Getting The Most From Your Lab Work  
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When reviewing labwork, certain cues can often be helpful in making the most of abnormalities. From the CBC to chemistry and urinalysis, there are important values that can help further direct the next steps for your patients—whether that be additional blood tests, imaging, or potentially emergent referral.

For a leukocytosis, there are many differentials, with some of the most common causes including stress leukogram, infection, lymphocytosis, and eosinophilia. The stress leukogram is characterized by mature neutrophilia, lymphopenia, and eosinopenia, while an infection is often characterized by a mature neutrophilia +/- band neutrophils and often toxic changes. In an acute and overwhelming infection, neutrophils may be low normal or even reduced, which in some conditions such as parvovirus, may be of prognostic value.

Corticosteroids can cause a severe elevation in leukocytes (mature neutrophilia), and often toxic changes are mentioned. However, it is important to be mindful that this is a normal physiologic response to corticosteroid therapy, and not an indication for antimicrobials per se. Lymphocytes can become elevated secondary to chronic antigenic stimulation, lymphocytic leukemias, and hypoadrenocorticism. Eosinophils are often elevated with allergic or parasitic diseases, as well as fungal disease, and can be elevated with hypoadrenocorticism as well.

When a leukopenia is encountered, this is not a need to automatically dispense antimicrobials. Lymphopenia is quite often due to stress or corticosteroids, and in these cases antimicrobial therapy is not indicated. If neutropenia is severe (<1500), prophylactic antimicrobials can be considered, with the exception being chronic cases where bone marrow fibrosis is suspected. Eosinopenia is most often a stress response.

Anemia should be classified as regenerative or non-regenerative, keeping in mind that there may be a lag of 3-5 days for adequate bone marrow response. Regenerative anemia may be caused by hemolysis (anemia with normal TP/TS), hemorrhage (anemia with low TS/TP). Non-regenerative anemia is typically related to bone marrow disease, renal disease, chronic illness or certain medications.

Elevated hematocrit is either relative (hemoconcentration) or related to polycythemia, which can often be differentiated by concurrent assessment of TP/TS. Thrombocytope尼亚 should always be confirmed by manual review of a slide to rule out clumping. Immune-mediated thrombocytopenia most often presents with a platelet count of less than 30,000 and evidence of clinical hemorrhage. Coagulopathies can lead to variable degrees of thrombocytopenia, depending on degree of hemorrhage. Blood loss (splenic rupture, etc) often leads to platelet counts in the 40-60,000 range, as does vasculitis. Thrombocytosis is most often associated with excess cortisol.

Elevated ALT supports some degree of hepatocellular damage, and it is important to be aware that chronic mild ALT elevations can lead to cirrhosis over time. To a lesser degree, we can appreciate ALT elevation secondary to severe cholestasis, as well. Elevated ALP may or may not indicate a pathologic condition, and can be seen secondary to endocrine diseases, benign vacular change, as well as cholestatic disease. Bilirubin can be elevated due to pre-hepatic, hepatic and post-hepatic causes. With severe bilirubin elevations, especially due to cholestatic disease or hepatic lipidosis, it may take weeks or more for values to normalize, even in the face of appropriate therapy.

Elevations in creatinine must be evaluated in light of hydration (PCV/TS, Albumin, urine specific gravity), to differentiate between pre-renal and renal disease. Post-renal azotemia is most often ruled out based on history and physical examination. Even if USpG is not available, you can use the PCV/TS and Albumin to help further differentiate renal from pre-renal causes, and the SDMA and BUN may also offer assistance, especially in emaciated patients. Renal causes, aside from chronic/acute renal disease can include pyelonephritis, Borrelia burgdorferi, as well as leptospirosis. Pre-renal causes can include hypotension, hypertension as well as hypoadrenocorticism. Elevations in BUN beyond renal disease include intestinal bleeding, dehydration, and higher protein diets. When BUN is reduced, this could indicate hepatic dysfunction. When confronted with an elevated BUN and normal creatinine, urine specific gravity and consideration of the patient’s body condition are key.

Hypoalbuminemia is generally caused by loss (via the kidneys or intestinal tract) or reduced production (hepatic disease), but be mindful that this is also a negative acute phase protein, so may be reduced in the presence of hypergobulinemia. Hypergobulinemia may indicate chronic antigenic stimulation, fungal infection, neoplasia, FIP, and is generally not appreciated with dehydration. Protein electrophoresis can be considered if severely elevated.

Hyperglycemia can be seen as a physiologic (stress) response or pathologic process (diabetes mellitus). In felines, the stress response can often approach hyperglycemia of 400-500mg/dl. Hypoglycemia can have several causes, including insulin overdose, pediatric patients, poor doers, severe malnutrition, and artifact (serum not separated expeditiously). Disease states that can lead to hypoglycemia include hepatic failure, hypoadrenocorticism, insulinoma, or paraneoplastic syndrome.
Hypocalcemia can be seen with many disease states including protein losing enteropathy, hypoparathyroidism, puerperal tetany, sepsis, renal disease, acute pancreatitis, and secondary to transfusions. Therapy and urgency should be assessed in light of the patient, as not all hypocalcemic patients require therapy. Hypercalcemia has a set list of differentials, many of which are ruled out with a history and minimum database. We use the pneumonic GOSHARNT- Granulomatous, Osteolytic/Osteoproliferative, Spurious, Hyperparathyroidism, Vit D toxicosis, Addison’s disease, Renal secondary hyperparathyroidism, Neoplasia, Idiopathic, and Toxins.

Hyperphosphatemia can be caused by transcellular shifts, reduced GI absorption, and increased renal excretion. Hyperphosphatemia is most often seen with reduced renal excretion and transcellular shifting. If associated with severe renal disease, especially if patient is ill/not eating, treatment with phosphate binders is not an urgent therapy.

Hypophosphatemia can be seen secondary to CHF, GI or urinary losses, effusions and edema. Hypoadrenocorticism should be a differential if signalment is appropriate. Also, diuretic therapy often leads to reduced sodium. Less common causes include syndrome of inappropriate ADH secretion and water intoxication. Hypernatremia is not a common diagnosis, and reflects either a gain of sodium greater than water gain (hypertonic fluids, salt toxicity) or a water loss greater than sodium loss (GI secretions, diuretics).

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Hypochloremia can be caused by excess loss of chloride relative to sodium as with GI fluid losses, loop or thiazide diuretics, and chronic respiratory alkalosis, or with a high sodium intake compared to chloride as with bicarbonate administration. Hypochloremia can occur secondary to loss of sodium in excess of chloride, as with diarrhea, or related to a gain of chloride relative to sodium (CRF, RTA). Be mindful that KBr will cause pseudohyperchoremia on labwork.

Hypokalemia can be caused by insulin administration, beta-blockers, theophylline and metabolic alkalosis (think proximal GI obstruction= hypokalemic, hypochloridemic metabolic alkalosis), RTA, renal failure, and hyperaldosteronism, as well as diabetes can lead to increased loss of potassium. Severe GI disease as well as malnutrition can also contribute to low potassium levels. Hyperkalemia can be caused by Addison’s disease, renal failure and urinary obstruction as well as metabolic acidosis, heat stroke and crushing injury. Potassium-sparing diuretics, ace-inhibitors, and administration of expired pRBCs can also cause elevations. Less commonly, pseudohyperkalemia can be associated with thrombocytosis/leukocytosis and is described in Akita breeds.

We hope to see you at our next event on Thursday, October 25th!

“Wealthy Emergencies”

presented by Rachel Lisankis, VMD, DACVO