Case reports: neurology at NOAH

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CASE 1

HISTORY AND CLINICAL SYMPTOMS

A 10-year-old, 17kg, neutered female Spaniel-cross presented barely responsive with sudden-onset seizures and right head tilt. The dog had a seizure eight hours previously with no prior history of seizures. The seizure lasted, on average, five minutes and was mild and generalised. Further clinical examination was unremarkable apart from slow proprioception on the right side.

INVESTIGATION

The dog was classified using the Glasgow Modified Coma Scale at a score of 16, otherwise summarised as good (Table 1). Full biochemical and haematologic blood profiles including electrolytes and bile acids were normal. Ophthalmology assessment was normal as was urinary analysis. Magnetic resonance imaging (MRI) of the brain was performed including T2, T1, fluid-attenuated inversion recovery (FLAIR) and contrast studies. In the FLAIR study, there was a hyperintense area in the right cerebellum (Figure 1) covering a significant proportion of the hemisphere. There was no contrast uptake and no mass effect. Hyperintensity was also observed on the T2 images. Cerebrospinal fluid (CSF) analysis was normal.

DIAGNOSIS

Obstruction of the right rostral cerebellar artery causing a significant area of infarction.

TREATMENT

Hyperbaric oxygen treatment (Figure 2) was initiated immediately along with anticonvulsant medication.

OUTCOME

The dog stabilised with no current recorded seizures for two months and is currently off all medications. A mild head tilt is still present.

DISCUSSION

Occlusion of brain arteries in dogs is not thought to be common. This may change as more canine brain MRI studies are completed and published. The consensus among the science community is that the occlusion is caused by a thrombus. Symptoms can be diverse depending on the area and size of the infarct. There are parallels with human stroke incidents and the neuropathological changes in dogs with cerebral infarcts/stroke are very similar. Treatment is based on multidisciplinary care and many dogs do recover eventually. A number of studies in human medicine have found that hyperbaric oxygen treatment can improve a patient's chance of recovery following a stroke. Even though haemoglobin at normal atmosphere pressure is 99.9% oxygen saturated, by applying increased pressure in an oxygen-rich chamber allows for more oxygen to dissolve into the plasma directly. Furthermore, the oxygen in the plasma can diffuse deeper into tissues than from haemoglobin alone. An infarct is composed of two parts, the umbra (central core) and the penumbra (outer shell). Stroke is a common disease in humans and the accepted wisdom is that after six hours, cell death occurs in the umbra but the penumbra contains cells that are damaged and a diminished gradient of oxygenation exists for a long time. Therefore, in an acute scenario, time to treatment is critical. It has been shown in some human studies that hyperbaric oxygen therapy can lead to recovery of penumbra even well after the acute phase has passed. This has been termed the idling neuron phenomenon, whereby neurons are alive but not functioning. The brain is particularly oxygen sensitive and increased oxygen levels will reduce brain oedema and is neuroprotective.

CASE 2

HISTORY AND CLINICAL SYMPTOMS

A two-year-old, 4kg, neutered female Bichon Frise presented with head tilt and ataxia. The dog had been in this state for up to a few days. She was not bright, alert or responsive. The left head tilt was not reducible, and the dog fell to the left when she attempted to walk. There was mild nystagmus and significant proprioceptive deficits in all four limbs. More in depth examination of the ears, eyes and mouth were unremarkable. All other parameters were normal.

INVESTIGATION

A Glasgow Modified Coma Scale of 14 was calculated for this case, which categorises the situation as guarded. Otoscopy under sedation was normal. Blood, urine and CSF tests were all within normal value ranges. Computed tomography (CT) scan revealed a left-sided fractured skull extending from the base of the temporal one to dorsal aspect of the parietal bone (Figure 3). There was no evidence that the large bone fragment was mobile or liable to depress the brain further.

DIAGNOSIS

Left-sided trauma causing depressed skull fracture and brain compression.

