CAPTURE MYOPATHY IN BIGHORN SHEEP

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Since the turn of the century our Rocky Mountain bighorn sheep populations have steadily decreased. The reasons for this general decline vary from the social and agricultural activities of man, deterioration of habitat and range, and various diseases.

One of the most effective management techniques for handling this decline in bighorn sheep populations is to trap sheep from overpopulated or expanding herds and transport them to historic bighorn sheep ranges. Three different clinical but similar pathological features of an exertion myopathy were encountered following transportation of Rocky Mountain bighorn sheep.

Sheep were trapped in Custer State Park, South Dakota and on Pike's Peak, Colorado. They were trapped under a 70x70-foot nylon drop net. Alfalfa hay and apple pulp were used as bait to lure the sheep under the net. Sheep went under the net with little or no apprehension or excitement. The net was then dropped, and they qickly entangled themselves in the net. Sheep were removed from the net, collared, given injections of antibiotics, drenched with an anthelmintic, and blood collected from the jugular vein. They were then placed in the back of a small enclosed truck and immediately transported to Fort Collins. Transportation time varied from 18 to 24 hours.

Three different clinical syndromes of capture myopathy were seen in the sheep. The first syndrome encountered was characterized by acute death. These animals showed severe depression shortly after capture and were reluctant to move. These sheep usually died from 3 to 12 hours following capture.

The postmortem findings in these sheep were meager. Mild pulmonary edema and congestion of the viscera were the only findings. Muscles were uniformly dark red with no noticeable gross lesions. This condition was found in 4 of 32 sheep.

The second syndrome was characterized by ataxia and myoglobinuria. These sheep were released 24 hours after capture and had characteristic clinical signs. They had a posterior ataxia and a mild to marked torticollis. Most were depressed. Sheep were oliguric and the urine was brown (coffee-colored). Females usually aborted within 1 to 3 days. Sheep died with this syndrome 2 to 5 days after capture. This form of capture myopathy affected 7 of 32 sheep, of which 3 died and 4 survived.

On postmortem examination the most obvious lesions were:

1. Enlarged, swollen, firm, dark kidneys that had a fine granular cut surface (myoglobin nephrosis).

2. The bladder was usually empty or only contained a small amount of brown urine.

3. There were multifocal, small to large, irregular, pale, soft areas in muscles in the cervical region, lumbar area and flexor and extensor muscles of the hock and stifle joints.

4. The cut surface of these muscle lesions were pale, dry, and granular.

The third type of syndrome encountered was characterized by bilateral rupture of the gastrocnemius muscles. These sheep appeared clinically normal when released from the truck 24 hours after capture. These sheep developed a bilateral rupture of the gastrocnemius muscles within 24 to 48 hours following release. There was no evidence of depression, myoglobinuria, or torticollis. The clinical signs included:

1. Marked drop in the hind quarter and hyperflexion of the hock. These animals could still walk or even run but with great difficulty.

2. The gastrocnemius muscles were swollen and firm. The distal extremity of the affected legs were cold.

These sheep were casted and placed in a small pen. One of the sheep with a bilateral rupture of the gastrocnemius muscles did survive and 2 months later she could run nearly as fast as the other normal sheep with only a mild limp in the hind legs. This condition occurred in 8 of 32 sheep, 7 of which died.

The postmortem lesions of these sheep can be summarized as having:

1. Massive hemorrhage in the subcutaneous tissues of the hind legs.

2. Multifocal, small to large, pale, soft, irregular, sometimes depressed lesions in the muscles of the forelegs, hindlegs, diaphragm, and cervical muscles. These pale lesions were accentuated by small white foci (1/2 mm) that were located regularly along individual muscle bundles. Few muscles were entirely pale to white; for example - common digital extensor of the forelegs.

3. Other muscles were dark red, firm, with a pitted dry cut surface. These muscles were completely to partially ruptured.

   These include:

   Gastrocnemius - 5 sheep bilateral, 2 unilateral

Subscapularis - 4 sheep bilateral

Middle and deep gluteal - 2 unilateral

Semitendinosis - 1 unilateral

The histopathological lesions in the skeletal muscles of these three forms of capture myopathy were all similar. No myocardial lesions were seen in any case. Even though there were no gross muscular lesions in the acute death syndrome, histological lesions were prominent. These include:

--acute granular necrosis of myocytes; with a phosphotunstin acid heomtoxin (PTAH) stain there is a loss of basophilia and striations

--myocytes greatly swollen;

--pyknotic sarcolemmal nuclei;

--fragmentation and cleavage of myofibrils;

--no inflammatory cell response;

--very little blood in vessels; however, vessel walls were normal.

A biopsy of ruptured gastrocnemius muscle (30 hours postcapture) with a PTAH stain demonstrates similar lesions seen in animals that died with the acute death syndrome.

Animals that died at 12 hours postcapture also had similar histological lesions.

An animal with the ataxic myoglobinuria syndrome that died 2 1/2 days following capture also had identical lesions, these include:

H&E stain:
1. acute granular necrosis; with loss of basophilia and striations on PTAH;
2. swollen myocytes;
3. pyknotic sarcolemmal nuclei;
4. loss of striation due to cleavage, fragmentation and disruption of myofibrils.

One animal with ataxic myoglobinuria syndrome that died at 4 1/2 days following capture also had similar muscular lesions except there was evidence of sarcolemmal nuclear proliferation and regeneration. The kidney tubules were filled with cast, possibly due to myoglobin.

An animal that was euthanized at 7 days postcapture which suffered from bilateral rupture of gastrocnemius muscle had similar muscular lesions with the addition of advanced fibrosis and proliferation of sarcolemmal nuclei with attempts at regeneration.

In summary, we have seen three different clinical syndromes in bighorn sheep that are associated with trapping and transportation or rather physical exertion with continual stress. It is thought that the initial lesion was an acute granular necrosis of striated muscle. This lesion was consistent in all sheep that died and the longer the animal lived the more regeneration of muscle was seen histologically. Evidence of regeneration did not begin until 4 to 4 1/2 days postcapture. It is tempting to speculate that the time of death or the set of clinical signs manifested were due to the extensiveness of the muscular necrosis and not different etiologies.

The etiology of this condition is still unclear. This condition does not seem to be an example of White Muscle Disease (vitamin E-selenium deficiency) because:

1. Adult animals are much more susceptible than young animals; with White Muscle Disease the young are more susceptible.

2. Vitamin E and selenium injections did not reduce the incidence.

3. The lesions in the recovery phase of this disease are identical to the White Muscle Disease so it could easily be confused with it.

This condition is thought to be similar to Azoturia (Equine Paralytic Myoglobinuria) in many clinical and pathological aspects. One can theorize that there was a combination of tremendous release of epinephrine (due to fear and excitement) which caused an increase in glycogenolysis in muscles. With muscle exertion, the muscle metabolism is rapid, thus quickly depleting its oxygen. This encourages anaerobic glycolysis, which increases the formation of lactic acid because pyruvic acid is produced faster than it can be decarboxylated and used in the citric acid cycle in mitochondria. This increase in lactic acid results in injury to the cell. Blood flow to the muscle is also reduced due to the physical contraction of the muscle itself which only complicates hypoxia and build-up of lactic acid. High concentrations of lactic acid can also produce vasospasm. Thus a vicious cycle is initiated which leads to a generalized acidosis and death.