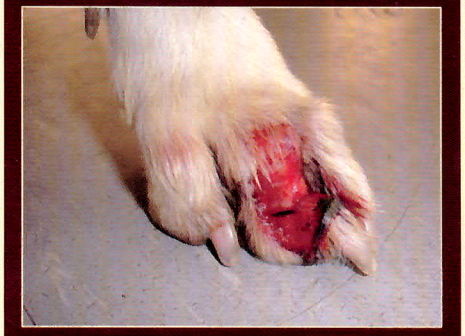
Care of the Racing & Retired Greyhound



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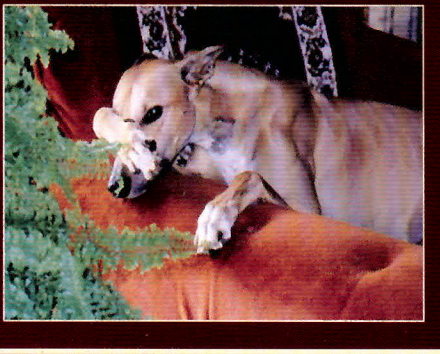

of the body fluids to buffer the excessive hydrogen ions produced by the exercise. This results in muscle cell membranes dysfunctioning, leaking, and even disrupting.

Predisposing Factor 3

When environmental conditions are hot and humid, the Greyhound has an increased difficulty in dissipating heat since it is primarily a non-sweating animal. Loss of body heat is dependent, to a small degree, on conduction (contact with cool surfaces) and convection (cooling air flow), but mainly depends on evaporative cooling from the respiratory system. Greyhounds in trailers or hot or humid kennels will pant a great deal. This adds to the humidity of the area. With increased humidity, cooling by evaporation from the lungs by panting is markedly diminished. In such situations, one observes markedly rapid increases in rates of respiration (times of breathing per minute). This may be accompanied by considerable salivary drooling (200 to 300 ml of saliva) in some Greyhounds. The result is that prior to racing, the Greyhound has an elevated body temperature (104°F or 40°C) and a lowered bicarbonate reserve and is dehydrated with low blood and muscle potassium levels–all due to the panting as described above which contributes to the development of acute exertional rhabdomyolysis.

Predisposing Factor 4

Any condition or diet which causes a depletion of the total body potassium level can lead to an animal predisposed to exertional rhabdomyolysis. For example a patient with a kidney infection or diarrhea may lose significant potassium. The addition of supplements or nutrients rich in table salt (sodium chloride), for example, those supplements designed for race horses. Equines lose body heat by the evaporation of sweat which is essentially a saline solution. This saline needs to be replaced and frequently supplements for the race horse will have high salt levels, sodium is always retained in preference to potassium salts and this will cause a loss of performance, cramping and even exertional rhabdomyolysis. Kennels feeding bread as a carbohydrate source should be aware that it also has a high salt content.

Clinical Signs

The Greyhound exhibits distress between 3 and 5 days after the race with acute pain when palpated over the longissimus, quadriceps and biceps femoris muscles (the major muscles over the back ["saddle" area] and above the stifle or knee). Myoglobinuria may be observed only once or twice after the race or trial; thereafter, the urine shows no obvious discoloration when voided, but may test positive on the dipstick test (see urine testing on page 127 in Chapter 7).

Treatment

The therapy is the same as described for the hyperacute form. The chance of death (mortality) in these acute cases reaches 25% if untreated, but recovery rates of 100% are now achieved if therapy is instituted within 12 hours of onset and a convalescence of 8 weeks is given.

Prevention

Prevention will depend on which of the predisposing factors is present. A tense and hyperexcitable Greyhound can be given 75 mg/kg body weight potassium citrate with 400 mg/kg glucose in 150 ml water and 50 ml milk (for palatability) about 15 minutes prior to traveling and kenneling. Many kennels for holding Greyhounds prerace are climatically controlled and temperature and humidity are not factors. If this facility is not available, then promotion of air movement using forced draught fans or extractor fans would be desirable. Reduction of body temperature (when elevated) by hosing the Greyhound (if permissible) before the race is recommended. Do not hose the kennels or kennel areas as this raises humidity levels if there is no forced draught or air movement.

Subacute Exertional Rhabdomyolysis.

This form is never fatal and myoglobinuria is rarely observed. This form is most commonly seen in fit Greyhounds that are trialing or racing too frequently. A possible explanation for this involves the potassium ion. Potassium ions are in high concentration inside cells and in low concentrations in the extracellular fluids (fluids in the body but on the outside of the cells). It has been documented in dogs that when a muscle cell contracts, there is an outflow of potassium ions from the cell. These potassium ions act upon the blood vessels, the arterioles and capillary bed to open them to increase blood flow (vasodilation). In effect, this helps dissipate the heat produced with exercise, flush out the waste metabolites (including the hydrogen ions), and increase the inflow of oxygen, glucose and bicarbonate buffer to the cells. If the potassium levels inside the cells progressively fall (i.e., reaches a stage of a relative potassium deficiency), then there is insufficient outflow of potassium ions on muscular contraction to stimulate the local vasodilation. In fact, all of the cellular changes of exertional rhabdomyolysis have been produced experimentally by lowering the intracellular potassium levels. There is also scien-

tific evidence to show that an increased amount of potassium and hydrogen ions are excreted in the urine after severe muscular exertion. This process is aggravated by the increased levels of cortisol and aldosterone (adrenal gland hormones) produced by the Greyhound under conditions of a stressful training-racing program. It is well documented that cortisol will promote sodium retention and potassium excretion via the urine.

