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Case Report

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Diagnosis of post-attenuation neurological signs syndrome in a cat with refractory status epilepticus and clinical response to therapeutic plasma exchange

Lisa Niemann, Katrin Beckmann, Claudia Iannucci, Adriano Wang Leandro and Alessio Vigani

Abstract

Case summary An 8-year-old female spayed British Shorthair cat that underwent surgical portosystemic shunt (PSS) attenuation developed progressive neurological signs 7 days postoperatively. Neurological signs progressed, despite medical management, and seizure activity became rapidly refractory to anticonvulsants. The diagnosis of post-attenuation neurological signs (PANS) was made based on the timing of the occurrence of clinical signs following surgery, absence of hyperammonaemia and suggestive MRI findings of the brain. The cat developed status epilepticus that required treatment with general anaesthesia and mechanical ventilation, from which the cat could not be effectively weaned without the recurrence of seizures. Therapeutic plasma exchange (TPE) was performed as a rescue therapy for PANS and associated refractory status epilepticus. A total of two plasma volumes were processed during one single TPE session. The seizure activity resolved immediately after the TPE session, the cat showed progressive improvement of neurological signs and remained stable thereafter. No significant complications associated with the TPE were observed. The cat was discharged 11 days after admission and was fully recovered.

Relevance and novel information This is an unusual report of PANS diagnosed in a cat based on clinical and MRI findings. The cat developed refractory status epilepticus and had a positive outcome following TPE as rescue therapy. The MRI findings in this report could be useful for the diagnosis of PANS in cats. We speculate that TPE could be taken into consideration as a possible therapeutic intervention in PANS syndrome.

Keywords: TPE; PANS; MRI; myelinolysis

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Introduction

The occurrence of post-attenuation neurological signs (PANS) is a known complication after portosystemic shunt (PSS) attenuation and has been reported in dogs and cats.[1] PANS is defined as any postoperative neurological signs ranging from obtundation, ataxia, behavioural changes, tremors, twitching and, in the most severe cases, seizures, occurring most commonly within 7 days post-PSS attenuation.[2] It can develop even in cases without any preoperative signs of encephalopathy and is characterised by higher refractoriness to medical management than hepatic encephalopathy (HE).[3] The aetiology of PANS is still largely speculative and, differently from HE, is typically unrelated to hyperammonaemia, hypoglycaemia or electrolyte disturbances. The characterisation of PANS in cats is still

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poorly described and data regarding the possible causes, diagnosis and therapy are scant.

Refractory PANS is commonly associated with a grave prognosis in cats.[3] It has been found that among cats that developed PANS, 56% with mild neurological signs recovered without specific treatment, whereas up to 22% of cats progress to develop refractory seizures with a high mortality rate.[4]

The use of MRI has shown promising results in better understanding the pathophysiology of this syndrome, very similar to post-liver transplant encephalopathy in humans. Unusual MRI findings in the brain have been described in a single dog affected by PANS.[5] In this dog, MRI changes 23 month after shunt ligation were compatible with extensive bilateral symmetrical cortical necrosis.[5]

Prophylactic levetiracetam administration prior to PSS attenuation is the only intervention shown to potentially decrease the incidence of postoperative seizures in dogs.[6] Controversy still exists on specific therapeutic recommendations for PANS-associated seizures. Antiepileptic drugs such as benzodiazepines, levetiracetam, barbiturates and propofol have all been described with mixed results regarding efficacy.[3,6,7]

Therapeutic plasma exchange (TPE) is described as an effective therapy for post-liver transplant encephalopathy in humans.[8] There are only sporadic reports of the use of TPE in veterinary medicine and only one report on the successful use in a dog with hyperammonaemic HE.[9] There are no descriptions of the clinical use of TPE in PANS, although the possibility of rapid removal of biochemical factors and solutes causing or triggering this syndrome may justify clinical investigation as a possible therapeutic application.

This is a report of unusual MRI findings indicative of PANS in a cat that developed refractory status epilepticus and responded effectively to therapeutic plasma exchange as rescue therapy.

