NeuroNews

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Ischaemic stroke in dogs

NeuroNews is an informal, but hopefully informative, newsletter covering a range of clinically relevant neurological topics. Cases detailed in these articles are patients referred to *NeuroVet* by veterinarians in South Australia, western Victoria/NSW and the Northern Territory.

Cerebrovascular accident ("stroke") may be broadly classified as ischaemic or haemorrhagic. In the canine patient, ischaemic stroke appears to be much more common than primary haemorrhagic stroke. Intracranial haemorrhage secondary to CNS disease (including tumours, trauma, meningoencephalitis and congenital vascular anomaly) is well documented in the veterinary literature, but will not be discussed in this article.

The neurological presentation following acute infarction is highly variable, depending on the location of the lesion, as exemplified by the two cases detailed below, but intracranial vascular anatomy dictates that there is usually clear lateralisation of signs. Following onset of signs, the neurological status of the patient typically does not deteriorate dramatically, in the absence of secondary complication. If signs abate within twenty-four hours, strictly speaking the problem should not be termed a stroke (which implies a degree of parenchymal necrosis) and, if suspected to be vascular in aetiology, should be referred to as a transient ischaemic episode.

Conditions thought to predispose to the development of ischaemic stroke include hyperadrenocorticism, chronic renal disease, hypothyroidism and endocarditis. However, in around half of recorded cases, no predisposing cause is defined antemortem.

The prognosis in ischaemic stroke is better in those patients in which no relevant underlying condition is found; most making a good recovery within a few weeks, requiring supportive care only. There is ongoing debate regarding management of hypertension in the acute phase of ischaemic stroke; however, in most cases, efforts should be directed at maintaining arterial BP within the normal range. If underlying disease is diagnosed, appropriate therapy is essential to optimise prognosis. The potential benefits of thrombolytic therapy and neurocytoprotective agents in canine ischaemic stroke are still undetermined in the clinical setting. However, if a thromboembolic syndrome is confirmed, low-dose aspirin or clopidogrel therapy may be utilised, in an attempt to reduce the risk of subsequent embolism. Recurrence rate is significantly lower in the absence of predisposing conditions.

Prior to the advent of advanced imaging, the diagnosis of stroke was typically made post-mortem. Although standard CT has good sensitivity in detecting intracranial haemorrhage, it is less reliable in defining cerebral ischaemia. MRI is widely considered to be the preferred diagnostic tool when it comes to evaluation of suspected stroke in the small animal patient (*Cook L, 2016: Clinical Use of Neuroimaging in Small Animals: CT and MRI)*, assisting the clinician in defining the type of stroke, thereby facilitating provision of appropriate management and providing confidence that advice given to clients is as accurate as possible.

CASE 1

8 yo FS large breed dog

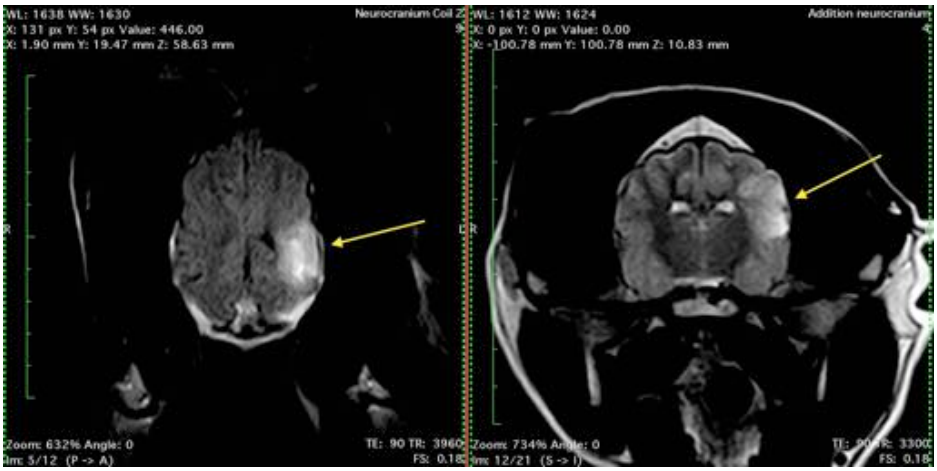
HISTORY
Peracute onset of dullness and circling to the left, two weeks previously; routine blood screen had returned unremarkable results. The dog was referred to *NeuroVet* as, although her status had improved, there were ongoing concerns regarding visual acuity.

NEUROLOGICAL ASSESSMENT
The dog appeared alert and was strongly ambulant, with no evidence of ataxia or paresis. Proprioception and myotactic and flexor reflexes appeared normal. Cranial nerve assessment was unremarkable, other than for the absence of right-sided menace response, in

the presence of normal palpebral and corneal reflexes. Obstacle courses were negotiated well with the right eye blindfolded, but less confidently with the left eye blindfolded.

INTERPRETATION
The above findings are consistent with a left-sided, post-chiasmic, optic pathway lesion.

