WHAT IS YOUR DIAGNOSIS?

A 10-month old neutered female Bichon Frise was presented to the Emergency Medicine Service for investigation of a two day acute onset of cluster focal seizures, preceded by a single generalised tonic-clonic seizure. One hour prior presentation, a cluster of four partial seizures had been witnessed.

Physical examination was unremarkable. However, on neurological examination, mildly decreased right nasal sensation was detected, with no other abnormalities, which could not be excluded as a post-ictal event.

1) What are your differential diagnoses for seizures in a young dog?

2) What diagnostic evaluations would you perform in this dog?

3) How would you treat this dog?
1) The differential diagnoses for seizures in a young dog include (non-exhaustive list):

- **V**: vascular (coagulopathy, thromboembolic disease);
- **I**: infectious (tick-borne diseases, distemper, toxoplasmosis/neosporosis), inflammatory (granulomatous/necrotizing/eosinophilic encephalitis);
- **T**: trauma, toxic (lead, organophosphates, ethylene glycol);
- **A**: anomalous (hydrocephalus, cortical dysplasia, lissencephaly);
- **M**: metabolic (hepatic/uraemic encephalopathy, hypoglycaemia, hypo(hyper)natraemia, hypokalaemia, hypocalcaemia);
- **I**: idiopathic (genetic – formerly idiopathic epilepsy, unknown – formerly probable symptomatic/cryptogenic);
- **N**: neoplastic (lymphoma, malformation tumour, astrocytoma);
- **D**: degenerative (storage diseases, mitochondrial encephalopathies).

2) Diagnostic investigations included:

- **Routine haematology and serum biochemistry**
  - Revealed mild neutrophilia and monocytosis, compatible with an inflammatory leukogram. No abnormalities were seen on serum biochemistry.
- **Serum bile acids stimulation test, blood ammonia and abdominal ultrasonography**:
  - The combination of these did not support the presence of a portosystemic shunt.
- **Toxoplasma sp and Neospora sp serology**:
  - Was negative for both.
- **Brain MRI**:
  - Revealed a lesion in the left frontal cerebral cortex, oedematous and contrast enhancing, affecting the white matter and the grey matter to a lesser extent, and exerting a mass effect. Mild adjacent meningeal contrast enhancement was also suspected.
- **Cerebrospinal fluid (CSF) analysis:**
  - Revealed a mild mononuclear pleocytosis with 82% of large mononuclear cells and 18% of lymphocytes, with no infectious agents seen.

The combination of these findings was suggestive of a meningoencephalitis of unknown origin, most likely **necrotising meningoencephalitis**.

3) **Treatment:**
- **Emergency seizure management:**
  - This was performed with two intravenous diazepam boluses of 0.5mg/kg, continued with a constant rate infusion of midazolam at 0.3mg/kg/h. In addition, a loading dose of intravenous phenobarbitone was started at 4mg/kg given at each seizure (no more than every 30min) up to a total dose of 12mg/kg (maximum recommended: 24mg/kg).

- **Meningoencephalitis management:**
  - Immunosuppression was started with a combination of cytosine arabinoside (cytarabine) at 50mg/m² SC q12h for 48h and prednisolone 1mg/kg PO q12h.
  - A maintenance dose of phenobarbitone 3mg/kg PO q12h was prescribed, aiming to be tapered and stopped after 2-4 weeks if no seizures observed.
  - Clindamycin was prescribed at 12.5mg/kg PO q12h, while protozoal diseases serology results were pending.

This dog is still alive and well over one year from initial diagnosis, and her cytarabine cycles have been gradually decreased from every three weeks to every six weeks (monitoring haematology two weeks after each cycle), as well as its prednisolone doses from 1mg/kg to 0.2mg/kg PO q12h.

**Discussion:**

Inflammatory meningoencephalitides commonly affect the canine central nervous system, behaving as immune-mediated disorders. They include several different subtypes, which can only be definitively distinguished by histopathological analysis, namely granulomatous...
meningoencephalomyelitis (GME), necrotizing meningoencephalitis (NME)/leukoencephalitis (NLE), and eosinophilic encephalitis, amongst others. They tend to be seen more commonly in young to middle-aged small-breed dogs, typically presenting with acute and progressive clinical signs. These vary depending on the localisation of the lesion within the central nervous system (focal, multifocal or diffuse). Presumptive diagnosis is usually obtained with a combination of MRI and CSF analysis, and exclusion of infectious diseases. Treatment is usually based in long term and gradually tapering of immunosuppressive drugs, namely corticosteroids used in conjunction with cytarabine. Other drugs have been used like procarbazine, ciclosporin, lomustine, azathioprine, and even radiation therapy. Response to treatment should ideally be monitored by repeat MRI and CSF analysis. Although prognosis is poor long term, survival times do vary from days to years, usually being worst with NME/NLE cases, diffuse/multifocal or brainstem lesions, and seizures on presentation.

References:


