

GRANULOMATOUS MENINGOENCEPHALOMYELITIS

(INFLAMMATION OF THE BRAIN, SPINAL CORD, AND MENINGES)

BASICS

OVERVIEW

- “Granulomatous” refers to inflammation characterized by the presence of nodules; “meningoencephalomyelitis” is inflammation of the brain, spinal cord and their surrounding membranes (the membranes are known as “meninges”)
- Granulomatous meningoencephalomyelitis (also known as “GME”) refers to an inflammatory disease that affects the central nervous system; it can be localized, widespread, or involve multiple locations
- Confirmation of the disease diagnosis is only possible through microscopic analysis of the affected nervous tissue, obtained through biopsy
- Granulomatous meningoencephalomyelitis (GME) is the most accepted and recognized central nervous system inflammatory disorder in the dog
- Many less serious viral and idiopathic disorders frequently are diagnosed erroneously as being “granulomatous meningoencephalomyelitis (GME)” (“idiopathic” means the disease is of unknown cause)
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae

GENETICS

- A genetic factor is not proven

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Historically, granulomatous meningoencephalomyelitis (GME) has been considered a disease of young to middle-age, toy-breed dogs (especially terriers and poodles); however, any medium and larger breed can be affected

Mean Age and Range

- Mean—5 years of age
- Range, 6 months to 10 years

Predominant Sex

- Both sexes can be affected, but females have slightly a higher number of cases than do males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs depend on the form of the disease and location of nerve tissue involved
- The cerebral form of disease (involving the brain) frequently results in seizure activity

Ocular form

- Sudden (acute) onset of blindness, with dilated and unresponsive pupils; the “pupil” is the circular or elliptical opening in the center of the iris of the eye; the “iris” is the colored or pigmented part of the eye

Focal form

- Cerebral lesions (involving the brain)—disorientation, behavioral changes, seizures, blindness, compulsive circling, head pressing
- Brainstem lesion (involving the part of the brain that is connected to the spinal cord that controls functions like breathing and heart rate)—drowsiness or sleepiness (known as “sommolence”), cranial nerve deficits (most commonly facial nerve deficit and lack of control of equilibrium, balance, and orientation [known as “vestibular dysfunction”]), one-sided weakness or partial paralysis (known as “hemiparesis”)
- Spinal cord lesion—weakness or partial paralysis of all four legs (known as “tetraparesis”) for lesions in the spinal cord involving the area of the first cervical vertebra through the fifth cervical vertebra (C₁ through C₅) or the sixth cervical vertebra through the second thoracic vertebra (C₆ through T₂) or weakness or partial paralysis of the rear legs (known as “paraparesis”) for lesions in the spinal cord involving the area of the third thoracic vertebra through the third lumbar vertebra (T₃ through L₃) or the fourth lumbar vertebra through the second sacral vertebra (L₄ through S₂) and a wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)

CAUSES

- Unknown

RISK FACTORS

- Unknown
- Some dogs develop clinical signs within 5 to 10 days of receiving vaccinations

TREATMENT

HEALTH CARE

- Stable patients can be discharged with recommended treatment
- Inpatient—for severely affected dogs; monitor patient closely for progression of nervous system deficits
- Intravenous fluids for patients that have lack of appetite (known as “anorexia”); fluids should be administered carefully to avoid overhydration and worsening of fluid build-up in the brain (known as “cerebral edema”)
- Provide a padded cage for dogs with lack of control of equilibrium, balance, and orientation (vestibular dysfunction), severe dementia or seizure activity
- Recumbent patients should be turned frequently (every 4 hours) to avoid pressure sores and lung congestion

ACTIVITY

- Depends on the severity of disease and location of nerve tissue involved
- Patients with a wobbly, incoordinated or “drunken” appearing gait or movement (ataxia) should be confined to a padded cage to avoid injury

DIET

- Ensure adequate caloric intake

MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids—dexamethasone followed by prednisone; the steroid dose should be adjusted by your pet’s veterinarian, according to response to treatment and side effects—the goal is to find the dose that keeps the clinical signs controlled with minimal side effects
- To prevent gastrointestinal ulceration, administer famotidine (an H₂-blocker to reduce stomach acid) with the steroid therapy
- Phenobarbital to control seizures
- Chemotherapeutic drugs used to decrease the immune response—azathioprine, cytosine arabinoside, or cyclophosphamide
- Medications to decrease the immune response (known as “immunosuppressive drugs”)—cyclosporine, leflunomide
- Radiation therapy is an alternative treatment in the focal form of the disease when other therapies have failed (the diagnosis should be confirmed by microscopic evaluation of tissue samples obtained by biopsy, before starting radiation therapy)

FOLLOW-UP CARE

PATIENT MONITORING

- Repeat nervous system examination periodically (every 2 to 4 weeks)
- Evaluate blood work (complete blood count [CBC] and biochemical profile) regularly to monitor for low white-blood cell count (known as “leukopenia”), low platelet count (known as “thrombocytopenia”), and liver and kidney function
- Monitor urine in patients on long-term steroid treatment—protein in the urine (known as “proteinuria”) or urinary tract infection are frequent consequences of long-term steroid treatment

POSSIBLE COMPLICATIONS

- Deterioration of clinical signs, despite aggressive treatment
- Repeated or prolonged seizure activity (known as “status epilepticus”)
- Dementia
- Brain pushes downward in the skull and herniates through the opening that leads to the neck (known as “tentorial herniation” or “brain herniation”) and death

EXPECTED COURSE AND PROGNOSIS

- Not all patients with central nervous system inflammatory disease have a poor prognosis
- Granulomatous meningoencephalomyelitis (GME) has been characterized as a fatal disease without enough scientific evidence; it is uncertain if dogs that survive inflammatory central nervous system disease had GME, as brain biopsies rarely are obtained to provide a definitive diagnosis

KEY POINTS

- Clinical signs overlap significantly among different central nervous system inflammatory diseases—a diagnostic work-up is very important
- Mortality rate for granulomatous meningoencephalomyelitis (GME) is clearly biased by the severe cases that go to postmortem examinations; brain biopsies rarely are obtained during life to provide a definitive diagnosis and to provide a more accurate mortality rate
- Some dogs with central nervous system inflammatory disease can be treated successfully; however, long-term treatment and client compliance are required
- Steroid therapy may be necessary for a prolonged period or for life

