Diagnosis by Breed:
A Hodge-Podge of Breed-Related Internal Medicine Problems

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Although we at times are not pleased by breeders, the very tight breeding that has went into producing the purebred dogs we see in practice can make our jobs easier in regard to diagnosing complex internal medicine problems. It is important to know about these diseases as the breeders are often better informed than veterinarians regarding some of these diseases. This is especially true with the advent of the Web and the enormous amount of information (and disinformation) available for retrieval.

Thrombocytopenia and femoral artery thrombosis in cavalier king charles spaniels

Most of us are familiar with the huge proportion of the CKCS that develop very early onset mitral and tricuspid valve endocardiosis. This breed does however have some other peculiarities that are worth mentioning. One such problem is the presence of benign thrombocytopenia. In this breed dogs have been detected that routinely have platelet counts below 100,000/ul. A bleeding tendency has not been detected. Part of the thrombocytopenia is spurious if only machine counts are used. Many of the breed have macrothrombocytes (2 to 3 times normal sized platelets). Automated counts will be falsely low because the platelets are counted as red cells. Other CKCS actually do have a low platelet count, even when a manual count is performed. A bleeding tendency however is not always present. This of course does not prevent a CKCS from having immune-mediated thrombocytopenia.

When presented with a CKCS with a low platelet count it is vital to confirm this with a manual count or at least estimate. The presence of a low platelet count and very large platelets could be consistent with this breed specific problem. A bleeding tendency should also not be present, in other words no signs of thrombocytopenia (petechia, ecchymoses, epistaxis) should be present and preferably a buccal mucosal bleeding time should also be in the normal range. Apparently bigger platelets are hemostatically more active than smaller ones. If in doubt immune suppressive therapy can be tried, if it is the CKCS idiopathic asymptomatic thrombocytopenia there should be little to no response.

Another newly recognized problem in the breed is the presence of femoral artery thrombosis. This has been detected in 6% of CKCS investigated. Generally there are no clinical signs other than a weak or absent femoral pulse. Lameness, pain, etc. are generally not present since collateral circulation can take over supplying blood to the affected limb. The disease appears to be localized to the femoral arteries, as other vessels have not been found to be involved. Treatment is not needed.

Alabama rot in greyhounds

Certainly not the most flattering name for a disease, however it is easy to remember. The more scientific name is “cutaneous and renal glomerular vasculopathy of Greyhounds (CRGV). It has also been termed Greenetrack Disease. This is a disease of undetermined etiology, though it has some resemblance to hemolytic uremic syndrome in humans that is caused by E. coli secreting shiga-toxin (E. coli O:157). To date infectious agents have not been identified in affected dogs, though they were searched for. The majority of dogs are racing dogs, however it has been seen in some pet dogs as well. It is uncertain whether the common practice of feeding racing dogs raw meat is a factor or not.

The disease can present as a multifocal ulceration and edema preferentially of the rear limbs or in combination with acute renal failure (often anuric or oligouric). Both males and females are affected. The skin lesions tend to be dramatic, initially just ulcers are present that tend to coalesce so that a large part of the skin of the rear limbs sloughs. Edema can be present as well. Many dogs also develop renal dysfunction with azotemia. In these dogs, prognosis is guarded to poor as the majority succumbs to the renal failure, though we have treated one dog successfully. Another common finding in these dogs is the development of thrombocytopenia, especially in dogs that have or develop renal dysfunction. The thrombocytopenia can be quite pronounced and cause clinical signs. Anemia also develops as a result of microangiopathic hemolysis, probably in the kidneys (fibrin strands form in blood vessels and the RBCs are lysed as they go through that vessel).

PKD in persians

Polycystic kidney disease is not a new disease, however efforts are underway to try to eliminate the disease from the breed. The problem is that the disease tends not to manifest clinically until late in life (usually not before the cat is 6 to 7 years of age) if at all. Fortunately, although clinical manifestations occur late, the cysts can be detected fairly early in life so that a screening program can be successful. The incidence is high, it may be that 30% of more of the breed is affected. The reason for this is that the disease is transmitted in an autosomal dominant mode, making for rapid spread within a line.

