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Granulomatous Meningoencephalitis

Client Handout

The Pet Health Care Library

Most people have never heard of GME or any other form of central nervous system reticulosis until they have a dog with progressive neurologic disease. Frequently what the owner is told is that GME is an inflammatory disease that acts in many ways like cancer and that very little is known about it. We understand how frustrating this is and hope to provide a summary of this condition in an understandable form.

Inflammation involves the infiltration of normal tissues by cells of the immune system. These cells are like the armed police of the body. They go to the area where they are called and release destructive biochemicals with the goal of obliterating an area of invasion by infectious organisms or of dead or diseased tissues.
Granulomatous inflammation involves infiltration by cells called mononuclear cells. These cells normally engulf and destroy debris. In GME, these cells form cuffs around the blood vessels of the brain and spinal cord (mostly in the white matter). The cuffs join at adjacent vessels forming actual masses/nodules.

We do not know were the cells of infiltration come from in GME. They may come from the circulation or come from local proliferation of cells already present.

The term reticulosis refers to several inflammatory infiltrative diseases of the nervous system including GME. In all cases, the infiltration is associated with blood vessels. In some cases, the infiltration is malignant (cancerous) and in others, such as GME, the infiltration is benign (non-cancerous). There is a very fine distinction between benign and malignant, however, and terminology in the literature is not clearly defined. Terms such as GME, malignant GME, inflammatory GME, and reticulosis overlap.

Types of GME

There are three types of GME: focal (limited to one location in the nervous system); disseminated or multifocal (involving many locations in the nervous system); and ophthalmic (involving the optic nerve/eye). A patient may have more than one type.

The focal type of GME typically has a slower onset (3-6 months) while the disseminated form is more rapid (2-6 months). Obviously, the disseminated form has a larger variety of signs within the same patient. The ophthalmic form most commonly shows up as sudden, generally permanent blindness. It can affect one or both eyes. The disseminated form has a particularly poor prognosis; in one study the median survival time after diagnosis was 8 days, a testament to the rapid progression and severity of this condition.

Making the Diagnosis

A basic blood panel/ urinalysis is important for any sick pet (not to mention for the monitoring of healthy elderly pets). A basic blood panel and urinalysis form the foundation of evaluation and determination of what medication can be used, and what other systems must be considered.

Tapping of the cerebrospinal fluid (spinal tap) is very helpful in the diagnosis of GME. General anesthesia is required to tap fluid from the nervous system and the procedure is not entirely without risk. Still, the cells of GME should be findable in the fluid. Prior administration of cortisone-type medication may reduce the cells found in the tap and must be considered in the interpretation of the tap.

This kind of testing also helps in ruling out other conditions that might present similarly:

- Viral encephalitis (including canine distemper and rabies) though further spinal fluid testing is generally more revealing.
- Parasitic encephalitis (such as toxoplasmosis)
- Fungal encephalitis (such as caused by Cryptococcus neoformans)
- Breed specific inflammatory disease (pug encephalitis, etc.)

MRI (magnetic resonance imaging) is able to image the brain in such detail that it is considered nearly a confirming test for GME. CT (CAT scanning) is not as definitive. If the diagnosis is felt to still be questionable after spinal tap, MRI should be considered despite expense.

The only way to confirm GME with 100% certainty is by biopsy though, obviously, diagnostics do not get any more invasive than brain surgery. For this reason, confirmation is frequently post-mortem.

Treatment

Immune-suppression with corticosteroids (such as prednisone) remains the center of therapy for GME. Once the disease is controlled, one may begin to gradually drop the steroid dose until the minimum dose required to control the disease is reached. This process can be expected to require 4 months or so. It is unusual for a patient to be able to fully discontinue medication but at least stronger immunosuppressive agents are rarely required.

A new drug called leflunomide has been released to treat immune-mediated diseases of a type that includes GME. This is a relatively expensive treatment, which as limited use but it remains as possible alternative to corticosteroid use for dogs that do not tolerate corticosteroid side effects. Procarbazine, a chemotherapy agent, is another emerging medication for the treatment of this condition.

If the GME is focal or localized, radiotherapy may be helpful. Radiation of the head or face can lead to infarction (abnormal clotting), which can in turn lead to seizures for periods as long as 5 to 6 months after therapy. Cataracts and dry eye are common results of radiotherapy should the eye be included in the field of radiation. That said, many cases of focal GME have enjoyed complete resolution after radiation.

If seizures have been a manifestation of GME, either disseminated or focal, medication will be used to control the seizures. Anti-seizure medication is the same regardless of the cause of the seizures so something specific for GME seizures is not needed.

Ophthalmic GME also uses oral corticosteroids for therapy but may also employ topical ones. If glaucoma results from GME then therapy for this is necessary. Again, therapy for this results of GME is addressed in a standard way; no specific GME glaucoma therapy is needed.