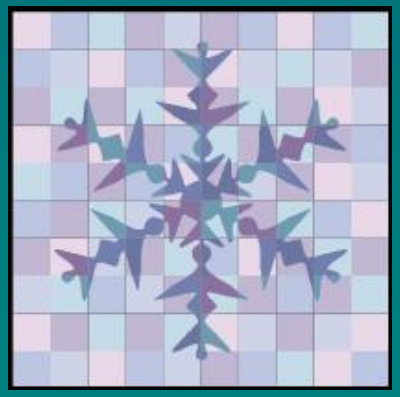
- Marijuana toxicosis can also cause an ataxia, a drunken appearance, polydypsia, and vomiting
- The course of illness is usually self-limiting and non-life threatening
- Treatment is usually supportive
- Owners frequently do not want to admit to the possibility of exposure
- Therefore, we must rule ethylene glycol to the best of our ability!

Other Similar Presenting Toxins

- Barbiturates
- Tricyclic anti-depressants
- Methanol
- Alcohol

Diagnostic Clues

- A very thorough history
- Questioning and re-questioning the owner
- Likelihood that the animal roams
- Time of year may give some clues



Ethylene Glycol Diagnostic Clues: Bloodwork/Venous Blood Gas

- Elevated anion gap
  - $(Na + K) – (Cl + HCO_3)$
  - Indicates presence of an unmeasured anion
  - R/O lactate, ketones, salicylates, phosphorous, ethylene glycol
- Metabolic acidosis
- Ionized hypocalcemia

Ethylene Glycol Diagnostic Clues: Bloodwork Continued

- Elevated osmolal gap (> 10 mOsm/L)
  - A difference in calculated osmolality compared to measured
  - Calculated takes into consideration sodium, glucose, and BUN
  - If the measured is much higher than the calculated, then there is presence of an unmeasured hyperosmolar substance

Ethylene Glycol Diagnostic Clues: Urinalysis

- May indicate calcium oxalate crystalluria
- Frequently not present until 6-12 hours after ingestion
- Isosthenuria and azotemia with calcium oxalate crystalluria is a grave prognosis if antifreeze is suspected

Ethylene Glycol Diagnostic Clues: Ethylene Glycol Blood Test

- A cumbersome, but useful test if ethylene glycol is still circulating in the blood
- Newer, easier tests are becoming available
- Use caution in cats and small dogs, as the test is not sensitive enough to detect small (albeit lethal) amounts in the blood
- Propylene glycol and sorbital cause false positive results

Feeling Good About Ruling Out Ethylene Glycol Toxicosis

- History makes ingestion unlikely
- Normal anion gap
- No metabolic acidosis
- Normal ionized calcium
- Normal osmolal gap
- No calcium oxalate crystalluria
- Negative ethylene glycol test