The cysts develop through apoptosis, that is dying off of the cells, inflammation is not generally involved. The cysts become progressively bigger, till little renal tissue is left. The liver can also be involved with multiple cysts developing as well, though this is relatively rare. Diagnosis is best achieved by ultrasonography using a high frequency probe (7.5 mHz). Although cysts can be

identified as early as a few months of age, ultrasound at 1 year is preferable. At this age the cysts have had time to enlarge so that a skilled ultrasonographer can properly diagnose over 90% of the cases correctly. Unfortunately, there is no specific treatment available. Once renal failure develops, standard supportive care is indicated.

Shar pei fever

This is an odd disease that may have some similarities to a disease in humans called Familial Mediterranean Fever (FMF). Predominant clinical signs are fever (lasting 1-2 days) and swelling of the tarsus (also sometimes called swollen hock syndrome). This can also be associated with the development of renal or hepatic amyloidosis. The clinical signs tend to wax and wane. Supportive care (analgesics, antipyretics) is indicated for very high fevers, otherwise signs tend to remit without intervention. More importantly however, renal failure through amyloidosis should be prevented. Many of the dogs that have this disease can be expected to develop renal insufficiency at 5 years of age and older. Unlike most cases of amyloidosis, the amyloid tends to be in the medulla so that proteinuria is uncommon, whereas renal insufficiency predominates. Colchicine has been recommended. This is based upon the fantastic response of people with FMF. There are also indications that dogs do well with this treatment, though fever episodes may continue. Dosage is 0.025 mg/kg to 0.03 mg/kg SID. Bone marrow suppression and GI signs can occur so CBCs should be monitored.

Perianal fistulas in german shepherd dogs

There really isn't anything new to report about this disease other than an update on therapy. The disease is easy to diagnose. Previous therapies have been cutting (surgical excision), frying (laser) or freezing (cryotherapy), with results that have been less than spectacular. Current research indicates however that this disease responds to immune-suppression. Initially use of high dose corticosteroids and a high fiber diet was shown to cause resolution of the problem in 33% of dogs and improvement in 33% of cases. This is an economical therapy worth trying prior to more invasive or expensive treatments. Further research showed Cyclosporine to be a valid therapeutic agent for this disease. Remission of fistulas was achieved in 85% of dogs treated after 16 weeks of therapy. Recurrence is common however if treatment is discontinued. Unfortunately this is a very expensive therapy that few can afford. Blood concentrations of Cyclosporine do need to be measured to determine the optimal dose, as there is wide variability in absorption between patients. Recently the use of topical Cyclosporine has shown promise. Remission occurs, however long term therapy is needed, as recurrence is common. Topical tacrolimus ointment (1%) has also shown promise with 30% remission and 30% improving significantly. Combination therapy may also be appropriate to limit costs by using oral cyclosporine initially to induce improvement and then following up with other therapies to complete healing.

Exercise induced collapse in labrador retrievers

Exercise induced collapse is being seen in younger adult Labrador Retrievers. Most are field trial dogs. Age of onset is variable, but usually around 7 months to 2 years. This may be because this is the age that they begin training. It could well be genetic in origin since some of the affected dogs are closely related to each other. Most of these dogs would make excellent working dogs since they are energetic with considerable retrieving drive.

Clinical signs generally only develop with strenous exercise. They begin to become weak and wobbly and then may collapse. Collapse may be sporadic or may occur almost every time the dog is worked hard. After stopping of exercise, the dogs generally will get worse for another 4 or 5 minutes. Signs usually begin in the rear legs and then progress forward. Dogs who are collapsed will consistently lose their patellar reflexes, and some develop increased extensor tone of their forelimbs. Some (but not all) affected dogs will seem to experience a loss of balance. They are conscious and usually trying to continue exercising. After 10 to 20 minutes of rest, the dogs return to normal. NOTE: A few affected dogs have died while exercising or during an episode of exercise-induced collapse, so an affected dog's exercise should ALWAYS be stopped at the first hint of incoordination or wobbliness

References

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