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Reversible facial nerve paralysis in a cat suspected to be associated with systemic hypertension

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Abstract

Case summary This report describes the appearance of facial nerve paralysis in a 16-year-old hypertensive cat. MRI was helpful in visualising and characterising mesencephalic and facial nerve lesions thought to be induced by hypertension. Neurological signs rapidly resolved under antihypertensive therapy.

Relevance and novel information Systemic hypertension is an important medical condition in geriatric cats causing damage in various target organs, including the brain. Hypertensive encephalopathy is an umbrella term for a multitude of different clinical manifestations of cerebral target organ damage. Facial nerve paralysis secondary to hypertension is recognised in human medicine, particularly in children, but so far has not been reported in veterinary medicine.

Keywords: Blood pressure; Doppler sphygmomanometry; hypertensive; encephalopathy; neurological; central nervous system

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Case summary

A 16-year-old female spayed cat was referred to the division of neurology in order to investigate the cause of paralysis of the right eyelid associated with a corneal lesion. Hyperthyroidism had been diagnosed 2 months previously and the cat had been treated with transdermal thiamazole ointment since then. Four days before presentation to our hospital, blepharospasm of the right eye had been detected by the referring veterinarian and the cat was reported to be neurologically normal. The corneal lesion was detected by the referring veterinarian the day before presentation to our hospital.

On physical examination, the cat was lethargic and a palpable nodule of the thyroid was found, but the vital parameters were within normal limits. Neurological examination revealed right-sided loss of the palpebral reflex, loss of facial expression, drooping whiskers, and normal physiological, and no elicitable pathological, nystagmus. Posture, gait, muscle tone, spinal reflexes and evaluation of the remaining cranial nerves were

unremarkable. The neurological deficits were neuro-anatomically localised to the right facial nerve. Further diagnostic work-up included haematology and comprehensive biochemistry, urinalysis, a fluorescein test, a Schirmer tear test, retinal examination, blood pressure (BP) measurement and MRI of the head. The only laboratory abnormality was a mild anaemia. The serum thyroid concentration (1.4 µg/dl) was within the

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Table 1 Haematological results of a 16-year-old cat with hypertension and facial nerve paralysis

	Result	RI	+7 months
Haematocrit (%)	32	33.45	32
Haemoglobin (g/l)	11.2	11.3–15.5	11.4
Erythrocytes ($\times 10^6/\mu\text{l}$)	8.3	7.0–10.7	8.2
MCH (pg)	13	14–17	14
MCHC (g/dl)	35	33–36	36
MCV (fl)	39	40–48	39
Thrombocytes ($\times 10^3/\mu\text{l}$)	488	180–680	538
Leukocytes	12.7	4.6–12.8	21.7
Segmented neutrophils	9.6	2.3–10.0	18
Eosinophils	0.97	0.1–0.6	0.45
Basophils	0.01	0–0.14	0.03
Monocytes	0.55	0–0.7	0.6
Lymphocytes	1.57	1.1–6.0	2.6

RI = reference interval; MCH = mean cell haemoglobin; MCHC = mean cell haemoglobin concentration; MCV = mean cell volume

Table 2 Biochemistry results of a 16-year-old cat with hypertension and facial nerve paralysis

	Result	RI	+7 months
Bilirubin ($\mu\text{mol/l}$)	<2.5	<3.5	<2.5
Glucose (mmol/l)	5.6	4–9	6.8
Urea (mmol/l)	8.5	7.4–12.6	6.5
Creatinine ($\mu\text{mol/l}$)	95	98–163	95
Protein (g/l)	77	64–80	75
Albumin (g/l)	35	32–42	31
Cholesterol (mmol/l)	3.2	2.6–6.8	3
ALP (IU/l)	23	16–43	20
ALT (IU/l)	29	34–98	29
Lipase (mmol/l)	8	6–21	8
Sodium (mmol/l)	157	150–157	151
Potassium (mmol/l)	4.5	3.8–5.4	3.8
Chloride (mmol/l)	124	113–123	116
Calcium (mmol/l)	2.39	2.40–2.80	2.37
Phosphate (mmol/l)	1.13	0.9–1.8	0.9
T4 ($\mu\text{g/dl}$)	1.4	<3.3	2.5