Case description

An 8-year-old spayed female British Shorthair cat weighing 4.3 kg presented to the University of Zurich Small Animal Hospital with ptyalism, weakness and facial muscle twitching intermittently for less than 1 week.

On complete neurological examination, mentation and cranial nerves were normal. The main abnormalities were generalised weakness with crouched walking and rapid exhaustion, spontaneous tremors, muscle twitching, reduced proprioception and increased patellar reflex.

Serum biochemistry revealed the following abnormalities: increased ammonia (313.3 µmol/l; reference interval [RI] <70), increased postprandial bile acids 76 µmol/l (RI <25), decreased cholesterol (1.9 mmol/l; RI 2.6–6.8), decreased alanine aminotransferase (26 U/l; RI 34–98), slightly decreased urea (7.3 mmol/l; RI 7.4–12.6) and decreased creatinine (86 µmol/l; RI 98–163). Complete blood cell counts revealed leukopenia (3.900/µl; RI 4.6–12.8) with the presence of bands (1650/µl; RI 232–1010).

Based on these findings, the primary differential diagnosis was HE. The cat was hospitalised pending further diagnostics and treated with levetiracetam (20 mg/kg IV q8h [Levetiracetam mepha; Mepha Pharma]), amoxicillin–clavulanic acid (20 mg/kg IV q8h [Co-Amoxi Mepha Trockensub 550 mg; Mepha Pharma]) and lactulose (0.5 ml/kg PO q6h [Rudolac, Streuli Pharma]) and intravenous maintenance fluid therapy with isotonic crystalloids at 2 ml/kg/h (Ringer-Acetat; Fresenius Kabi).

Abdominal CT was performed and revealed an intrahepatic PSS. Medical management for HE was continued in the hospital, as indicated above.

After 24 h without any focal seizures and an improved neurological status, the cat was discharged home with medical management in preparation for the PSS attenuation with oral levetiracetam (30 mg/kg q8h [Levetiracetam mepha; Mepha Pharma]), amoxicillin–clavulanic acid (20 mg/kg q8h [Co-Amoxi Mepha Trockensub 550 mg; Mepha Pharma]) and lactulose (0.5 ml/kg q8h [Rudolac; Streuli Pharma]).

The cat remained asymptomatic following discharge until 2 weeks later when a fluoroscopy-guided coil embolisation of the intrahepatic shunt was performed under general anaesthesia without any complications.

The cat was discharged 3 days after shunt ligation with no clinical signs of HE or any evidence of portal hypertension after the embolisation. Medical therapy was continued at home with addition of the antithrombotic clopidogrel (4 mg/kg PO q24h for 8 days [Clopidogrel 75 mg; Sandoz–Novartis]).

One week after the procedure, the cat presented with recurrence of weakness, apathy and ear twitching. The following day, it developed generalised tonic–clonic seizures, which stopped with the administration of midazolam (0.5 mg/kg IV [Dormicum; Roche]). Owing to the increase in seizure activity, the cat was additionally treated with phenobarbital (3 mg/kg IV q12h [Phenobarbital; Bichsel]).

An abdominal ultrasound was performed on the same day to evaluate the correct localisation of the intrahepatic coil. The coil was detected in the shunt vessel, with no remaining blood flow and no primary or secondary signs of portal hypertension.

The biochemistry profile showed the following abnormalities: mildly increased postprandial bile acids (32 µmol/l; RI <25), increased alanine aminotransferase (137 U/l; RI 34–98) and decreased urea (4.7 mmol/l; RI 7.4–12.6). Ammonia was normal (31.7 µmol/l; RI <70 µmol/l).

The cat's mentation declined rapidly over the following night with an increased number of seizure events that were no longer controllable with antiepileptic

