White Muscle Disease/ Nutritional Myodegeneration

A PowerPage Presented By

White muscle disease occurs in certain areas of North America due to deficiencies in vitamin E and/or selenium. Vitamin E deficiency may occur when animals are fed poor-quality hay, whereas selenium may be deficient in the soil of some regions. White muscle disease is a well-known and investigated disease process that should be reviewed in preparation for board examinations. The intent of this PowerPage is to present the most important information in regard to this disease.

Key Points

- White muscle disease (WMD) is a myodegenerative disease associated with **deficiency of vitamin E and/or selenium** and can occur in horses, ruminants, pigs and poultry
- WMD is more common in **young growing animals** (foals, calves, kids, lambs)
- **Cardiac and/or skeletal muscles** are typically affected by WMD
- The cardiac form typically results in acute death whereas the skeletal muscle form is responsive to treatment

Pathogenesis

Pathogenesis:

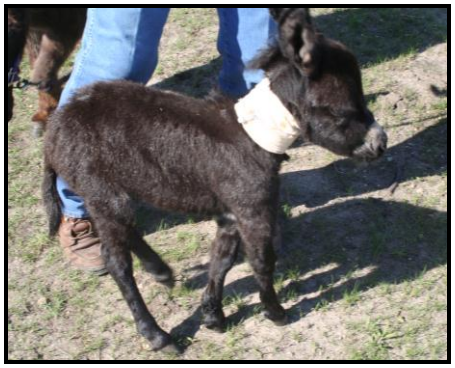
- The body produces reactive oxygen species (free radicals) during normal metabolism such as hydrogen peroxide, superoxide radical, and hydroxyl ion that are normally counteracted by endogenous antioxidants. Suboptimal concentrations of vitamin E and/or selenium impairs the animals' ability to control oxidative damage to cells, resulting in muscle damage. Specifically, polyunsaturated fatty acids that are integral in cell membranes are predisposed to oxidative damage (lipid peroxidation). In addition, muscle cells are prone to damage because of their high metabolic activity.
- Vitamin E or selenium may be deficient in certain regions
    - Selenium is a co-factor for the enzyme **glutathione peroxidase** (reduces H_2O_2 to H_2O).
    - Vitamin E (also known as alpha tocopherol) is incorporated within cell membranes and is a lipid-soluble antioxidant. Vitamin E breaks the chain reaction of lipid peroxidation within the cell membrane.
- Two main clinical manifestations of WMD are the cardiac form and skeletal form

Clinical Signs

- **Cardiac Form** – sudden onset; animal may be severely debilitated or found dead. Animals frequently die within 24 hours despite medical therapy. Lesions may be found in the heart, diaphragm and/or intercostal muscles.
  - Depression
  - Respiratory distress
  - Nasal discharge (from pulmonary edema)
  - Rapid and possibly irregular heartbeat
  - Weakness & recumbency

- **Skeletal Form** – slower onset; more responsive to medical therapy. Common muscle groups affected include the biceps, semitendinosus, semimembranosus, gluteal muscles and musculature of the neck.
  - Muscular weakness and/or stiffness
  - Recumbency, may be unable to stand
  - If able to stand, muscle weakness and trembling
  - Skeletal muscles may be hard and painful
  - Respiratory difficulty/distress (involvement of the diaphragm)
  - Dysphagia (muscle involvement of the tongue, pharyngeal and masticatory muscles)
  - Lethargy

4-week-old miniature donkey presented for prolonged recumbency and decreased nursing. Diagnostic evaluation revealed marked elevations in muscle enzymes (CK, AST), decreased serum vitamin E and glutathione peroxidase activity and histologic confirmation of myodegeneration consistent with WMD. Supportive therapy allowed recovery of the animal. Notice the stiff gait while standing (foal had difficulty rising on his own and would collapse after short periods of standing).



Diagnosis and Treatment

- **Clinicopathologic Findings**:
  - Acute disease may demonstrate significant **elevations in AST and CK** along with **hyperkalemia, hyponatremia** and/or hypochloremia
  - **Myoglobinuria** may be detected in some affected animals
  - Decreased serum selenium concentrations; **decreased glutathione peroxidase activity** is a suitable means to evaluate selenium
  - Decreased whole blood selenium (can also be measured in liver samples) and/or decreased glutathione peroxidase activity
  - Decreased serum vitamin E concentrations.

- **Gross and Histologic Findings**
  - Grossly, affected muscles of the **limbs, diaphragm, heart and tongue may appear gray to white** (hence the name). Microscopically, bilaterally symmetric myodegeneration is consistently observed. Hyaline degeneration of myofibers may be observed; regeneration of myofibers may be observed in the recovery stages.

- **Treatment**
  - Limit physical activity
  - Administer vitamin E and selenium
  - Anti-inflammatory medications and supportive care (nasogastric feeding, antibiotics, fluid therapy)

Control

Ensure adequate vitamin E and selenium concentrations in deficient regions; supplementation of pregnant animals if necessary.

References

- Lofstedt J. White muscle disease of foals. Vet Clin North Am Equine Pract 1997;13:169-85.
- Van Metre DC, Callan RJ. Selenium and vitamin E. Vet Clin North Am Food Anim Pract 2001;17:373-402.