Practical hematology and biochemistry: how to interpret bloodwork

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Hematocrit (PCV)

This parameter allows to assess the state of hydration of the horse. The hematocrit value is defined as the volume occupied by the erythrocytes contained in 100 ml of blood (expressed in percentages). It is a very variable parameter between different animals and increases under stress or exercise conditions due to splenic contraction. It usually appears increased in case of colic. It is very useful for monitoring the degree of dehydration and hypovolemia. It may appear diminished in case of anemia (Figure 1). Normally in horses it is non-regenerative anemia and is associated with chronic inflammatory processes. Chronic regenerative anemia may reflect bleeding in the intestine or abdominal cavity. Acute hemorrhages are only reflected in the analytical after 12-24 hours.

- Piroplasmosis: hemolytic anemia. Compare with hemoglobin and bilirubin.
- Gastric ulcers, RDC: chronic, regenerative anemia.
- Tumors of bone marrow: very rare in horses.
- Chronic inflammatory processes: compare with fibrinogen and other parameters (iron, leukocytes, proteins).

Leukocytes

In the horse, three leukocyte responses can be observed:

• Physiological leukocytosis: Fairly frequent in horses under two years of age. In this situation, the number of mature PMNs and / or lymphocytes increases (Rossdale et al., 1992). It is produced by the release of adrenaline as a result of fear, excitement or physical exercise, which cause an increase in blood pressure and heart rate as a result of a splenic contraction.

1. Neutrophils

- More irregular nuclear membranes than in other species (Fig. 2). Do not confuse with hypersegmentation.
- Frequently find neutropenia as a result of endotoxemia.
- Deviations to the left (Fig. 3).

Neutropenia in the equine species often occurs as a consequence of endotoxemia, which results in the marginalization of neutrophils in small diameter vessels. In these cases, toxic neutrophils are frequently observed, due to the participation of bacteria and their toxins in the pathogenesis of leukopenia. It is known that 25-35% of horses with colic have endotoxemia.

Clinical signs include:

• Paleness of mucous membranes with endotoxic halo.
• Fever.
• Alterations in capillary refill time.
• Decrease in intestinal motility.
• Increase in heart and respiratory rate.
• Reduced arterial pulse.
• Clear signs of dehydration.

2. Lymphocytes

Lymphopenia:

- Stress
- Administration of glucocorticoids
- Viral infections
- Combined immunodeficiency of Arab colts
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- Combined immunodeficiency of Arab colts

Lymphocytosis

- Excitation, exercise
- Epinephrine administration
- Chronic immune stimulation
- Lymphocytic leukemia

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3. Eosinophils (Fig.4)
Eosinopenia: relationship with the stress response. Prognosis maker.
Eosinophilia:

- Reactions of eosinophil-mediated hypersensitivity (sometimes)
- Parasitosis (larva migrans, habronema, strongyles)
- In some cases of alimentary lymphomas, carcinomas of transitional cells or MEED

4. Monocytosis

- Processes with extensive tissue destruction (necrosis)
- Chronic diseases
- Abscesses
- Stress, corticosteroids, autoimmune diseases
- Bone marrow neoplasia

5. Basophils

- Rare on horses
- Hypersensitivity reactions
- Immunomediated vasculitis

Platelets
Thrombocytopenia:

- Decrease in production (spinal neoplasms, congenital alt)
- Increased use (CID, hemorrhage, thrombosis)
- Kidney (splenomegaly)
- Increased destruction (Ehrlichia, EIA, immune-mediated diseases, some drugs and snake bites)
- Laboratory error (repeat analysis with citrate as anticoagulant)

Coagulation

Prothrombin time (TP).
• Integrity of the extrinsic pathways and detection of the integrity of one more specific coagulation factors (II, V, VII, X and fibrinogen).
• PT will appear increased when there is a deficiency of vitK, liver failure or CID.

Partial Time of Thromboplastin or Partial Time of Activated Thromboplastin (PPT).
It evaluates the intrinsic activity of coagulation and detects deficiencies in the specific coagulation factors II, VIII, IX, X, XI, XII and fibrinogen. An increase in PPT indicates defects in coagulation, deficiency of vitamin K and CID.

Fibrinogen
The concentration of fibrinogen increases with inflammatory processes. Fibrinogen increases on days 1-2 after the attack, and does not begin to decrease until 3-4 days. High concentrations lead to a worse prognosis. In horses, plasma fibrinogen is a marker of inflammation more effective than white blood count in peripheral blood. It decreases when there is CID. The normal range varies depending on the laboratory.