RI = reference interval; ALP = alkaline phosphatase; ALT = alanine transaminase; T4 = thyroxine

reference interval. The laboratory results are shown in Tables 1–3. BP measured by Doppler sphygmomanometry was severely elevated on a first set of measurements, with an average systolic value of 230 mmHg. To corroborate this finding, a consecutive set of measurements was obtained 6 h later, and the systolic BP was still high at 215 mmHg. The Schirmer tear test was normal. A fundic examination did not reveal any retinal abnormalities. With the fluorescein test, a centrally located corneal lesion was confirmed, and a partial tarsorrhaphy was performed. Cerebral MRI (Ingenia 3T;

Table 3 Urinalysis of a 16-year-old cat with hypertension and facial nerve paralysis

	Result
Colour	Light yellow
Specific gravity	1029
pH	6
Protein	+
Glucose	–
Ketone	–
Bilirubin	–
Blood/haemoglobin	–
Sediment	Inactive

Philips) was performed under general anaesthesia and revealed a small (height 3 mm \times width 4 mm \times length 4.3 mm), ovoid, ill-defined lesion in the mesencephalon (Figure 1). Compared with grey matter, the lesion appeared hypointense in T2-weighted (T2W) and fluid-attenuated inversion recovery images, and isointense in T1-weighted (T1W) images. The lesion caused an area of signal void in T2W* (gradient echo) images and did not enhance after intravenous injection of gadolinium-based contrast medium (0.2 ml/kg [Dotarem; GE Healthcare]). The findings were consistent with an intra-axial early subacute haemorrhage in the mesencephalon. The right facial nerve was of normal thickness but enhanced more than the left facial nerve after contrast injection (Figure 2). Additional findings included a small space-occupying lesion originating from the pituitary gland, a thyroid nodule, mild fluid accumulation in the left tympanic cavity, mild mandibular and medial retropharyngeal lymphadenomegaly, mild thickening of the right nasal mucosa with mild fluid accumulation, and mild contrast enhancement of the conjunctivae of the right eye (Figure 3). Retinal abnormalities (ie, detachment) were not visible.

Based on all of the findings, the diagnoses were: right-sided facial nerve paralysis; corneal ulceration of the right eye; systemic hypertension probably associated with hyperthyroidism; well-controlled hyperthyroidism; cerebral, acute haemorrhagic infarct located in the mesencephalon; neuropathy of the facial nerve; suspicion of a pituitary adenoma; otitis media on the left side; non-destructive right-sided rhinitis; and regional lymphadenitis.

The cat received eye drops (Tobrex; tobramycin q8h for 10 days), was placed on antihypertensive medication (Amodip; amlodipine 1.25 mg PO q24h) and was maintained on transdermal thiamazole ointment (5 mg/0.1 ml at a dosage of 0.1 ml q12h applied to the ear pinnae). BP was rechecked after 5 days and was 140 mmHg.

Three weeks after initial presentation the sutures from the partial tarsorrhaphy were removed. Facial nerve function was considered normal at this time. BP

