Vitamin E and Selenium in Horses

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What causes deficiency?
Low dietary intake is the main cause of deficiency for both of these nutrients. Other factors, such as increased oxidative stress due to fat supplementation of rations, exercise and poor gastrointestinal absorption can also contribute and exacerbate the low dietary supply.

Selenium – Deficiency can vary between regions and is related to soil type used to grow the hay with lower availability of selenium from soils of volcanic origin and acidic pH. Plants take up inorganic selenium via active transport and incorporate it into organic forms. Therefore, the plant species as well as rate and season of growth will also affect selenium availability from a specific forage. Selenium concentration is often higher in younger leaves and in slower growing plants.

Vitamin E - Vitamin E is produced by photosynthesis in plants and plant concentrations are influenced by stem-to-leaf ratio, plant species, stage of plant maturity and climate conditions. At early stages of growth, grasses often contain higher concentrations of vitamin E compared to legumes. However, as the legumes mature and produce more leaf, which has higher vitamin E content than the stems, the differences between legumes and grasses become less significant. Wet, mild climate conditions favor higher concentrations of vitamin E in forage independent of growth cycle and plant type. Vitamin E content in hay is decreased by storage and drying which increases oxidative damage. Rapid dehydration and ensiling of hay causes smaller losses, however, fresh grass remains the best source of vitamin E for grazing animals.

What are the clinical conditions associated with deficiency?
Both vitamin E and selenium function as anti-oxidants preventing excessive damage to tissues under high oxidative stress. Deficiency leads to damage of cellular membranes with the tissues most commonly affected including muscles and peripheral nerves.

Equine Motor Neuron Disease (EMND) – A neurodegenerative disorder affecting motor nerves with secondary muscle wasting. It is associated with low plasma concentrations and a dietary deficiency of α-tocopherol. These horses are typically on restricted pasture access.

Vitamin E Deficient Myopathy – A disease of α-tocopherol deficiency with clinical signs related to muscle atrophy and weakness without evidence of damage to motor nerves. These horses may or may not have restricted pasture access and may or may not have low vitamin E serum concentrations. This may indicate an impaired mechanism of vitamin E uptake into the myocytes, not strictly due to deficient dietary intake.

Nutritional Myodegeneration – Disease of cardiac and skeletal muscle caused by a dietary deficiency of selenium and to a lesser extent vitamin E. Severe deficiency can result in heart failure and profound muscle damage.

Equine Degenerative Myeloencephalopathy/Equine Neuroaxonal Dystrophy – These are two closely related disorders associated with low dietary vitamin E early in life (under 1 year of age). There is a possible hereditary component, although the exact mechanism and manner of inheritance has not been described. Both are largely diseases of rule out as definitive diagnosis is currently only achieved by histopathologic evaluation of the spinal cord and brainstem tissue. Spontaneous recovery has not been described and vitamin E supplementation after the onset of signs has minimal impact.

Reaching a diagnosis
Best strategies include obtaining a complete history (particularly diet and progression of clinical signs) and physical examination. Common clinical signs of advanced disease include weakness, generalized muscle atrophy affecting the hind end most prominently, muscle fasciculations, recumbency and an "elephant on a ball" stance. Horses with selenium deficiency may have signs of chewing difficulty related to masseter atrophy and signs of cardiac failure. More subtle signs may include poor performance, mild neurologic deficits and decreased immune function. Both deficiencies can result in elevations of muscle enzymes (CK and AST) on serum chemistry. Measurement of vitamin E in plasma or serum, and selenium in whole blood or liver tissue supports diagnosis of deficiency.

Blood Sample collection:
Vitamin E: The sample should be chilled, protected from light and the serum or plasma separated as soon as possible after collection. Hemolysis will significantly reduce measured vitamin E concentration. Contact with the rubber stopper should also be minimized (keep blood tubes upright).

- Significant deficiency which is often related to clinical disease is indicated by values below 1 ug/mL
- Subclinical deficiency is possible with values between 1-2 ug/mL
- Adequate vitamin E status is indicated by values over 2 ug/mL

Selenium: Whole blood analysis is more accurate compared to serum, however, tissue samples such as liver may also be submitted. Artefact from sample handling is much less likely than for vitamin E analysis

- Severe deficiency which is often related to clinical disease is possible with values below 80 ng/ml
- Mild to subclinical deficiency is possible with values between 80-160 ng/ml
- Adequate selenium values are over 160 ng/ml but below 300 ng/ml

Biopsy of Sacrocaudalis dorsalis muscle: The muscle is located dorsal-lateral to the spinous processes of the coccygeal vertebra. Surgical incision or punch biopsy provides diagnostic specimens. Biopsy of this muscle will help differentiate between EMND and Vitamin E responsive myopathy.

Other diseases to potentially consider and rule out include musculoskeletal disorders leading to primary lameness, neurologic conditions such as cervical vertebral myelopathy (CVM) and to a lesser extent in this region EPM, as well as muscle disorders such as polysaccharide storage myopathy (PSSM) among others.

Treatment strategies
Vitamin E Supplementation: Water soluble, natural vitamin E products (d-α-tocopherol) are preferred to synthetic sources (dl-α-tocopherol) since synthetic vitamin E is not as bioavailable (less potent), values take longer to increase, and it potentially does not transfer as well into nervous tissue. When treating animals with deficiency it may take several weeks (2-4) before clinical signs improve, particularly in EMND. If using a synthetic product, consider increasing the amount of units provided by 30-50%.

- When treating low vitamin E, supplement with 5,000 – 10,000 IU/day PO
- In apparently healthy animals with normal vitamin E concentrations, 1-2 IU/kg PO (500-1000IU/day for an average 450kg horse) is adequate

Some options for natural, water soluble vitamin E supplements include Nano•E (Kentucky Equine Research, Versailles, KY), Elevate WS (Kentucky Performance Products LLC, Versailles, K) and Emcelle tocopherol (Stuart Products, Bedford TX).

Labeling: Many product labels vary in how they describe vitamin E. For the synthetic form 'dl' or 'all-rac' precede tocopherol, and for the natural form 'd' or 'RRR' precede tocopherol. Some supplements listed as natural will often contain a combination of natural and synthetic forms and others do not specify either one directly and usually can be assumed to be largely synthetic ('vitamin E supplement' or sometimes 'alpha-tocopherol-acetate' on labels).

Selenium Supplementation: Selenium enriched yeast is more readily absorbed compared to sodium selenite, although the difference between the two sources is relatively small and adequate blood selenium concentrations can be achieved with both supplements.

- Daily supplementation with 1-3mg total PO for an average 450kg horse

Injectable Selenium and Vitamin E: Combination products often have inadequate levels of vitamin E to treat vitamin E deficiency and solitary products become necessary.

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