Total proteins
In cases of dehydration appear increased (false hyperproteinemia by hemoconcentration).
Hypoproteinemia:

- Increased bowel losses
- Decrease in production
- Kidney losses

1. Albumin
Hyperalbuminemia: dehydration
Hypoalbuminemia:

- Gastrointestinal losses: inadequate nutrition, alterations in digestion, alterations in absorption, diarrhea.
- Renal losses: Glomerulonephritis
- Of the plasma proteins it is the one with the lowest molecular weight, that is why it is the first one that is lost. Long half-life (rare hypoalbuminemia in acute liver failure due to lack of production)

2. Globulins

Apart from dehydration, the total concentration of globulins can be increased by:

- Acute or chronic inflammatory processes (caused by the increase of acute phase proteins and the concentration of immunoglobulins):
  - Abscesses
  - Paraneoplastic syndrome.
- Severe parasitism (increases in beta-1-globulins)
- Chronic hepatitis, immunomediated and autoimmune alterations and tumors (lymphosarcomas): increase gamma-globulins

3. Acute phase proteins

SSA, Haptoglobin
Increase in inflammatory problems
High sensitivity, low specificity

Urea and creatinin

Most biochemical parameters of plasma or serum increase with dehydration, including urea. However, increases in both urea and creatinine, although they remain within normal limits, may reflect a prerenal renal failure due to hypovolemia.

- They do not differentiate between acute or chronic renal failure
- They do not distinguish between prerenal, intrarenal or postrenal.

Most sensitive creatinine

High concentrations in neonatal foals: possible placental insufficiency

Urea: depends on diet

In small animals more standardized values of urea (horses that eat alfalfa, usually more urea concentration)

Glucose

It is measured in blood to compare with other biological fluids (ex: synovial fluid, peritoneal or cerebrospinal fluid) and determine if there is infection.

Hyperglycemia is considered a marker of poor prognosis since it is directly proportional with plasma cortisol concentrations and therefore indicates the degree of stress.

Hepatic enzymes

- Strangulating ID lesions do not affect the liver enzymes, however, proximal enteritis can cause an increase in ALT, GGT and AST.
- This may be due to an ascending infection from the common bile duct, absorption of toxins or mediators of inflammation in the portal circulation or by hepatic hypoxia as a consequence of SIRS or endotoxic shock.
- GGT may be increased by the presence of liver viruses without having clinical consequences
- There are no liver protectors for equine patients ¡¡

Bilirubin

Increases in total bilirubin concentration have been observed in a variety of equine diseases, not related to primary liver diseases, such as anemia, impaction and anorexia.

- Indirect: relationship with ligand and Kupffer cells
- Direct: hepatocytes (ligand)

Anorexia is the most frequent cause of jaundice in adult horses.

Neonatal foals usually have more indirect bilirubin concentration due to lack of development of ligands.

- Indirect
  - Hemolysis
  - Anorexia
  - Intestinal obstruction
  - Heart failure, administration of certain drugs ...
- Direct:
  - 25%: hepatocellular disease
  - 30%: cholestasis

1. GGT
   - Membranes of the biliary and renal epithelium
   - In exclusive liver and pancreas blood
   - In horses we consider that GGT is liver
   - Acute diseases: slight increases
   - Chronic diseases: increases considerably
   - Foals from 2 weeks to 1 month: higher values
   - Donkeys and hybrids: up to double or triple that on adult horses

2. LDH
5 isoenzymes:
LDH-1: cardiac muscle and erythrocytes
LDH-2: heart muscle and blood cells
LDH-3: lung
LDH-4: kidney, placenta, pancreas
LDH-5: liver and skeletal muscle
Always compare with other more specific enzymes

3. AST (GOT)
- All cells contain AST
- Greater concentrations in liver and skeletal muscle
- Increase in muscle damage and acute hepatic necrosis
- Always compare with other more specific enzymes

Iron plasma concentration
- Marker of systemic inflammatory / infectious processes
- More sensitive than the white blood cell count
- Faster than fibrinogen
- Negative marker
- Care: horses do not have iron deficiency anemia due to lack of iron in the diet

Alkaline phosphatase
Unspecific enzyme. Elevated levels in liver, bone and intestine. High levels in foals, associated with skeletal growth.
It is attached to the membrane of the mitochondria, so it is not usually released into the bloodstream in necrosis processes or permeability alterations of the plasma membrane. Strangulating lesions of ID have an impact on liver enzymes; however, proximal enteritis may cause an increase in the concentration of ALT, GGT and AST. This may be due to ascending infection from the common bile duct, absorption of toxins or mediators of inflammation in the portal circulation or by hepatic hypoxia resulting from SIRS or endotoxic shock.

