Teleneurology report

Date: 18/10/2019
Referring veterinary surgeon: [redacted] **Hospital:** [redacted]
Email address: [redacted]
Patient name and surname: [redacted]
Species (canine/feline): Dog **Breed:** CKCS **Age:** 10y **Sex:** Male
Body areas scanned and charged: MRI brain **Service required:** Standard 1-3 days

Relevant clinical history, clinical findings and diagnostic test results:

Presented on the 15th of October as an emergency consultation for further investigation and management of acute onset of seizures. The owner described the episodes of seizures as episodes of hypersalivation, mandibular trismus, and increased muscle tone of the thoracic limb. No other autonomic signs were described and no lack of consciousness was described. Treatment with diazepam and propofol had been administered, but seizure activity remained. A total of 16 episodes were reported within less than 24 hours. As part of his previous medical history he had been receiving treatment for cardiac disease after having several episodes of possible collapse. [redacted] remain hospitalized for 48 hours receiving supportive treatment based on fluid therapy and antiepileptic medication. A loading dose of phenobarbital was administered resulting in initial control of the seizure activity. However, 3 more episodes of partial seizures were observed and levetiracetam was added to the therapy.

Report

Thank you for submitting this MR study of the brain on [redacted].

Main findings

On sagittal images (image 1), there is moderate crowding of the caudal fossa causing the caudal margin of the cerebellum to become flat rather than convex. The cerebellar vermis is mildly herniating through the foramen magnum with little CSF around the neural structures. In addition to that, there is moderate 'kinking' of the myelencephalon and mild dilation of the central canal in the cranial cervical spinal cord. There is a small caudo-dorsal expansion of the 3rd ventricle with mildly enlarged quadrigeminal cistern. The lateral ventricles are moderately enlarged.

There is a fairly sharply defined homogeneous intra-axial lesion in the left medial part of the caudal thalamus. This lesion is T2W and GRE hyperintense, T1W mildly hypointense and FLAIR isointense/slightly hypointense (images 2 & 3). It is hyperintense on dDWI and hypointense on eDWI (image 4). There is no associated contrast enhancement or mass effect.

Conclusion & recommendations

- Suspected non-hemorrhagic left paramedian thalamic infarct
- Mild Chiari-like malformation and mild hydromyelia
- Mild supracollicular fluid accumulation

Both Chiari-like malformation and supracollicular fluid accumulation are common in the breed and likely incidental findings here. The lesion in the caudo-dorsal and medial part of the thalamus is in the area supplied by the caudal

perforating arteries and paramedian branches of the caudal cerebral artery. Its imaging characteristics and location are strongly suggestive of a paramedian thalamic infarct. The hyperintensity on dDWI and hypointensity on eDWI would be suggestive of recent infarct.
The acute onset of cluster of neurological paroxysmal events here would be suspicious of a vascular event. CKCS are predisposed to brain infarct and based on our previously published studies on the subject an associated medical condition can only be identified in about 50% of affected dogs. Commonly associated/causative diseases include hyperadrenocorticism, hypothyroidism, chronic kidney disease, protein-losing enteropathy and nephropathy, systemic hypertension, heart disease and more sinister causes such as neoplastic disease which can be associated with embolic process but also hypercoagulability syndrome. The presence here of concurrent structural heart disease and mild hypercoagulability may be predisposing factor to this suspected brain infarct and/or future infarct.
There is no specific treatment for brain infarct beyond supportive care and management of suspected associated/causative disease(s) if one(s) can be identified. With that in mind, I would suggest continuing with pimobendane and clopidogrel as well as symptomatic anti-epileptic medication. In case [redacted] does not experience any more seizure-like event within the next 6 months, I would suggest to very slowly take him off phenobarbitone over a three-month period. Should [redacted] experiences further seizure-like event over the next 6 months (or during the withdrawal period), I would suggest keeping him on phenobarbitone as life-long therapy. I would be interested to review a video of any future seizure-like event to help better characterize their nature.

I hope this report is helpful. Do not hesitate to contact me if I can be of any further help.

Best regards

Laurent Garosi
DVM, FRCVS, Dip ECVN
RCVS & EBVS® European Specialist in Veterinary Neurology

