SH Ch 61-75 WS (10/9/19)

Wednesday, October 02, 2019 2:00 PM

A 3y F/S Labrador is presented for tachypnea, weakness, and scleral hemorrhage. Five days previously she is known to have ingested brodifacoum-based rodenticide for which no intervention was pursued, until now. Physical examination and initial laboratory findings are as follows:

T 98.4 P 162 R 64 mm pale CRT <1s Weight 20kg BP 100/40 (60)
Bounding pulses, muffled heart and lung sounds
Breathing with paradoxical abdominal wall motion and increased inspiratory effort
Thoracic wall pain on palpation, generalized weakness
The patient has a syncopal event during the physical examination

PCV 12% TS 4.2 Na 151 K 3.4 Cl 121 iCa 0.82
pH 7.21 pCO_2 34 HCO_3 14 SPO2 at 96% on room air
Glu 154 Lac 3.2 Crea 1.4 -100% with O2 supplementation

1. Provide a ranked problem list and the interventions you wish to provide in the next 10 minutes, and next 60 minutes

Difference between frozen and fresh frozen:
-FFP: clotting factors, anti-thrombin,
-FP: LOOK UP DIFFERENCES FOR NEXT WEEK in regards to treating rodenticide patients
-ALSO LOOK UP ANTI-COAGULANTS USED IN DONOR BLOOD

-Vitamin K dependent factors: 2, 7, 9, 10

Problem	10 Mins	60 mins
Brodifacoum toxicity w/scleral hemorrhage		(1) PT/PTT, CBC w/diff, Chemistry, Blood gas, electrolytes, type and crossmatch (PRE TRANSFUSION) (1) 5mg/kg SQ Vitamin K (takes 4-6 hours to see improvement in PT, normalization within 24-48 hrs)
Suspected Hemothorax due to anti-coag rodenticide ingestion -Tachypnea, muffled heart and lungs, resp diff, chest wall pain	(5) T-fast and A-fast (4) 0.2mg/kg Methadone IV (1) O2 supplementation	(2) Thoracic radiographs (3) Therapeutic Thoracocentesis +/- autotransfusion (Based on ventilation and oxygenation evaluation)
Hypovolemic Hemorrhagic Shock w/Anemia -Shock, tachycardia, pale mm, weakness, hypotension, mild hypothermia	(2) IVC x2 (pull for full bloodwork) (3) FFP (thawed in fridge, 5mL/kg over 10 mins, at least 20mL/kg total possibly 30mL/kg), then DEA 1.1 neg whole blood transfusion (or pRBCs in other IV Catheter)	(1) second IVC if not already placed (3) Address hypocalcemia (co-factor for clotting factors) -not in same IVC as blood! -Calcium gluconate: 100-150mg/kg, when using 10% CaGluc=1mL/kg
Syncopal Event	(1) ECG, SPO2	
Acidemia: compensated metabolic acidosis		

Recheck clotting times:
-PT: 1-2 hours after first transfusion (want it to be within 2-3 times normal with no clinical bleeding), then consider 4 hours post transfusion, then again at 12 hours post-transfusion

2. Approximately 15 minutes into the transfusion the patient becomes more tachypneic, her temperature rises, she collapses, has a very thready femoral pulse, and loses consciousness. Describe in detail how you will manage this severe transfusion reaction. Include all the drug doses, any mixing instructions, etc exactly as you would tell them to the individuals assisting you.

Anaphylactic shock: treatment is epinephrine, epinephrine, epinephrine, fluids
-Vasodilatory (address w/epi) and hypvolemic (address w/fluids) and maldistributive shock

Airway (intubate if patient not fighting you regarding it), monitoring equipment (If not already on: ECG, BP, SPO2, ETCO2 if intubated)
Stop current transfusion(s)
Epinephrine (0.01mg/kg **IM for first dose**)
IV bolus: Plasmalyte 900mL (1/2 shock dose) over 15 mins (Then consider Vetstarch 5mL/kg bolus in 15 mins then 1mL/kg/hr)
-Reassess patient: if not responding then epinephrine CRI

Recheck bloodwork (PCV/TS, Venous blood gas, iCa, electrolytes, glucose)
-If rapid drop in PCV/TS: bolus more blood in (different bag)

Which is more likely to cause a reaction? Plasma or RBCs? Unsure, no good studies in vetmed.
-Type animal, crossmatch majors and minors to what is hanging (if using whole blood)
-If you can't figure out which then hang new bags OR start plasma at 10th rate of initial (can always tap patient later and give own RBCs back)

3. Discuss the major differences in diagnosis and management between diabetic ketoacidosis and hyperglycemic hyperosmolar syndrome. Be as detailed as possible.