Lactate
- Blood lactate is a marker indicative of the relationship between energy expenditure and oxidative capacity.
- It is used in the clinic as an indicator of a situation of oxidative energy imbalance in relation to intense physical activity, hypoxia and ischemia.
- The most important diagnostic and prognosis marker in equine medicine.
- Beware of clearance in horses with fluid therapy
- In decision making in horses with colic: measure peritoneal lactate
- Endotoxemia can cause hyperlactacidemia due to increased oxygen consumption and a marked increase in pyruvate production. The normal concentration of lactate in a horse at rest is 1-2 mmol / l.

Hyperlactacidemia is defined as a moderate increase in plasma lactate levels (2-5 mmol / l) without acidosis. Lactic acidosis occurs when blood lactate levels exceed 5 mmol / l and triggers a metabolic acidosis.

CK (CPK)
- Skeletal muscle, myocardium and brain
- CK of CSF is not exchanged with plasma
- Miolysis: increases from 3 to 5 times the normal concentration
- In human:
  MM: Skeletal muscle
  MB: Myocardium
  BB: brain and epithelial tissue

Electrolytes

1. Calcium
It has been shown that 86% of horses admitted to a hospital with colic had calcium values below normal (Garcia-López et al, 2001), and that 66% of horses operated on for colic had postoperative hypocalcemia, unlike elective surgeries in which only 17% showed postoperative hypocalcemia.

Hypocalcemia:

- Hypoparathyroidism
- Septicemia
- Exercise-induced hypocalcemia
- Hypocalcemia due to vitamin D deficiency does not occur in horses
- Tetracyclines, oxalates
- Administration of furosemide
- Overdosing of bicarbonate
- Acute renal insufficiency
- Rhabdomyolysis

Clinical signs: Diaphragmatic flutter, tetany, seizures, ileus.

In patients in shock, high concentrations of plasma lactate are associated with low ionized calcium. It is suspected that lactate can be a chelator of calcium ions.

Hypercalcemia

- Paraneoplastic syndrome
- Chronic renal insufficiency
- Vitamin D2 or D3 poisoning
- Secondary nutritional hyperparathyroidism
- Neonatal hypercalcemia and asphyxia

2. Magnesium

Hypomagnesemia:

Several studies have shown that 44-54% of horses suffering from colic manifest hypomagnesemia (Costa et al., 1999, López et al., 2001). 73% of the horses with ID volvulus presented ionized magnesium concentrations below the normal range during their admission.

Magnesium losses in horses with colic:

- Lack of food / anorexia.
- Renal and gastrointestinal losses.
- Stress, glucose and adrenaline release.
- Diarrhea and gastric reflux.
- Sweat.

Hypomagnesemia is often associated with other electrolyte abnormalities. Hypokalemia occurs due to the inhibition of renal potassium transport and this form of hypokalemia can be refractory to treatment with potassium supplementation if magnesium is not also administered.

3.Sodium

Hyponatremia:

The prevalence of hyponatremia in postoperative colic is 30%.

Causes of hyponatremia in horses with colic:

- Excessive secretion of antidiuretic hormone as a consequence of postoperative pain causes water gain.
- Diarrhea, renal failure, bladder rupture, severe blood loss, administration of diuretics and hyperhydration.
- Repeated administration of water by nasogastric tube to resolve impactions without supplementing with saline solutions.
- Sequestration of liquid in the third space.

Hypernatremia:

It has been described that 12% of postsurgical cramps present hypernatremia.

Causes of hypernatremia in horses with colic:

- Loss of water or net gain of sodium.
- Diarrhea, gastric reflux, renal failure and diuresis.
- The prolonged restriction of water or excess administration of salt or sodium bicarbonate can cause iatrogenic hypernatraemia.
-

4. Potassium

50% of the horses submitted to surgery for colic present postsurgical hypokalemia. Nonspecific symptoms (muscle weakness, reduction of intestinal motility).

Causes of hypokalemia in horses:

- Decreased consumption or excess losses, for example in diarrhea.
- Increase in renal losses.
- Chronic fluid therapy with RL that is poor in potassium.
- Gastric reflux, which causes chlorine losses that cause metabolic alkalosis and as a consequence hypokalemia.
- Drugs: glucocorticoids, diuretics, antibiotics.