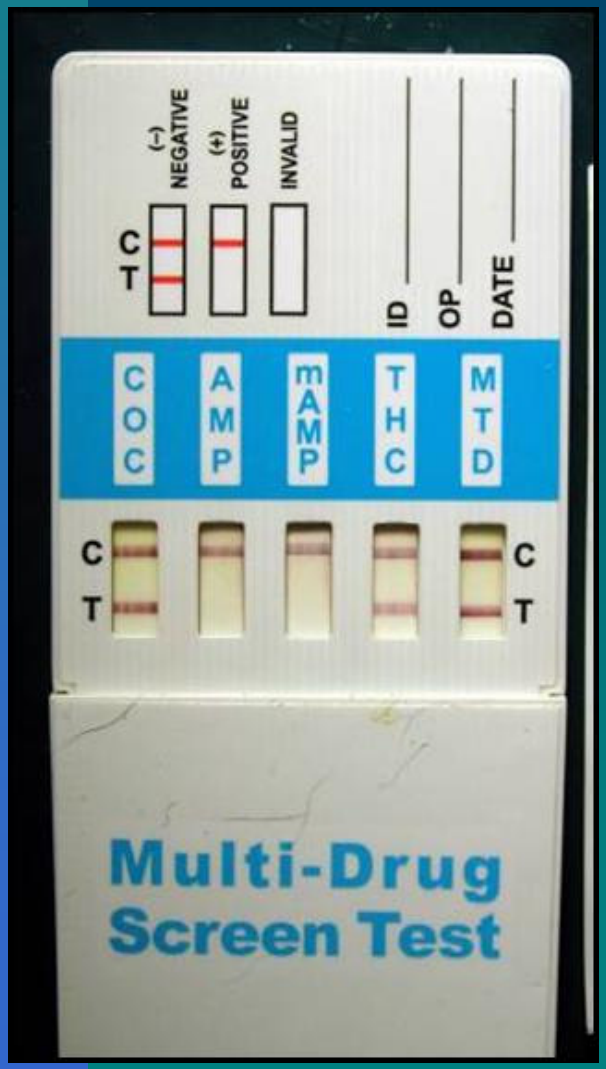
Marijuana Diagnostic Clues

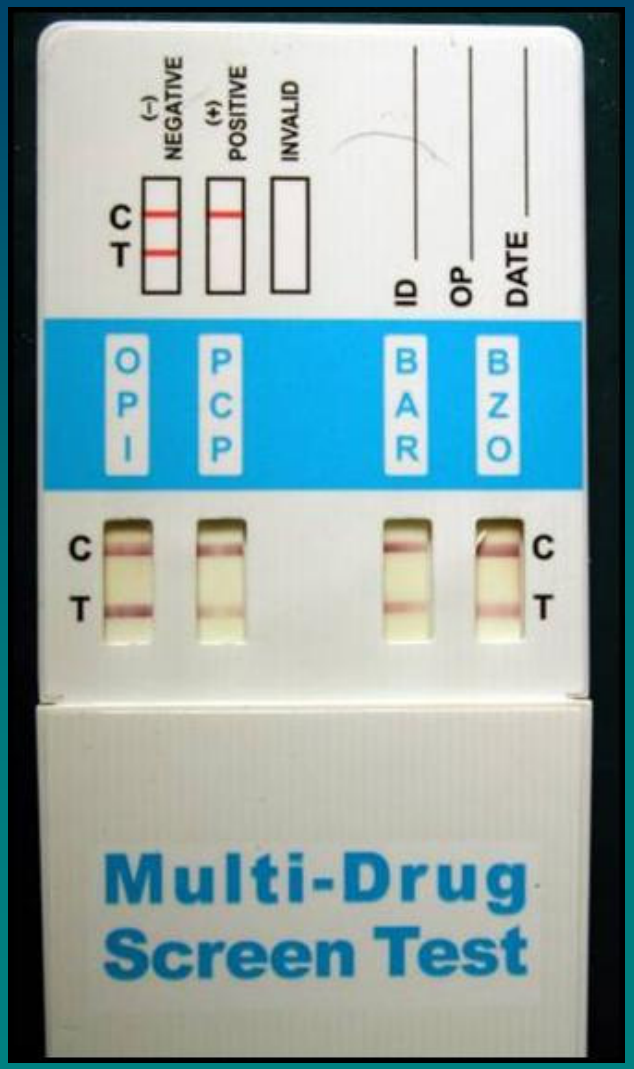
- Trying to get the info out of the owner is sometimes a touchy subject
- Identify substance in vomitus
- Consider using human urine drug test kits

Human Urine Drug Tests

- Medimpex United Inc.
  - Multidrug 9 panel test kit
  - Cost is about $20 to client
- Not validated in dog
  - May cross-react with other drugs
  - Unknown how sensitive the test is or how long you need to wait to test
- Should get owners permission

Urine Drug Test Results





Summary

- Ruling out ethylene glycol toxicosis is imperative
  - Early detection is life-saving
  - Missing the diagnosis is deadly
- Understanding the tests that should be run is necessary for proper diagnosis
- The availability of illicit urine drug testing has allowed more accurate diagnosis of suspected drug cases

Questions?