Review on capture Myopathy in Wild life and its effect on health of Animals

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Summary: Capture myopathy the name is given to **Complex Disease** that is usually involves transport. It can be caused by a single factor, but follows more a series intertwined events. The range of clinical signs begins in the early stages as hyperthermia (body temperature is above normal - animal is hot - sweats, pants), trouble breathing, fast heart rate. The animal with clinical signs of myopathy and its prognosis is poor. There are several interrelated ways in which capture myopathy can develop in animals. In the most acute form there is a very rapid buildup of acid in the animal's blood it can die within a minute. Capture myopathy occurred when the muscle is exerted (used) its metabolism changes from aerobic (uses oxygen) to anaerobic (uses stored energy in the muscles). This leads to the buildup of lactic acid causes acidosis.
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1. Introduction

This condition has many names: exertional, transport, stress, degenerative myopathy and white muscle disease. There are a lot of myths and misinformation about this condition. The first myth to destroy is that this is a condition seen only in marsupials (Voermans *et al*., 2009). It affects all species - and is most widely recognized in hoofed animals such as deer. However, birds - such as long-legged water birds and raptors may also be affected. All ages and sexes are susceptible from a Joey in a pouch onwards. On game farms the disease is virtually certain be caused by bad management and on the well run operation should be a non existing entity.

The end point in capture myopathy scenario is almost always death. This may occur over a very short period of time or have a more prolonged onset that causes that animal to become chronically lame, unable to stand finally creates enough damage to kill (Virginia and Richardson, 2000).

The name capture myopathy has emerged from a pack of many names by which syndrome has been described over the years. Even now the name is not universal. Many species may be affected. Virtually hooved animals, many birds, and some others have been shown to be susceptible. Even human can develop a very similar conditions. On the other hand the disease never been clearly demonstrated in a carnivore (Voermans *et al*., 2009).

2. Capture Myopathy

2.1. Definition

Capture myopathy the name is given to **complex disease** that is usually involves transport. It can be caused by a single factor, but follows more a series intertwined events (Seene, 1994).

2.2. Causes capture myopathy

There are several situations that can cause capture myopathy. These include trapping, capture, transport and even simple restraint. In other words - humans cause this condition! However, it is also used successfully as a hunting tool by large carnivores such as dogs. In general the cause includes shock, fear, ambient temperature, over exertion, diet and overheating etc (Chawla and Jasvinder, 2011).

2.3. Occurrence of capture myopathy

When the muscle is exerted (used) its metabolism changes from aerobic (uses oxygen) to anaerobic (uses stored energy in the muscles). This leads to the buildup of lactic acid causes acidosis. Lactic acid in the bloodstream drops the pH in the body, affecting heart output. If the heart does not pump oxygen to the muscle it starts to die. When the muscle dies over the next 7 days, it releases a product called myoglobin (breakdown product of muscle). Myoglobin damages the excretion part of the kidney (the renal tubule). Other organs are affected: the lungs become congested and bleed. The liver becomes swollen and pale (Voermans *et al*., 2009).

2.4. Clinical signs

We need to appreciate that we are seeing a disease in a spectrum of severity, over a number of days. It may occur in any muscle group in front and back legs or heart. It has been classified into four appearances that can help to understand what carers can see: 1) hyperacute - very sudden onset with death often noted. 2) Acute - from heart muscle necrosis and occurs over 2- 4 days. 3) Subacute - kidney failure from the release of myoglobin. 4) Chronic - die over 2-4 weeks due to heart failure and paralysis.

The range of clinical signs begins in the early stages as hyperthermia (body temperature is above normal - animal is hot - sweats, pants), trouble breathing, fast heart rate. The animal may become weak or have a stiff gait. Muscle tremors - either involving a few muscles or an entire muscle group may be seen. The animal may collapse and die (Voermans *et al*., 2009).

2.5. Species

Of animals seen in game farm the prong-horn, a difficult animal to maintain in captivity in any case is probably the most susceptible to capture myopathy. It has however, been seen in bison and all the deer species.

2.6. Method of restraints

The most likely time for game farm animals to develop CM is during transport to a new property, especially if they have recently come from wild. Vigorous exertion combined with other stressful conditions will certainly predispose the animal to this CM. On well managed game farm animals this type of situation probably will never arise.

However, chasing is not the only method of restraints that can lead to CM. it has been seen in both bighorn sheep and snow geese that were caught at baits sites under drop and cannon nets. It is possible the vigorous isometric muscle exercise played a role in the development the conditions (Chawla and Jasvinder, 2011).

2.7. Season

There are at least two ways in which season can have an effect on how an animal respond to restraints. The first is the animal's nutritional state. The translocation of wapiti from Elk Island National Park has long been practiced. It has been found that moving animals after the end of February creates an acceptable incidence of CM.