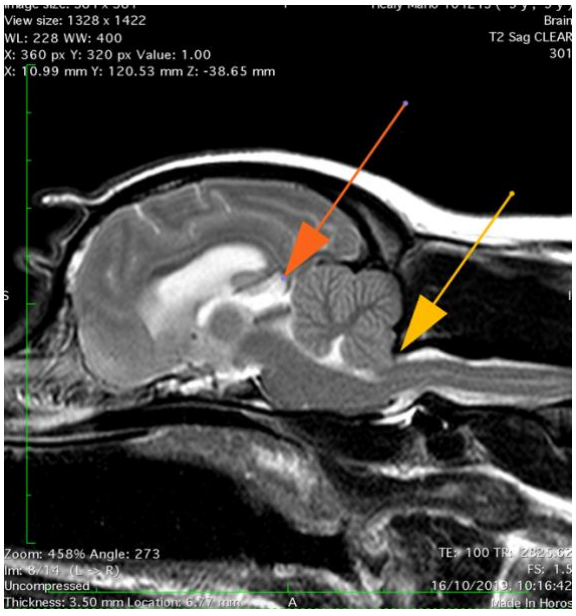

Image 1 – Sag T2W brain and cranial cervical

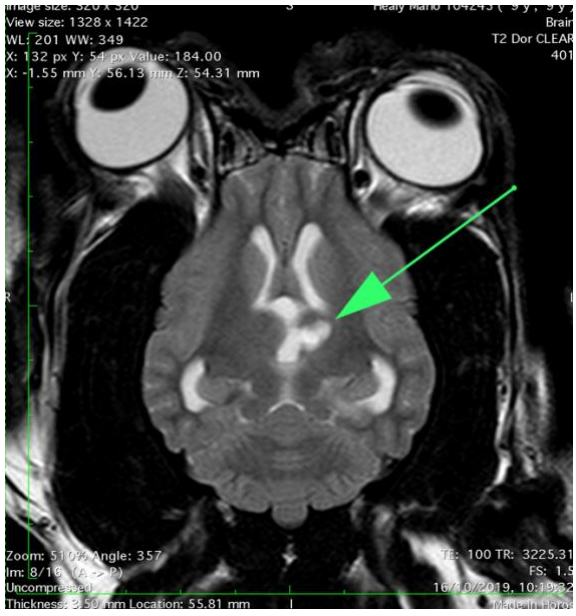

Image 2 – Dors T2W brain

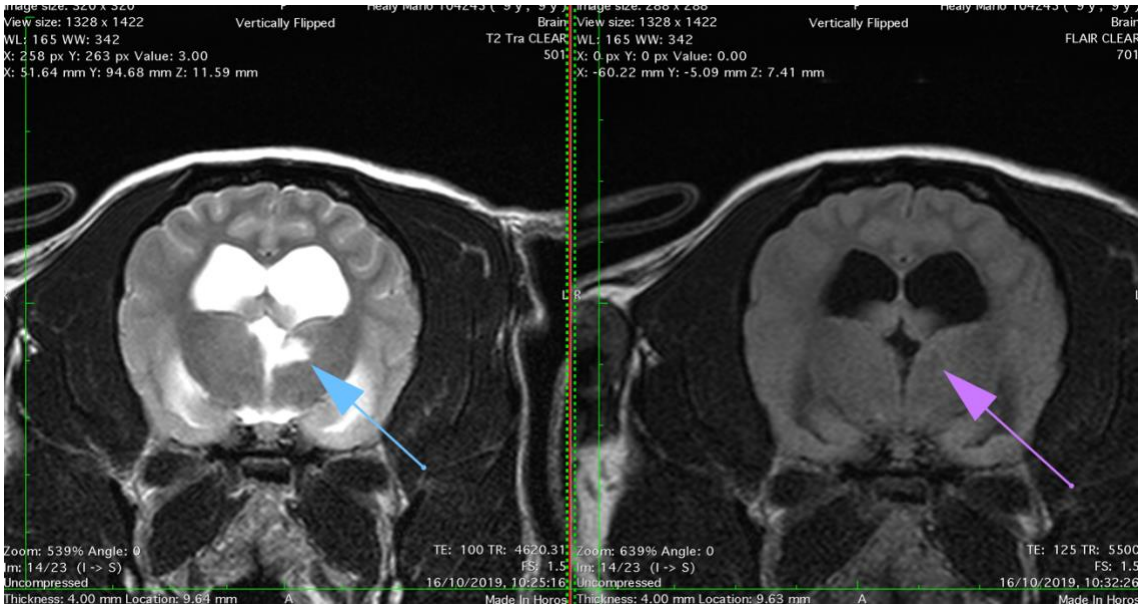

Image 3 – Trv T2W (left) and FLAIR (right) caudal thalamus

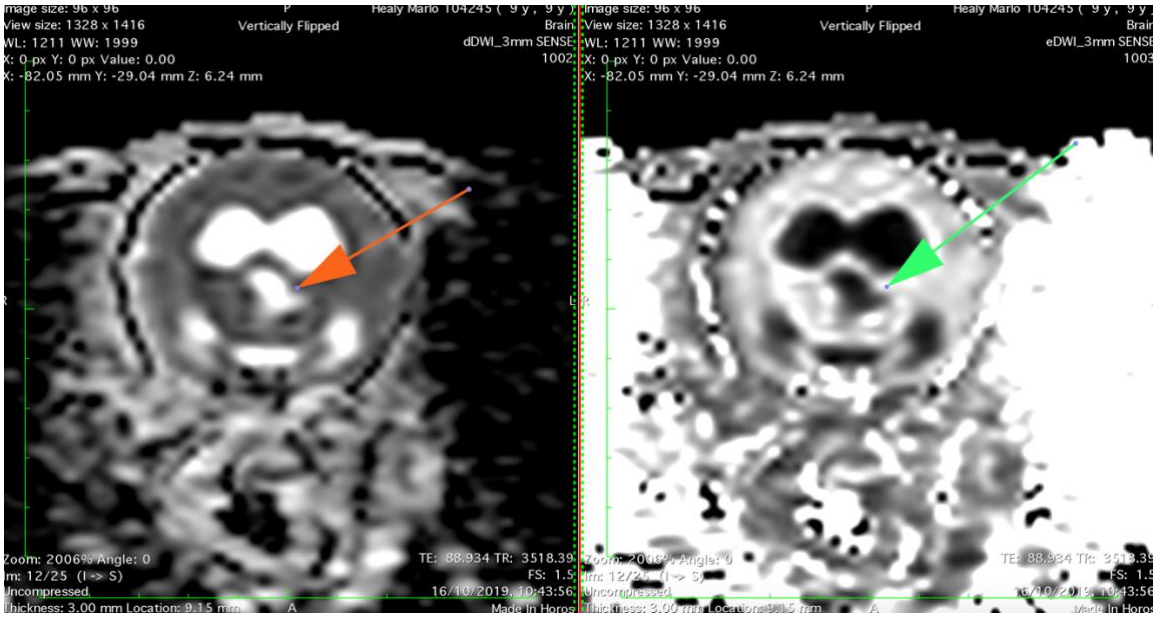

Image 4 – Trv dDWI (left) and eDWI (right) caudal thalamus