TREATMENT

Supportive treatment including precise intravenous fluid (IV) and tube feeding administration was started immediately, along with analgesia. The offer for surgical intervention was declined by the owner. Hyperbaric oxygen therapy was

employed in the absence of a surgical option. Hydrotherapy was used daily. Intensive treatment was continued for 10 days.

OUTCOME

The dog gradually improved and, by 10 days, was able to walk unaided and eat by herself. She still had a head tilt and this remained present at the last check-up.

DISCUSSION

Trauma causing skull fracture in dogs and cats has rarely been reported in the literature. Part of the reason for this paucity of information could be because most of the patients

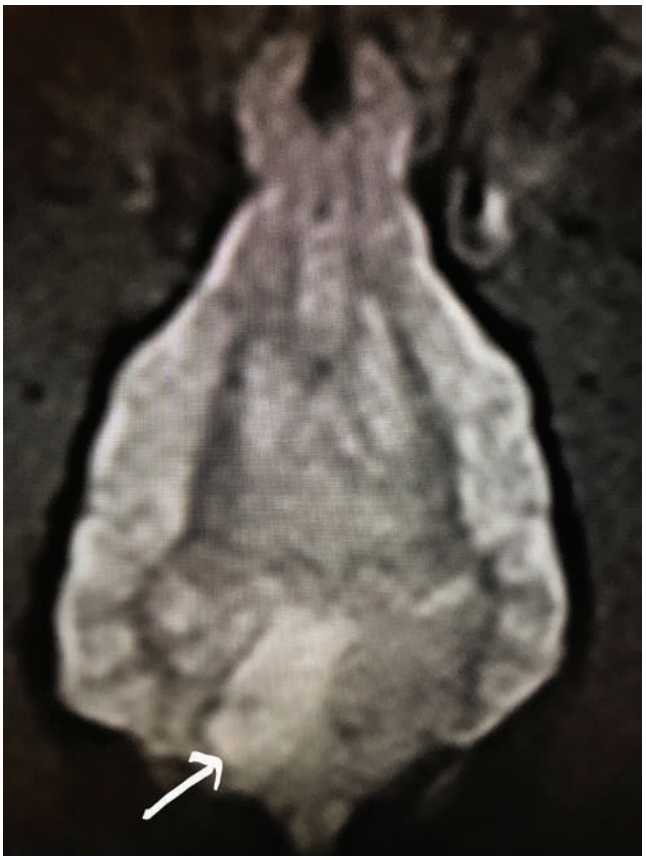

Figure 1: MRI showing infarct lesion

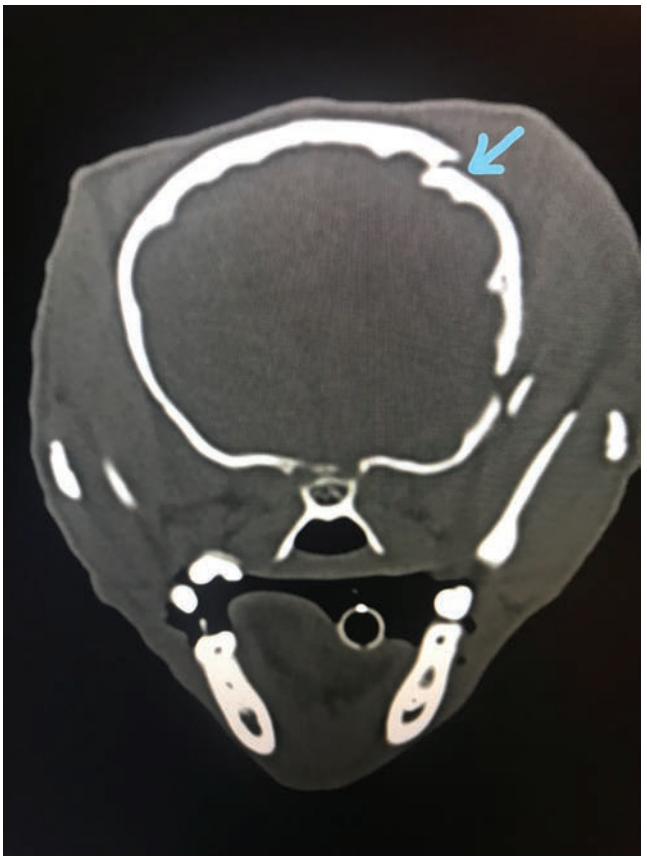

Figure 3: CT scan showing skull fracture

Figure 2: Hyperbaric oxygen chamber

die before treatment is completed or shortly after treatment was initiated. There is some controversy between medical and surgical treatments for traumatic brain injury, and when to opt for one or the other. Traumatic brain injury causes increased intracranial pressure, cerebral oedema, decreased perfusion pressure and damage to the blood brain barrier. Medical management is aimed to lessen the secondary damaging effects of the latter. The objectives with medical management are to maintain blood pressure, provide oxygen, prophylactic anticonvulsants and mannitol use. There is no agreed consensus on the use of corticosteroids in traumatic brain injury in human medicine mainly because it is not certain which cases are oedema caused by vascogenic means or by cytotoxic mechanisms. Many authors assert that corticosteroid use is contraindicated in traumatic brain injury. Only patients with vascogenic oedema may benefit from corticosteroid use. In human neurology any patient with worsening neurological status or increasing intracranial pressure are likely to be selected for surgery. The case presented here was not acute and surgery was declined by the owner. Hyperbaric oxygen therapy provided an opportunity to reduce any residual oedema or inflammation, improve oxygenation of any idling neurons, reduce intracranial pressure and improve cerebral metabolism. Human studies have shown that traumatic brain injury can result in the cerebral metabolic rate of oxygenation being reduced by up to 50%. A Single Photon Emission Computed Tomography (SPECT) studies in human cases of traumatic brain injury have shown improved cerebral blood flow and metabolism following hyperbaric oxygen therapy.