Causes of hyperkalemia:

- In horses with colic, the predominant cause of acidosis is the accumulation of lactic acid associated with poor tissue perfusion in hypovolemia, so it is less frequent to find hyperkalemia than hypokalemia.
- Quarter horses from the line of "Impressive" can suffer "periodic hypercalcemic paralysis". It only increases potassium during crises.
- It is typical of foals with bladder rupture, together with hyponatremia.
- Plasma chloride concentrations should be interpreted together with sodium concentrations.
- The presence of lipemia can cause artifacts depending on the method of analysis used.

5. Chloride

Causes of hypochloremia:

- Diarrhea can cause severe chlorine losses due to excess secretion and lack of absorption.
- 12% of horses subjected to surgery for colic suffer post-surgical gastric reflux, and this condition can cause hypochloremia. It is also hydrogen-rich reflux, which is why it is usually accompanied by metabolic alkalosis.
- Administration of diuretics, sodium penicillin or bicarbonate.
- It is usually associated with hypokalemia, due to an intracellular deviation of potassium to try to correct the electrolyte imbalance.

Causes of hyperchloremia in horses with colic:

- 54% of post-surgical horses with colic present hyperchloremia
- Loss of water and excessive electrolyte gains, normally associated with hypernatremia.
- Metabolic compensation of respiratory alkalosis, tubular renal acidosis and water and sodium losses with diarrhea.

6. Phosphorus

Hypophosphatemia:

The prevalence of hypophosphatemia in postoperative colic is 44%. It has been described in human medicine as an indicator of poor prognosis. However, since phosphorus is primarily intracellular, hypophosphatemia is not necessarily indicative of total body depletion.

- Parenteral nutrition is usually poor in phosphorus.
- ID resections of distal regions, since it is in this area that most of the phosphorus is absorbed.
- Sucralfate (containing aluminum) can reduce the affinity of phosphorus in the ID.
- The intracellular increase of phosphorus can be produced by increases in the circulation of catecholamines, glucose and alkalosis.
- Glucocorticoids can increase the losses of renal phosphorus and hypocalcemia and hypomagnesemia also predispose to hypophosphatemia.

Hyperphosphatemia:

The prevalence of postoperative hyperphosphatemia is 12%. Ischemic damage to the intestinal walls can cause phosphorus to escape into the intestinal lumen and into the peritoneal cavity, and once it is recovered here via the portal and lymphatic system.

RECOMMENDED READINGS

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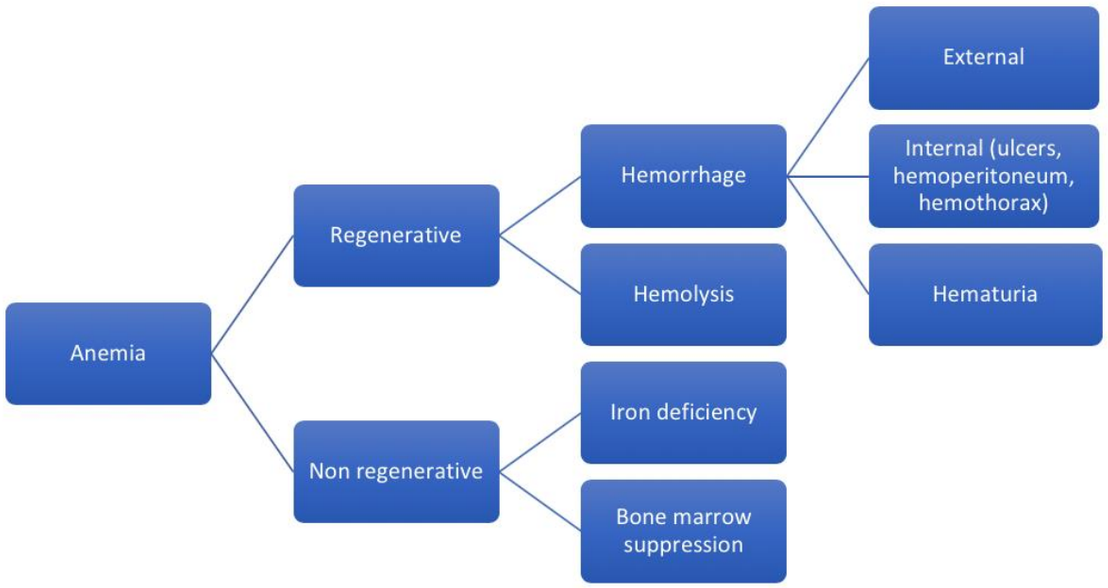

Figure 1.