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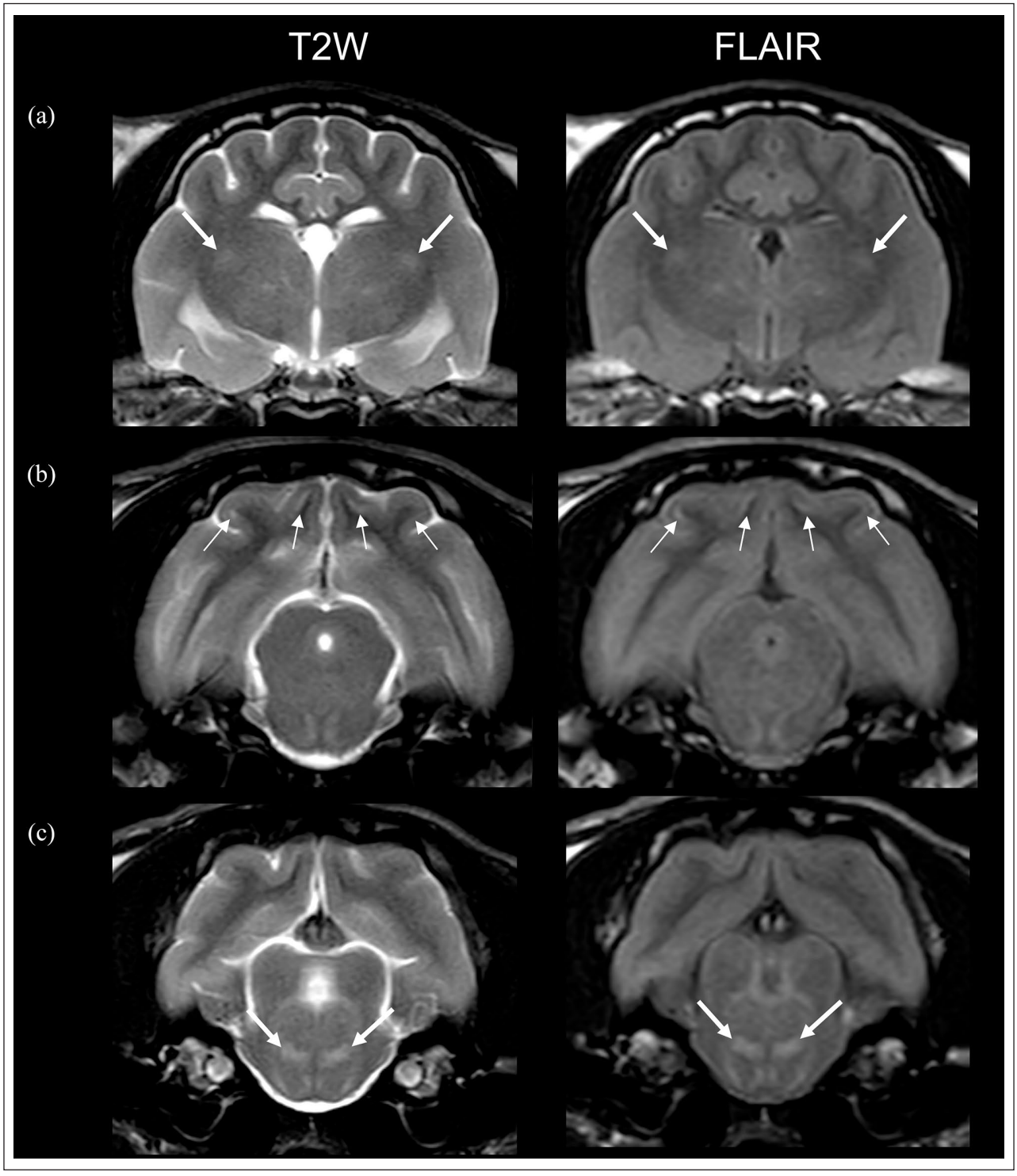


Figure 1 T2-weighted (T2W) and fluid attenuating inversion recovery (FLAIR) sequences in transverse planes at the level of (a) the interthalamic adhesion, (b) mesencephalic aqueduct and (c) cochleae. Small, ill-defined, bilateral and symmetrical intra-axial hyperintense lesions affecting (a) the lateral geniculate nuclei, (b) occipital cortical grey matter, reticular formation and (c) pons are indicated by the arrows

therapy. The cat was induced into general anaesthesia for a 3T MRI of the brain. MRI revealed bilateral symmetrical areas of increased T2 signal intensity at the lateral geniculate nuclei, tegmental reticular formation and pons. Moreover, bilateral and symmetrical, fine, laminar areas of increased T2 signal intensity were visible at the cortical grey matter, adjacent to the grey–white matter transitions of the occipital lobes (Figure 1).