CONCLUSIONS

In both of these cases the primary presentation of head tilt would not be an unusual symptom seen by every practitioner. With the increasing use of MRI or CT imaging the diagnostic possibilities have been significantly expanded. Not every head tilt case has an ear problem. After reviewing many MRI or CT imaging studies of head tilt patients, one as a clinician is surprised by the number of possible cases, heretofore were possibly diagnosed incorrectly. In many cases the clinician's hands are tied and the options are reduced. Hyperbaric oxygen therapy is a relatively inexpensive method of treatment and offers a very useful adjunctive treatment modality in brain and spinal neurology.

References available on request.

GLASGOW MODIFIED	COMA SCALE	
	Symptoms	Score
MOTOR ACTIVITY	Normal gait normal reflexes	6
	Hemiparesis, tetraparesis decerebrate rigidity	5
	Recumbent intermittent extensor rigidity	4
	Recumbent, constant extensor rigidity	3
	Recumbent, constant extensor rigidity and opisthotonus	2
	Recumbent hypotonia of muscles depressed or absent spinal reflexes	1
BRAINSTEM REFLEXES	Normal PLR and oculocephalic reflexes	6
	Slow PLR and normal to reduced oculocephalic reflexes	5
	Bilateral unresponsive miosis with normal to reduced oculocephalic reflexes	4
	Pinpoint pupils with reduced to absent oculocephalic reflexes	3
	Unilateral unresponsive mydriasis with reduced to absent oculocephalic reflexes	2
	Bilateral unresponsive mydriasis with reduced to absent oculocephalic reflexes	1
LEVEL OF CONSCIOUSNESS	Occasional periods of alertness and responsive to environment	6
	Depression or delirium capable of responding but response may be inappropriate	5
	Semicomatose responsive to visual stimuli	4
	Semicomatose responsive to auditory stimuli	3
	Semicomatose responsive to only repeated noxious stimuli	2
	Comatose unresponsive to repeated noxious stimuli	1
TOTAL SCORE	3-8	GRAVE
	9-14	GUARDED
	15-18	GOOD

Table 1. The Modified Glasgow Coma Scale for evaluating and charting brain injury in veterinary medicine.