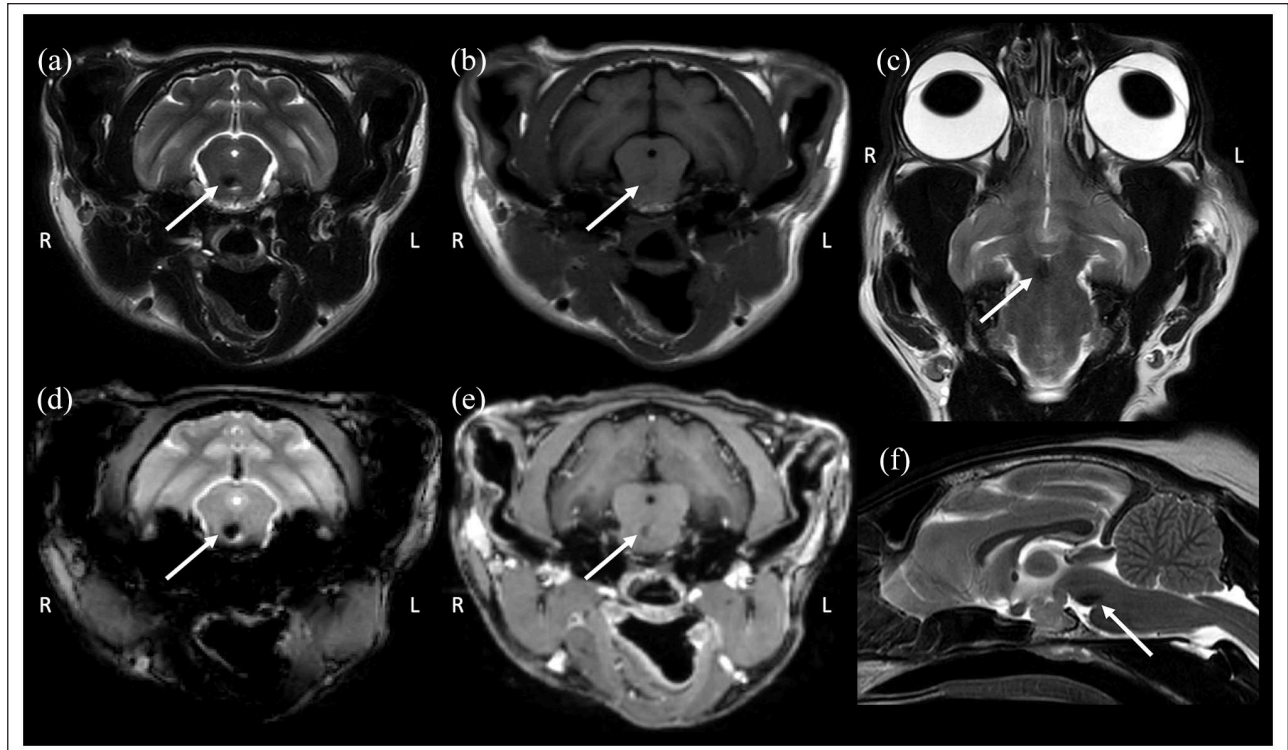


Figure 1 Cerebral MRI examination images of a 16-year-old cat with acute unilateral facial nerve paralysis. (a) Transverse T2-weighted (T2W); (b) transverse T1-weighted (T1W); (c) dorsal T2W; (d) transverse T2W* (gradient echo); (e) transverse T1W (gradient echo), after intravenous injection of contrast medium; and (f) sagittal T2 images. A small, ill-defined lesion (white arrows) was identified in the mesencephalon. Compared with grey matter, the lesion was hypointense in T2W and isointense in T1W images. It caused signal void in T2W* images and did not enhance after contrast injection. The findings were consistent with acute haemorrhage. R = right; L = left

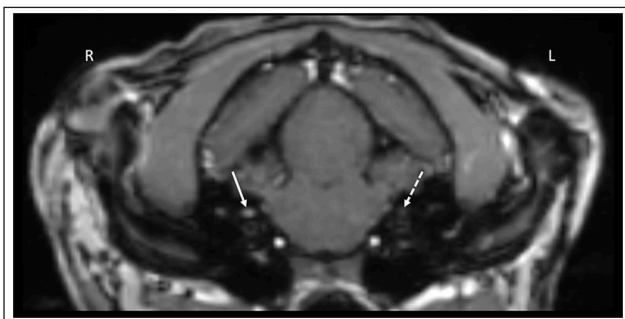


Figure 2 Transverse T1-weighted (gradient echo) image after intravenous injection of contrast medium. The right (R) facial nerve (solid arrow) had the same diameter as the left facial nerve (dashed arrow) but showed enhancement after contrast administration compared with the unenhanced left (L) side

was 170 mmHg; amlodipine was kept unchanged (1.25 mg, q24h, in the morning), but telmisartan was added (1 mg/kg, q24h, in the evening). Two months after initial presentation, the cat remained neurologically normal, serum thyroxine was normal (1.6 µg/dl) and BP was 150 mmHg. The cat was episodically rechecked. At the time of writing, 20 months after initial presentation,

based on a telephone call with the owner, the cat remained clinically neurologically normal.

Discussion

In human medicine, non-congenital facial nerve paralysis is uncommon and mostly diagnosed as idiopathic (Bell's palsy).¹⁻³ In addition, facial nerve paralysis has been reported as the initial feature of systemic hypertension, mainly in children, but also in adults.⁴⁻⁹ In children, hypertension leading to facial nerve paralysis is mostly caused by renal disease.^{4,5,7,8} The pathology behind this is believed to be haemorrhage into the facial nerve canal and/or vascular insults, which result in pressure on the facial nerve causing paralysis. The facial nerve is thought to be particularly vulnerable to haemorrhagic or oedematous injury due to the enclosed space of the fallopian canal.⁴

In cats, reports on facial nerve paralysis are scant and otitis media is considered the most common cause.¹⁰ Other identified causes of isolated facial nerve dysfunction have included surgical and non-surgical trauma and neoplasia; in 25% of the cats in one study, facial nerve paralysis was judged to be idiopathic.¹¹ Finally, facial nerve dysfunction has been described to be associated

