

# Clinical Experiences with White Muscle Disease

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For many years veterinarians saw clinical cases and read reports of primary myopathic diseases in food animals and the equine. During the 1950's and the 1960's, investigative research teams found one of the probable causes to be a deficiency of either alpha tocopherol and/or selenium in the diet. Since this breakthrough, innumerable clinical manifestations of white muscle disease (WMD), selenium responsive disease (SRD), or selenium alpha tocopherol deficiency (STD) have been encountered.

Our practice area is in south central Washington and north central Oregon. The area is diverse in its agricultural activity, ranging from dryland and irrigated alfalfa to cereal grain production and to high mountain rangeland. The elevation ranges from about 1,000 feet to 4,000 feet above sea level. The average annual rainfall within the region is from 4 to 35 inches. The climate is basically a four-season type. Because of the diverse geographical configuration one would expect a lack of consistency regarding the incidence of STD. However, such is not the case. The inconsistency exists only in the manifestations of the disease.

The etiology of white muscle disease as a dietary deficiency is well documented. While it would appear that selenium may be the most important individual elemental deficiency, alpha tocopherol deficiency must also be considered a factor. Certain conditions seem to predispose white muscle disease. The use of high level inorganic fertilizers, especially nitrates, sulfates and phosphates, predispose WMD. The mechanism may be an interference by these products in the plant's uptake of selenium. Stress factors associated with systemic disease or parasitism seem to aggravate the onset of white muscle disease. Forced exercise will almost always initiate the development of the muscular dystrophy signs.

The pathogenesis of STD seems less than absolutely understood. Apparently selenium and alpha tocopherol act as antioxidants and are an integral part of the chemical process of dephosphorylation at the cell membrane level. During selenium tocopherol deficiency the cellular oxygen consumption increases two to four times normal levels. The sarcoplasmic proteins coagulate, causing the opaque or white appearance characteristic of Zenker's necrosis. The affected muscle fibers become necrotic and the area is infiltrated by connective and adipose cells. The necrotic fibers are phagocytized and may be replaced

by calcium and phosphorus salts. Affected myopathic tissue contains up to twice as much phosphorus and twenty times as much calcium as does normal muscle tissue. If the myopathic coagulation process stops prior to nuclear deterioration, the affected cell may regenerate.

In our practice area, white muscle disease manifests itself in a variety of forms, at various ages and in most species. Bovine white muscle disease is the most complex in its forms. When the cows are maintained on wheat or barley stubble or cereal grain chaff plus non-protein-nitrogen supplements, we see a neonatal form of white muscle disease. Calves born of these cows are normal or slightly weak. However, within the first 24 to 72 hours they seem to dehydrate and if unattended will die. Clinically these calves seem willing to suckle but are unable to do so. The tongue is firm and less mobile than normal. The calf is unable to develop oral suction. Necropsy reveals severe dehydration. Bronchopneumonia or foreign body pneumonia are common sequelae. Consistently the base of the tongue reveals a degenerative (Zenker's necrosis) myopathy. This form of white muscle disease may be related to the weak calf syndrome in our area.

**At one to two weeks of age we often encounter treatment-resistant calf scour complexes. In some cases SGOT tests will reveal high levels (100 or more Karman units). In these selected cases with the high SGOT, supplemental injections of BO-SE seem to facilitate the treatment of the pneumoenteritis problem. In such herds, neonatal injections of the selenium tocopherol products seem to prevent much of the calf scour problems.**

The classic white muscle disease commonly occurs in our practice area. This form is seen in calves two weeks to six months of age. The signs are usually noticed after stresses such as working the calves or cattle drives to or from the summer ranges. The signs are progressive weakness, dyspnea, coughing and ascites. The calf may suddenly die with or without convulsive seizures. Necropsy will reveal extensive degenerative-to-mineralized myopathies. The left ventricle is the most consistent site of muscle damage. The right ventricle is only occasionally affected. Skeletal muscles most affected are the flexors of the stifle and the extensors of the hock, the gluteals, the longissimus dorsi, the scapular muscles and the intercostal muscles. Usually pulmonary con-

gestion and an excess of pericardial, pleural and peritoneal transudates is noted. In relatively chronic cases, secondary bronchopneumonia is common.

In yearlings on irrigated and heavily fertilized pastures we occasionally encounter an ill-thrift condition. These are chronic poor-doers with dry, pale hair-coats, cachexia and persistent diarrhea. Fecal examinations are often positive for gastric and intestinal parasite ova. Worming appears to improve these individuals but the response is less than anticipated. SGOT tests will be high. Concurrent therapy with an injectable selenium-tocopherol product and worming may markedly improve the therapeutic results.