It appears, therefore, that some Greyhounds subjected to a stressful racing program, i.e., two races or trials per week, suffer a relative potassium deficiency. This is relative in the sense that, while the deficit is not sufficient to produce marked changes in the blood or extracellular fluids, it is sufficient to cause a failure to induce the vasodilation of the blood vessels during muscular contraction. The outcome is then a retention of the heat produced by exercise and a reduction in blood flow to the muscle cells (ischemia). This leads to the subacute form of exertional rhabdomyolysis.

Clinical Signs

The trainer may notice that the Greyhound did not run well and appeared "tied up" or had "shortened stride" after 200 to 300 meters. The Greyhound may appear to be normal in all respects until the muscles in the saddle area (the longissimus thoracis muscle group) are palpated. Then the muscle soreness is very evident on palpation, but this often takes 24 to 72 hours to develop fully. Observations suggest that the inflammatory changes of myositis with localized disruptions of the muscle cell membranes do not reach a peak for 24 to 48 hours after the race. Myoglobinuria or red urine is rarely seen but a urine analysis will frequently give a positive test for blood (hemoglobin and myoglobin). Abnormal levels of protein may also be found in the urine indicating protein loss via the kidney. When pH is measured in affected Greyhounds, it is often alkaline with pH values of 7.0 to 8.0.

Treatment

Immediate treatment is aimed at minimizing any damage to the kidney from the precipitation of the myoglobin protein. Therefore, keep the urine alkaline with potassium citrate or potassium acetate at 0.5 grams twice a day for 10 to 14 days. Anabolic steroids may be indicated to combat loss of body protein (promotes positive nitrogen balance). Good nursing comprises a warm environment, soft bedding, normal racing diet, and only walking exercise for 2 weeks. The application of heat will help reduce the soreness of the affected muscles. This may involve the use of an infrared lamp, hair dryer, hot towels, or, with magnetic field therapy at high frequency for 10 minutes once or twice a day for 5 days. Nonsteroidal anti-inflammatory drugs such as phenylbutazone may be used during the initial few days to reduce inflammation and pain. Recovery takes 4 to 7 days with therapy.

Prevention

Avoidance of this form of exertional rhabdomyolysis is based on the theory that a relative potassium deficiency is the precipitating problem. Also prevention is directed at finding some indicator that the Greyhound is overstressed by the current exercise program. Some Greyhounds, even when physically fit, if stressed by an excessive frequency of fast work (i.e., two to three races or trials per week) will develop more acidotic urine than normal with a pH range of 5.0 to 5.8. Normal Greyhound urine should be in the pH range of 6.0 to 6.5 for maximum performance based on testing a midstream collection of the first urine sample in the morning. A prolonged acidotic state (3 to 6 weeks duration) will induce a relative potassium deficit. This critical stage is reached (can be detected through urine pH analysis) when a Greyhound passing acidotic urine over a period of time suddenly undergoes a dramatic shift from acidotic pH values of 5.0 to 5.8 to alkalotic pH of 7.3 to 8.3 without any dietary or work load changes. When this happens, it indicates a metabolic alkalosis (increased pH of the blood) which is associated with a relative potassium deficiency. If the Greyhound is raced or trialed at this stage, a subacute rhabdomyolysis may result.

Prophylaxis of subacute exertional rhabdomyolysis lies in monitoring the urine of Greyhounds that are under this type of racing stress or that have had problems with this disease in the past. The urine sample for testing must be taken at least 8 hours after feeding, 2 days or more after a hard run and from a Greyhound not on any medication for 24 hours. Urine dipsticks are sufficient to measure urinary pH (see page 131 and Figures 7-6 and 7-7 on Plate XII). If a change from acidotic urine to alkalotic urine occurs under the circumstances prescribed above, then oral potassium supplementation should be instituted with potassium chloride (KCl) at 0.75 to 1 gram daily or a slow release potassium preparation.

In addition, it is strongly recommended that trainers reduce the frequency of trials and/or races to a level at which the work load is sufficient to maintain the urinary pH at 6.0 to 6.5. For Greyhounds with cooperative trainers, it is preferable to alkalinize the urine with potassium citrate or potassium acetate when the pH is first detected to be below 6.0 and to adjust the training program at that stage. However, given the practicalities of a racing kennel, this ideal is not