FURTHER INVESTIGATION
Neurocranial MR imaging was performed utilising GE, T2 and pre- and post-contrast T1 and FLAIR sequences in all planes. This revealed the presence of a T2 /FLAIR hyperintense, T1 isointense and contrast enhancing lesion in the left parietal/rostral occipital lobes, with predominantly grey matter involvement. The strong T2 hyperintense signal was consistent with oedema. There was no evidence of the "mass effect" typically seen in intracranial neoplasia:



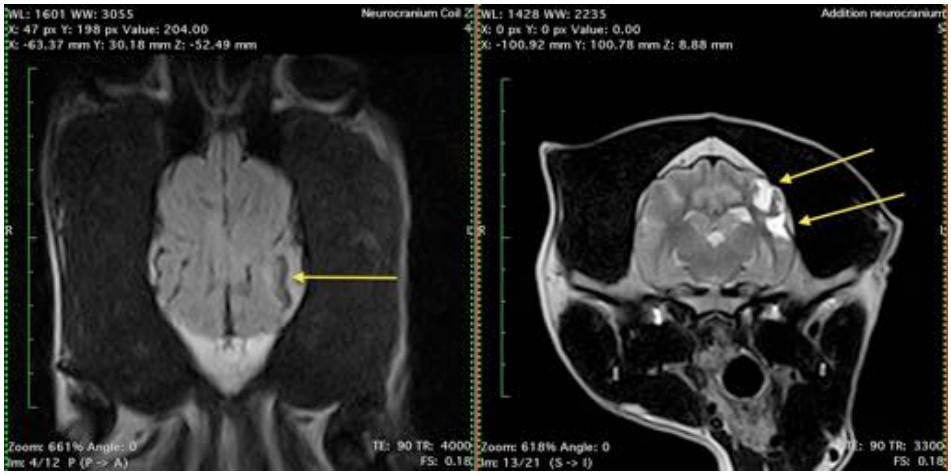
FLAIR Dorsal *T2 Axial*

CSF was obtained via lumbar centesis: there was no pleocytosis and protein level was within normal range. Results of CSF Neuro-PCR, protozoal and fungal immunology and culture were all negative.

DIFFERENTIAL DIAGNOSIS
- Focal inflammatory disease
- Focal cerebrovascular disease

MANAGEMENT
Given the initial possibility of inflammatory disease, empirical treatment (prednisolone and initially clindamycin) was administered, pending repeat imaging four weeks later. The dog's status improved significantly over this period.

Follow-up images revealed marked reduction in oedema and grey matter cavitation, consistent with post-infarction necrosis. The affected region was considered to be within the territory of the left middle cerebral artery:



FLAIR Dorsal *T2 Axial*

Free T4ED / TSH assay, urinalysis and culture and urine protein-creatinine ratio, performed two weeks after withholding of medications, all returned unremarkable results. The cause of the infarction therefore remained undefined.

The dog made continued gradual improvement and is clinically normal, other than for a mildly reduced right-sided menace response, fifteen months post-onset.

CASE 2

5 yo FS large breed dog

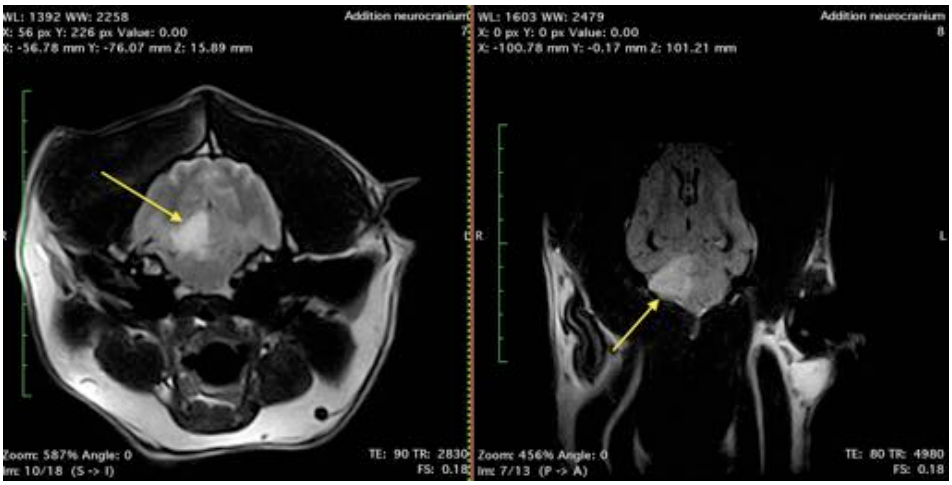
HISTORY
The dog was found to be unable to rise the evening prior to referral to *NeuroVet*. There had been no signs of abnormality when she was last observed, around twelve hours previously.

NEUROLOGICAL ASSESSMENT
She appeared alert, but restive, and was exhibiting non-ambulatory quadriparesis and profound generalised ataxia, with no meaningful voluntary movement in the limbs other than the LFL. Proprioception was absent in all but the LFL. There were marked UMN signs in the right-sided limbs. There was also a tendency to roll. Nociception appeared normal.

Neurocranial assessment revealed dorsolateral strabismus in the right eye and variable positional nystagmus. Menace response was reduced in the right eye. Gag reflex was deemed weak.

INTERPRETATION
Predominantly right-sided, caudal fossa encephalopathy with marked cerebellar component. In this case, the menace deficit was deemed likely to be secondary to ipsilateral cerebellar disease, rather than an indication of impairment to visual pathways, the cerebellum being facilitatory to the blink component of the menace response. Given the acute onset, the differential was thought likely to be headed by traumatic and vascular syndromes.

FURTHER INVESTIGATION
Following administration of mannitol, MR imaging was performed, utilising GRE, T2 and pre- and post-contrast T1 and FLAIR sequences in all planes. This revealed the presence of a T2/FLAIR hyperintense and T1 iso- to hypointense, non-enhancing lesion in the right rostral cerebellum:



T2 Axial *FLAIR Dorsal*

DIAGNOSIS
Sequence characteristics, wedge shape and sharp midline margin are all consistent with cerebellar infarction secondary to occlusion of flow through the right rostral cerebellar artery.

MANAGEMENT
Regrettably, in this instance the owners requested euthanasia.

As always, I would be delighted to discuss investigation and management of any neurological cases with you.

Best wishes,

Ian Douglas

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