CRITICAL THINKING CHEMISTRY CASE CHALLENGES

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Put on your thinking caps! Who knew that chemistry analysis could be fun! Especially in emergencies!

The truth is that chemistry analysis is invaluable in emergencies, as is the minimum database. But once the patient is more stable, a full chemistry profile should be run to determine causality, or the extent of damage caused by shock.

So we know something is going on with this patient's liver, correct? Well let's categorize the extent of damage a little more…

LIVER CHEMISTRIES

When evaluating liver enzymes, typically there are three different ways to assess damage. First, the leak enzymes are evaluated (ALT, AST). These enzymes are free in the hepatocyte cytoplasm and are released when they are damaged. Second, the cholestatic enzymes are evaluated. These include: Alkaline Phosphatase, and GGT. These enzymes occur when cholestasis, or obstructed bile flow, occurs. Third, liver function can be evaluated using a variety of tests including: Bilirubin, Bile Acids, Ammonia levels, Albumin and Globulin levels, Glucose, Urea (BUN), Cholesterol, and coagulation factors.

In examining "Jackson's" blood work, we can see he definitely has hepatocyte damage, and has some elements of cholestasis. His ALT, AST, AlkP and GGT are increased. In addition, his total bilirubin is slightly elevated and his albumin is slightly decreased. These all fall in line with acute hepatic failure.

RENAL CHEMISTRIES

BUN is a marker for glomerular filtration. Meaning most of the urea produced by the body (as the last step in protein metabolism) is excreted into the glomerulus and through the urine. Therefore, a decrease in available nephrons will lead to increased levels of BUN in the blood. However, the BUN is not always indicative entirely of renal function. Azotemia, referring to increase nitrogenous waste products in the blood, can be characterized as pre-renal, renal, or post-renal. Pre-renal azotemia can be caused by an increase of urea delivered to the kidney, as a result of increase protein intake. This is typically seen in upper GI bleeding, as RBC's are digested and absorbed. In addition, if renal blood flow decreases (through hypovolemia or dehydration) GFR will decrease and thus BUN will increase. The renal causes of azotemia are any renal disease process, glomerular, tubular, interstitial, that cause a decrease in GFR as available nephron numbers decrease. Post-renal causes of urine leak or obstruction, such as uretroliths, bladder trauma, or uroliths can cause increases in BUN. Creatinine, a product of muscle breakdown, functions in a similar way to BUN, in that it is entirely filtered through the glomerulus. It also can increase if there is a disproportionate amount of muscle breakdown, such as cachexia. Similar causes to BUN increases (non-renal in nature) can cause increases in creatinine.

One helpful value might be the BUN/Creatinine ratio. However, it is not entirely reliable. Theoretically, increases in BUN that are disproportionate to the decrease in GFR may be caused by non-renal mechanisms. It appears that a BUN/Creatinine ratio of 30:1 or greater can indicate GI hemorrhage.

A must when evaluating renal markers is to assess renal concentrating ability, with a USG. The USG is a measurement of solutes vs. free-water capacity of urine. A concentrated USG means less free-water, (retention) and a dilute USG means additional free-water excretion. In times of dehydration and hypovolemia, ADH acts to retain free-water, concentrating the urine. USG should be >1.030 (dogs) >1.035 (cats) in these situations. If a patient has a decreased production of ADH or some other disease process their USG will be very dilute, or <1.008. Patients with a USG 1.008-1.012 (some say 1.012-1.025) this represents isosthenuria, or filtrate the same concentration as from the glomerulus, indicating poor renal concentrating function.

A patient with azotemia, dehydration, and a USG of 1.012, almost certainly has renal disease or damage.

CALCIUM DISORDERS

Calcium has three forms: ionized (metabolically active), complexed (chelated with other ions) and protein-bound (to albumin). Total calcium measures all three. Ionized will measure the metabolically active form. You can have a severely hypoalbuminemic patient with total hypocalcemia but normal ionized calcium.

Fortunately there are some acronyms to discuss hypercalcemia:

H- Hyperparathyroidism
A- Addison's disease
R- Renal Disease
D- Vitamin D toxicosis (Rodenticide)/Granulomatous
I- Idiopathic (cats)
O- Osteolytic
N- Neoplasia
S- Spurious (lab error)

Hypercalcemia is often seen with neoplasia (anal sac adenocarcinoma, lymphoma, multiple myeloma), Addison's disease, Hyperparathyroidism, and Idiopathic causes. In a patient where a cause cannot be readily determined (anal sac tumor, etc) a parathyroid panel to a lab may prove useful. A patient with hyperparathyroidism will have excess PTH production in lieu of hypercalcemia, meaning there is an autonomously functioning parathyroid gland. A normal response to hypercalcemia is a low PTH level. In addition, if a patient has a high calcium level, a low PTH level, they may have neoplasia excreting a PTH-rp (related protein) which will act like PTH and increase calcium levels. This can be measured in the lab. Anal glands should always be evaluated in a patient with hypercalcemia. This turned out to be an idiopathic hypercalcemia case.

PANCREATIC CHEMISTRIES

Elevated amylase levels have been reported in dogs with renal failure, GI disease and hepatic disease. Amylase is present in several tissues and is not PANCREAS-specific. Lipase is also present in several types of tissue and can be elevated in renal, hepatic, GI disease, and with corticosteroid therapy. They are not sensitive or specific for pancreatic injury. Patient with pancreatitis may have normal total enzyme function.

The options we have to diagnose pancreatitis are:

If lipase is 3x greater than upper reference range: 55-73% accuracy
If TLI is >50ug/dL: 37% accuracy
Radiographs: 24%
Ultrasound: 68%
Snap/Spec CPL test: 93%

The snap/spec CPL is a test identifying and tagging canine pancreas-specific lipase. This makes it a much more sensitive and specific indicator of pancreatic injury in dogs and cats.
In this case, this dog's test was positive, and he received symptomatic treatment.

REFERENCES AVAILABLE UPON REQUEST