STD diseases are also common in other species. Weaner pigs from 20 to 80 pounds show unthriftiness, ascites and sudden death. Necropsy reveals focal hepatic necrosis (hepatosis dietetica). Injections of selenium-tocopherol control this condition.

Sheep are commonly affected by white muscle disease. The most common form is stiff lamb disease. The stiff lamb form is seen at two weeks to three months of age. The lambs are at first very thrifty but begin to be obviously tired when exercised. The muscles of the shoulders and hind quarters undergo atrophy. The scapula appear to fall away from the thorax. Autopsy shows degenerative and mineralized myopathies of the involved skeletal areas. The acute cardiac form of WMD is also seen in lambs. However, in lambs the right ventricle is most often myopathic. These forms of white muscle disease are readily controlled by the prophylactic use of the selenium-tocopherol products in the neonatal lamb.

During the past 10 years, we have encountered a condition wherein the lambs are stillborn or very weak at birth. Autopsy reveals degeneration and mineralization of the glossal and intercostal muscles. Histopathological examination confirms Zenker's necrosis. These cases usually respond to treatment of the ewe during the last month of pregnancy with a selenium-tocopherol product.

Researchers in Australia reported an increase in the percentage of multiparous births to ewes treated during the flushing period with a selenium-tocopherol product. Similar treatment in the local farm flocks seem to reduplicate this report.

The equine in our area is also frequently affected by the selenium-responsive diseases. The most common form is a mild azoturia (tying-up) syndrome involving the large muscle masses of the loin, gluteals and hind legs. These horses stiffen with exercise. If stopped and rested they may recuperate spontaneously. If the exercise is continued, the condition worsens rapidly. The affected muscle masses are hyperthermic and hyperesthetic. The urine may contain slight amounts of myoglobin. SGOT tests reveal levels of 200 to 600

Karman units (90 to 270 International units). Intravenous treatment with E-SE (Burns-Biotec) at the rate of 1 cc per 100 pounds body weight is usually effective. The injections may be repeated.

**A few cases of the cardiac form of white muscle disease have been diagnosed in foals less than two months of age. These involved severe mineralized myopathy, mostly of the left ventricle.**

Tentative diagnosis of the selenium-responsive diseases is based upon the clinical observations. However, in order to confirm their existence, SGOT tests are of considerable value. Where a herd problem exists, necropsy is the most dependable diagnostic aid. Sacrifice of a severe case may be desirable in order to have an absolute diagnosis.

Response to the treatment of white muscle disease is inconsistent. Mild cases may respond to a therapeutic dose of injectable selenium-tocopherol once every four days for four treatments. In the more severe cases the product is used daily for seven to ten treatments. In young calves where pneumonia is a common sequel, antibiotics are administered concurrently with the selenium-tocopherol.

As is the case in most diseases, prevention is the best approach. Routinely, on ranches where white muscle disease is endemic, all calves are given 2 cc of BO-SE intramuscularly at birth or as soon as possible thereafter. In a few cases these BO-SE injections are repeated at six weeks and three months of age. In a limited number of instances, where the neonatal form is very severe and non-responsive to calfhood prophylaxis, MU-SE at the rate of 1 cc per 200 pounds body weight is administered to the cows during the last month of pregnancy. Calves of these cows are given BO-SE within the first two weeks postpartum.

The products we use are manufactured for Burns-Biotec Laboratories, Division of Chromalloy:

BO-SE: each cc contains: Sodium selenite, 2.19 mgm (1 mgm selenium); Alpha-tocopherol acetate, 68 IU; Polysorbate 80 USP, 250 mgm.

MU-SE: each cc contains: Sodium selenite, 10.95 mgm (5 mgm selenium); Alpha-tocopherol acetate, 68 IU; Polysorbate 80 USP, 250 mgm.

E-SE: each cc contains: Sodium selenite, 5.48 mgm (2.5 mgm selenium); Alpha-tocopherol acetate, 68 IU; Polyoxethylated vegetable oil, 250 mgm.

**White muscle disease is a multifaceted, nutritional condition affecting most meat animals and horses which is manifested in a variety of forms. All ages of animals may be affected at least to some degree. The effects may be acute and fatal or chronic with only an insidious reduced rate of gain or performance. The response to selenium-tocopherol injections prophylactically is most gratifying.**