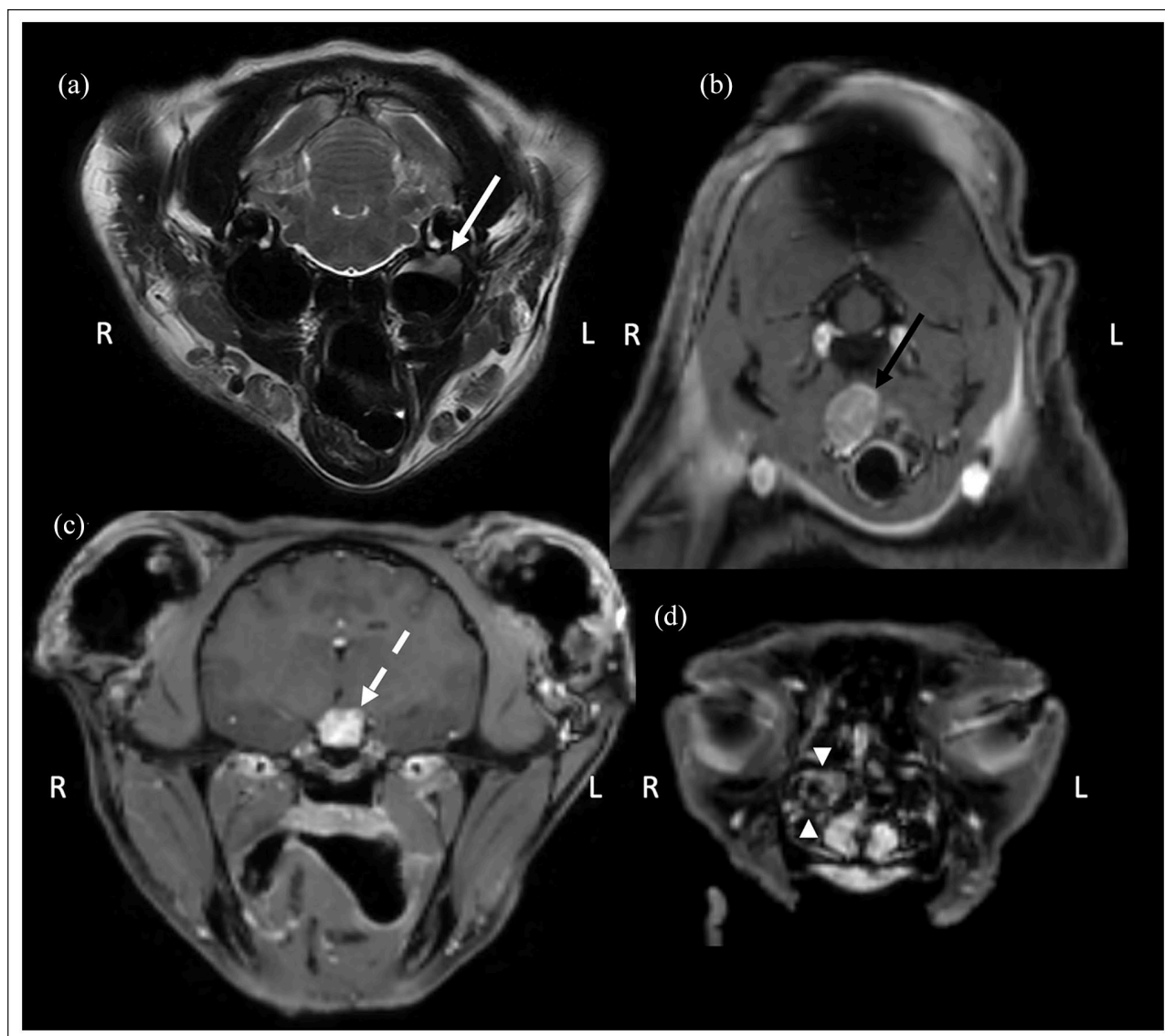


Figure 3 MRI examination images. (a) Transverse T2-weighted (T2W) image at the level of the tympanic bullae. (b–d) Transverse T1-weighted (T1W) (gradient echo) images at different levels after intravenous injection of contrast medium: (b) thyroid gland; (c) pituitary gland; and (d) nose. The left tympanic cavity was filled with a small amount of fluid (white solid arrow). Enlargement of the right thyroid gland was suspicious for an adenoma (black arrow). Additional findings included mild pituitary enlargement (white dashed arrow) and right-sided mild, non-destructive rhinitis (white arrowheads). R = right; L = left

with a generalised polyneuropathy.¹² Ultimately, the idiopathic disorder is a diagnosis of exclusion that is based on the inability to diagnose any causative abnormality using routine MRI or CT, evaluation of cerebrospinal fluid and metabolic studies; the availability of high-quality MRI is fairly recent, however.