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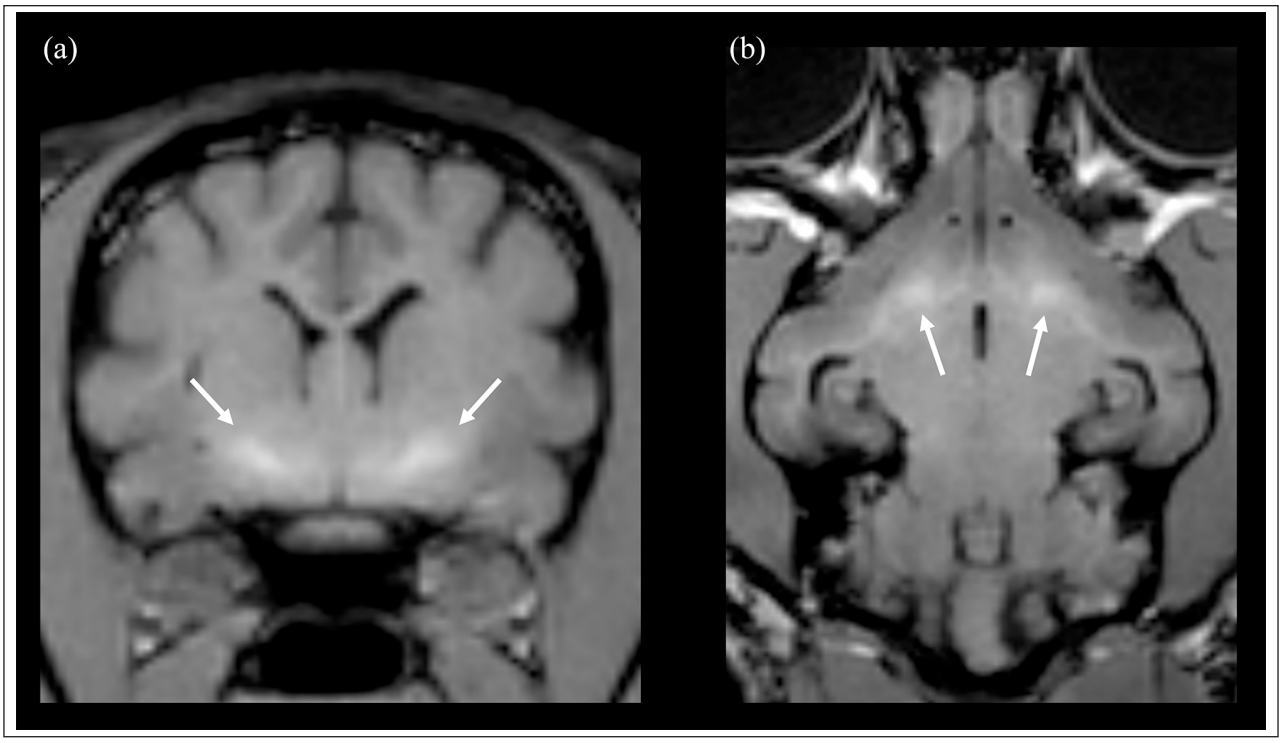


Figure 2 Precontrast T1-weighted (T1W) images in a transverse plane at the level of (a) the optic chiasma and dorsal plane at the level of the (b) rostral commissure. Bilateral, symmetrical T1W hyperintensities of the lentiform nuclei are indicated by the arrows

The lentiform nuclei showed a diffusely and symmetrically moderate increased T1 signal intensity (Figure 2).

Imaging diagnosis of a multifocal symmetrical encephalopathy was determined. The primary differential diagnosis for the changes was a metabolic toxic disease, in line with the presumptive diagnosis of PANS. The bilateral changes in the pons mirror recently reported changes in suspected osmolar myelinolysis.[10] Additionally, concomitant postictal cortical brain oedema or early-stage laminar cortical necrosis were considered the most plausible cause of the occipital lobe bilateral T2W hyperintensities. The T1 hyperintensities at the lentiform nuclei were compatible with manganese deposition and were considered to be residual from the PSS.