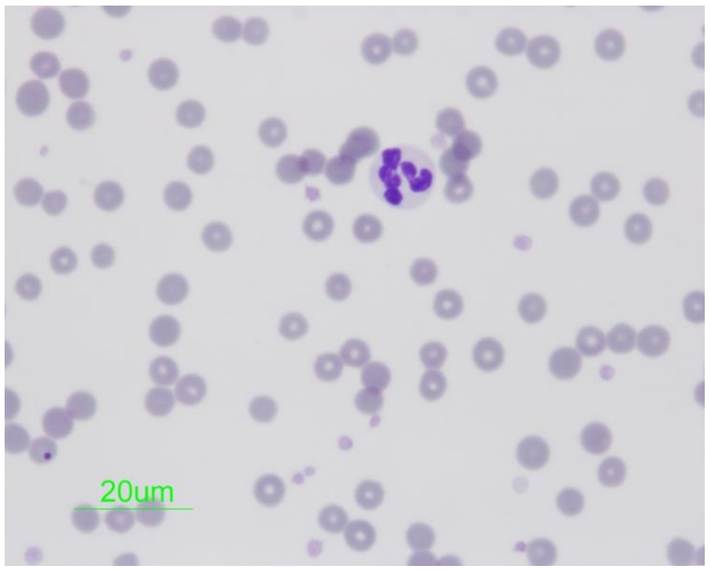

Figure 2. Normal hypersegmented neutrophil surrounded by erythrocytes

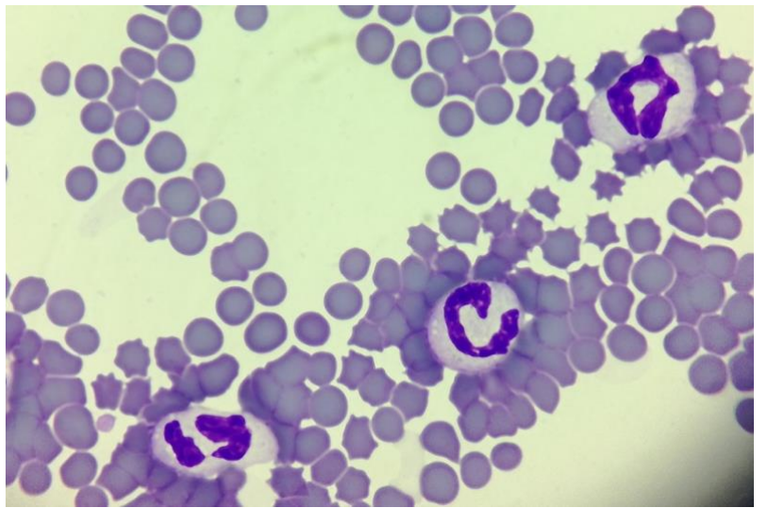

Figure 3. Toxic neutrophils (band cells)

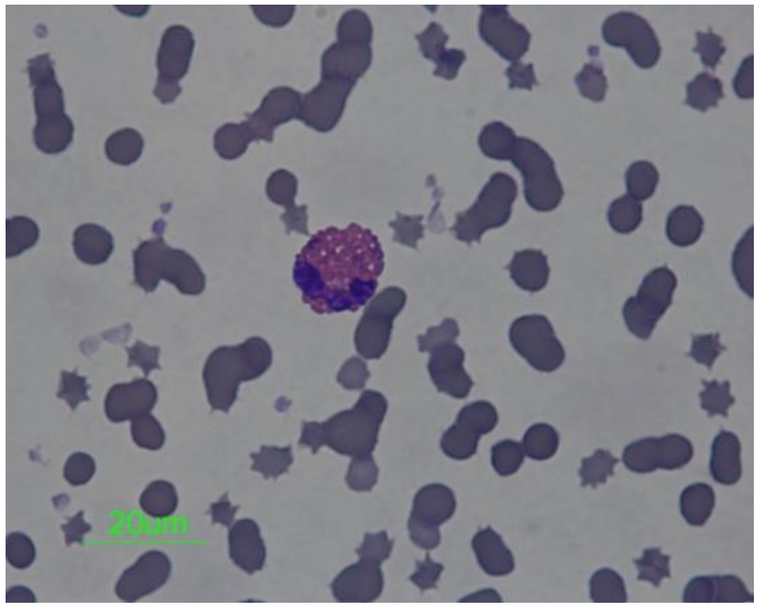

Figure 4. Normal eosinophil