Reported MRI changes in hypertensive facial nerve paralysis in children are limited and include pontine haemorrhage at the site of the facial nucleus, ischaemic stroke in the pons affecting the post-nuclear facial fibres, multiple ischaemic infarcts in the periventricular white matter and an oedematous nerve sheath with vessel

engorgement.⁶ In adults, diffuse enhancement of the facial nerve from the fallopian canal to the stylomastoid foramen had been considered diagnostic for Bell's palsy (ie, idiopathic facial nerve paralysis);¹³ however, Bell's palsy in humans is no longer not only considered idiopathic, but also caused by hypertension.⁹

In the reported cat, the MRI revealed several abnormalities: (1) changes in the mesencephalon, but not in the area of the facial nerve nucleus, consistent with hypertension-induced haemorrhage; (2) ipsilateral contrast enhancement of the facial nerve, indicating a disrupted blood–nerve barrier, allowing leakage and

accumulation of contrast material;¹⁴ and (3) changes in the contralateral bulla tympanica together with signs of rhinitis. The last might imply that there was also ipsilateral otitis media, it was just not visible on MRI. Thus, the unilateral facial nerve paralysis in this cat could have been a result of otitis media, hypertension or neuritis, or it could have been idiopathic. Whereas resolution of the paralysis within 3 weeks of the commencement of antihypertensive treatment may suggest that hypertension played a central pathophysiological role, spontaneous resolution of the facial nerve paralysis was also possible.

In people, various case series have reported the incidence of facial palsy to be 4–17% in children with malignant hypertension.^{5,7,8} Apparently not uncommonly, a wrong diagnosis of idiopathic facial nerve paralysis is made because malignant hypertension is overlooked. In one retrospective review, the time between first facial nerve deficits and the diagnosis of severe systemic hypertension was a median of 45 days (range 0 days to 2 years).⁶ Thus, it is recommended that BP is measured in all patients with facial nerve paralysis.^{6,15} The majority of patients with hypertension-induced facial nerve paralysis showed gradual resolution of the paralysis when BP was well controlled, and reoccurrence of signs corresponded with an exacerbation of the hypertension.⁶

Systemic hypertension was thought to be caused by hyperthyroidism in the cat of this report, as hyperthyroidism is reportedly the second most common cause of hypertension in cats.^{16,17} The most common cause of hypertension in cats is chronic kidney disease, but the urine specific gravity at admission and the repeatedly (low) normal kidney values were not suggestive of this. Other causes of hypertension in cats are rare. In the view of repeatedly normal serum potassium, no additional diagnostic tests, such as abdominal ultrasound and serum aldosterone measurements, were performed to rule in hyperaldosteronism. However, normal potassium does not rule out hyperaldosteronism (in people).¹⁸ Additional rare causes are hypersomatotropism and pheochromocytoma.^{19,20} The MRI finding of a pituitary mass could support hypersomatotropism. However, hypersomatotropism that does not eventually cause diabetes mellitus would be unusual.¹⁹ Other functional pituitary tumours, such as those leading to pituitary-dependent hyperadrenocorticism, are possible; again, chronic hyperadrenocorticism in a cat without diabetes mellitus or dermatological abnormalities would be very unusual, however.²¹ In the cat in this report, no further endocrinological tests were performed to rule in hyperaldosteronism, hypersomatotropism, hyperadrenocorticism or pheochromocytoma. Finally, the cause of hypertension might have been idiopathic; however, idiopathic hypertension is usually a diagnosis of exclusion,

and as hyperthyroidism is a recognised cause, this was the suspected cause in this cat.

Conclusions

Systemic hypertension may induce isolated facial nerve paralysis in cats. Considering similar reports in human medicine, it may be speculated that hypertension as a cause of so-called idiopathic facial nerve paralysis has also been underdiagnosed in cats because BP may not have been routinely measured in affected cases.


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Ethical approval The work described in this manuscript involved the use of non-experimental (owned or unowned) animals. Established internationally recognised high standards ('best practice') of veterinary clinical care for the individual patient were always followed. Ethical approval from a committee was therefore not specifically required for publication in *JFMS Open Reports*. Although not required, where ethical approval was still obtained it is stated in the manuscript.

Informed consent Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (experimental or non-experimental animals, including cadavers) for all procedure(s) undertaken (prospective or retrospective studies). No animals or people are identifiable within this publication, and therefore additional informed consent for publication was not required.

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