The cat was kept under total intravenous anaesthesia with ketamine (Ketanarkon 100; Streuli Pharma), dexmedetomidine (Dexdomitor; Zoetis) and midazolam (Dormicum; Roche) continuous rate infusion, and mechanically ventilated (Hamilton Medical) with synchronised intermittent mandatory ventilation for 24 h.

Because of the patient's rapid decline in neurological status despite the aggressive medical management, failed attempt to wean the patient without re-occurrence and following discussion on the prognosis with the owners, the decision was made to attempt rescue therapy with TPE for the PANS-associated refractory seizures.

A 6 F × 10 cm haemodialysis catheter was placed percutaneously in the right jugular vein. A single standard session of TPE was performed using the PrisMax device (Baxter Healthcare) with a paediatric Prismaflex TPE 1000 set (Baxter Healthcare) as an extracorporeal circuit. The TPE session lasted 2.5 h, exchanging a total of 300 ml plasma, representing 200% of the patient's plasma volume (154 ml) estimated using the formula 55 ml × BW (kg) × (1 – PCV), where BW denotes body weight and PCV the packed cell volume.[11] Isovolaemic fluid replacement with type-matched feline fresh frozen plasma was used during TPE. Circuit anticoagulation was maintained using ACDA 30% solution (ACD A; Bichsel) at an infusion rate automatically adjusted for by the TPE device to maintain a constant circuit citrate concentration of 3 mmol/l. Calcium substitution with calcium gluconate 2.5% solution (B Braun Medical) was administered to maintain normal serum ionised calcium concentration.

During TPE the cat was maintained under general anaesthesia and mechanically ventilated as prior to the session. Electrocardiography, capnography, pulse oximetry and non-invasive blood pressure measurements were monitored during the session. No significant adverse events were present during the TPE session, besides a self-resolving period of bradycardia. At the end of TPE, the cat was spontaneously breathing, maintaining a normal oxygen saturation on room air and was then extubated. There was no evidence of seizure activity in the recovery period or thereafter. The cat

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became conscious and responsive within 24 h. Medical management with anticonvulsants and antibiotics was continued post-TPE. Reduced neuromuscular function persisted for 48 h before the cat was able to stand. On day 5 post-TPE the cat had normal motor function, was ambulatory and eating spontaneously.

The following abnormalities were noted on serum biochemistry 24 h post-TPE: increased total bilirubin (98.3 µmol/l; RI 0.1–3.5), total proteins dropped to 45 g/l (from 70 g/l prior to TPE), low serum albumin (19 g/l: RI 32–42) and decreased phosphate (0.68 mmol/l: RI 0.9–1.8). Packed cell volume dropped to 23% from 35% prior to TPE. Serum ammonia was 55.4 µmol/l.

The cat remained hospitalised with supportive care and monitoring for almost 2 weeks before discharge. The cat was discharged with phenobarbital (2 mg/kg PO q12h [Phenobarbital; Bichsel]) and levetiracetam (15 mg/kg PO q8h [Levetiracetam Mepha; Mepha Pharma]). Neuromuscular function had improved dramatically at the 1-month recheck and no seizure activity was reported by the owner. Complete blood count and serum biochemistry were normal. The last follow-up was 2 months after discharge and the cat was in good general condition, without any seizure activity.

Discussion

The cat in our report underwent surgical attenuation of PSS. Seven days after it developed progressive neurological signs up to refractory seizures. The diagnosis of PANS was suspected based on history, absence of hyperammonaemia and the following unusual findings in the brain MRI.