2.8. Mechanics

An important way in which oxygen reaches to the muscle via their pumping at the time of animal movements. At the same time toxic materials are carried away. In captured animals that is not moving this pump is no longer working. This can further compromise the animal. Not only fresh oxygen not arriving, but also lactic and other products are building up. Even cooling is reduced as the blood does not leave the area. This system plays an important part in the development of capture myopathy (Chawla and Jasvinder, 2011).

2.9. Manifestations

There are several interrelated ways in which CM can develop in animals. In the most acute form there is a very rapid buildup of acid in the animal's blood it can die within a minute. The nutritional stress should be alleviated by proper feeding on game farms. Of special importance is the vitamin E level in the ration. High level of vitamin E (above 200 IU per kg of feed) of this vitamin in ration will protect the deer from capture myopathy (CM) that develops during handling procedure (Dingerkus and Montgomery, 2002).

2.10. Ambient temperature

An environmental temperature plays an important role in the CM story. Restraints should be avoided during hot days. Anything above 25^{o}c is probably too hot. In summer one probably arranges to carry out any restraint during the early part of the day.

2.12. Fear

Of all situations leading up to CM the degree of lameness and reduction of fear may play the most important role. Well acclimatized animal trained to the handling the system on game farm are unlikely to be frightened when being handle. Newly caught ones are obviously more nervous and certainly more susceptible to a complete kidney shut down.

2.13. Differential diagnosis:

2.13.1. White muscle disease:

Muscle is a pretty simple organ. All it can do when it is sick is to die! So white muscle disease, which is seen in calves due to a lack of vitamin E and selenium deficiency has the same appearance as myopathy when you slice the muscle and look at it under a microscope (Chawla, 2011).

2.14. Treatment of capture myopathy:

The bad news is that if you have an animal with clinical signs, its prognosis is poor. In other words - it is not going to get better. In other words - treatment is not effective and continuing to let the animal suffer becomes a welfare issue (Chawla and Jasvinder, 2011).

The muscle has died. It cannot re-grow. This has an implication with the welfare of the animal then being treated, only to suffer horrible cramping, pain from failing kidneys, inability to breathe from

congested lungs and then to die up to 1 month later. It also has an implication for release - if the muscles are destroyed - then how is it ever going to be 100% fit for release? Field treatment in the hands of many experienced veterinarians has not been successful. Please remember, that humans are sometimes not saved in similar situations (ecstasy overdose, malignant hyperthermia in susceptible people). However, in situations where it is recognized at the hyperthermia stage, it is believed to be possible to treat it. Treatment is quite intensive and expensive (Chawla, 2011).

1) The first step is to sedate the animal. *Although from a prevention perspective, this should have already been performed.* Sedation with valium may reduce anxiety and assist in muscle relaxation.

2) the most important treatment for this condition is **intravenous fluids** eg: with Hartmans solution or 0.9% saline. Essentially, what you are treating is acute kidney failure. The goal of giving fluids is to: 1) Improve the blood supply to the kidney. 2) Dilute the damage that myoglobin does to the kidney. 3) Dilute the lactic acid in the blood stream, thus improving heart function. 4) Expand the blood volume and address the mechanisms of shock. 5) Reverse the hyperthermia.

3. Another **muscle relaxant** that is used by zoos is **Dantrolene.** This is used in humans for a similar condition - malignant hyperthermia. The drug is given intravenously within 6 hours of reconstitution. It can damage the liver and kidney. It is a human prescription only drug - few vets stock this (Chawla, 2011).

4. **Cortisone** can be used for its anti-inflammatory properties. It may also help to reverse hyperthermia.

5. **Vitamin E and selenium** can be used. This comes as **Selvite-E.** The dose rate is 1ml per 50 kg. It is given every 7 days under the skin. More frequently will harm the animal. Selenium works in the cell membrane as an antioxidant, and similarly, Vitamin E out of the cell.

3. Prevention And Control

This is the key to this condition as you cannot undo the damage, you want prevent it from occurring in the first instance. The animal's life is in your hands. The goal is planning: plan the capture, plan to use sedation, plan what to do if something goes wrong.

1) The method of capture needs to be well-planned, with sufficient people-power to quickly trap the animal. And that is the key - we do not chase macro pods, we encourage them into a trap. Time spent observing the animal and the escape route it wants to follow, and then coordinating your movements is invaluable (Chawla, 2011). 1) Minimize the pursuit time - ideal is less than 3 minutes! After this, muscle enzymes are beginning to climb and the damage begins. 2) Reduce struggling by covering eyes and placing the captured animal into a bag. 3) Keep the human noise down. No dogs should be present. 4) Reduce the amount of handling time and then release to a less stressful place as soon as possible.

2) Sedation. Consider the use of **valium, azaperone** (**Stresnil** for pigs), or **fluphenazine** (**Modecate** - a human antipsychotic used very successfully in zoos for translocation of animals but takes up to 3 days to start to work). 3) Do not catch up animals when the ambient temperature is over 20°C. Do not leave a sedated animal in direct sunlight. 4) Ensure that you keep the animal's temperature down: good ventilation, damp cloths if required.

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