Prognosis: 70% for both DKA and HHS (contrasting previously quoted prognosis)

You can be DKA and hyperosmolar. You can be HHS and have ketones.
-Osmolality is the true difference

	DKA	HHS
Pathogenesis/pathophysiology	**DKA:** Pathophysiology: Low insulin, high glucagon=increased lipolysis causing increased acetyl CoA which is not used and is a precursor for ketones -Counter regulatory: Cortisol, GH, epinephrine, glucagon Sources of volume loss: -osmotic diuresis: glucosuria, ketonuria -vomiting/diarrhea How do you break the cycle? -insulin supplementation: helps move glucose into cells and decreases ketone production (acidemia due to ketonemia is what makes patients feel sick, insulin helps improve acidemia) -Rate of glucose decrease is ideally: 50-100mg/dL/hr	**HHS:** Pathophysiology: increased glycogenolysis/gluconeogenesis/osmotic diuresis and dehydration causing decreased GFR causing decreased glucose loss -difference between DKA and HHS is the kidneys -temporary or permanent renal dysfunction
History, clinical signs, exam findings	Historical/Clinical Findings: -Present for: Lethargy, V+, D+ -Why V+/D+? Ketones! Causes ileus and gastroparesis=abdominal pain -Common comorbidities cats: cholangiohepatitis +/- hepatic lipidosis, pancreatitis, UTI , kidney disease (in that order) -Common comorbidities dogs: UTI, pancreatitis, concurrent infection (abscesses, bite wounds), other endocrine disease, Cushings, hyperthyroidism (in that order)	Historical/Clinical Findings: -Present for: profound PU/PD, mentally inappropriate
Major lab findings	Major Lab Findings: -Ketonemia (a few hours), ketonuria (2-7 days) -Urine dipstick: tests for acetoacetate (takes a week to see because they primarily make B-hydroxybutyrate) -How do you clear ketones? Are either breathed off, metabolized into free fatty acids (the intermediate of which is bicarbonate, see a rebound alkalosis as DKA resolves) or gotten rid of in the urine -Acidosis: Right heart doesn't do well (might need to reach for vasopressors quicker)	Major Lab Findings: -severe hyperglycemia -severe hypernatremia (this is what's truly needed to drive osmolality up), concurrent hyperchloremia -azotemia -occasionally liver abnormalities -hyperosmolar: >350 mOsm/kg -normal dog: 290-310 mOsm -normal cat: 300-320 mOsm
Treatment	Treatment: -Dehydration: lots of fluids, promotes further osmotic diuresis which needs more fluids -weights are the best way to measure this in the first few days	Treatment: -slowly rehydrate: bring Na down no more than 1meq/hr (shoot for 0.5meq/hr) -Accepted safe target=10meq/day -Rate of glucose decrease is ideally: 50mg/dL/hr (stay under 100!!)

4. Discuss the major similarities and differences between diabetes insipidus and the syndrome of inappropriate anti-diuretic hormone secretion. Be as detailed as possible.

	DI	SIADH
Pathogenesis/pathophysiology	**DI:** -lack of secretion or lack of appropriate renal response to vasopressin -Primary (Neurogenic, Central): not released from pituitary -hereditary, brain tumor, trauma, hemorrhage, stroke, granuloma (anything affecting pituitary, congenital or acquired) -Secondary (Nephrogenic, Peripheral): kidney not responding -loss of the gradient: look up for next week -drugs that cause DI: look up for next week	**SIADH:** -increased (inappropriately) vasopressin release -Hallmark: LOW SODIUM, have to have this! -ADH acts at DCT and collecting ducts at receptor V2 -binds to V2 and tells aquaporin to go into the membrane and moves water into the cell -Free water intake excessively so Na drops -Did not lose Na, just relative hyponatremia
History, clinical signs, exam findings	History: PROFOUND PU/PD (usually sudden in onset), flooding out rooms of the house with urine, intensely water seeking -no access to water: mentally inappropriate and hypovolemic	History: -can be mentally inappropriate -Free water gain=EDEMA -Iatrogenic or acquired usually (altered/damaged hypothalamic axis) -usually just came off or are on the ventilator -PPV causes decrease in ANP which decreases the stimulus -why decrease in ANP in response to PPV?
Major lab findings	Major Lab Findings: -hypernatremia (not always if they have free access to water) -hyposthenuria: less than 1.008 or 1.007 USG -hypovolemia	Major Lab Findings: -hyponatremia
Treatment	Treatment: -Let them drink! -Primary: supplement vasopressin (eye drops, DVAVP=synthetic analogue to vasopressin, alternate eyes b/c mild conjunctivitis, TID pretty religiously) -Secondary: -Example: Pyometra=endotoxin produced by e-coli blocks the receptor	Treatment: -Correct underlying process -Restrict water intake, administer Furosemide with urine output monitoring via IV Catheter -When urine output takes a jump then give them AVP -massive free water diuresis, Na shoots up from 110 to 150 -then carefully control Na increase (get them to 130 ideally) -if their mentation improves significantly at 120 then stop and sit there for a day then slowly increase -Mannitol