The T1 hyperintensity of the lentiform nuclei was due to manganese accumulation,[12] and is considered a typical finding in HE in cats and dogs.[13] In humans and in a few canine cases,[13] this T1 hyperintensity has been demonstrated to persist months after successful HE therapy and it is therefore expected to still be present just 1 week after the PSS attenuation in a cat previously diagnosed with HE.[14] The linear T2 hyperintensity in the occipital lobe most likely resembles linear cortical necrosis. It has been reported after prolonged seizure activity[15] and was detected 2 years after shunt ligation in a dog previously suffering from generalised tonic–clonic seizures.[5] It is unclear if this finding represents a cause or a consequence of the seizures in our cat. The third MRI finding is perhaps the most relevant for the diagnosis of PANS. The bilateral symmetrical areas of increased T2 signal intensity in the lateral geniculate nuclei, tegmental reticular formation and pons appear particularly similar to those lesions previously reported in a suspect case of pontine and extrapontine myelinolysis in a cat.[10] Similar findings have also been described in a dog diagnosed with experimentally induced pontine myelinolysis 4 days after correction of the hypernatraemia and in a dog with experimentally induced hyponatraemia.[5,16]

Pontine and extrapontine myelinolysis, referred to as osmotic demyelination syndrome (ODS), is associated with severe and acute neurological signs, and has been described as a fatal complication in humans after liver transplantation.[17,18] In the present case, neurological signs associated with ODS might have been missed because of the severe seizure activity and the postictal signs in combination with sedation caused by the antiepileptic drugs.

Although the pathophysiology is not completely understood, ODS is caused by a rapid shift in plasma osmolarity. Commonly, it is due to a sudden increase in plasma sodium concentration causing osmotic injury and altering astrocyte and neuronal function. Pre-existing impaired astrocyte function has also been demonstrated in humans with HE, predisposing them to a higher risk for the occurrence of ODS, even in the absence of drastic changes in plasma osmolarity.[19] Increased plasma osmolarity measured 48 h post-liver transplantation has been detected in humans who developed clinical signs and MRI abnormalities consistent with ODS.[19] Increased plasma sodium and osmolarity have also been suspected as a possible risk factor for development of PANS in dogs.[2]

Interestingly, cases of confirmed pontine and extrapontine myelinolysis without detectable shifts in plasma sodium have been reported. In these cases, the accumulation of other osmoles is the most likely the reason behind the rise in plasma osmolarity for the development of structural brain injury and neurological dysfunction. Among the known organic substances regulating the osmotic pressure of neuroglial cells, significantly increased concentration of inositol and myoinositol in the plasma and brain tissue of patients with liver cirrhosis developing ODS have been reported.[20] Abnormalities in myoinositol concentrations have been found in dogs suffering from HE, supporting the theory of a reduced capacity to respond to osmotic changes in these dogs.[21]

In our cat, plasma sodium, as well as calculated plasma osmolarity, were unchanged before and after PSS attenuation (pre-attenuation plasma sodium 156 mmol/l [calculated osmolarity 333 mOsm/kg] and post-attenuation plasma sodium 153 mmol/l [calculated osmolarity 326 mOsm/kg]). It is thus unlikely that the change in primary plasma osmoles could be the cause of the suspected ODS lesions detected by MRI. Unfortunately, plasma osmolarity and inositol/myoinositol concentration were not directly measured.

TPE was shown to be effective in improving neurological function in post-liver transplant patients who developed ODS.[8] The removal of osmotically active solutes, such as inositol, has been indicated as a plausible explanation for the efficacy of TPE treatment in these cases. It was shown that TPE favours the outcome in ODS and leads to a marked and rapid improvement of motor and neurological function.[8]

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In veterinary medicine, there has only been one case describing the use of TPE during the course of HE in a dog.[9] In this report, hyperammonaemic HE secondary to PSS was managed with TPE treatment when it became refractory to medical management. Although it was not possible to confirm a cause-and-effect link, there was a good time correlation between completion of the TPE treatment, reduction of plasma ammonia concentration and the resolution of neurological signs.

The use of TPE in PANS is even more prone to speculation due to the current lack of a conventionally measurable solute to be removed. In our cat, there was a very rapid time sequence between TPE and the complete resolution of seizure activity, allowing us to reasonably speculate a possible direct effect of the treatment by removing osmotically active substances (ie, inositol) responsible for ODS.

There are special considerations for TPE in cats. The extracorporeal circuit volume represents a significant expansion of the circulatory volume of the patient, with consequent haemodilution proportional to the ratio circuit volume:patient blood volume. When the ratio is above 15%, common in small patients (<10 kg), it can lead to severe haemodilution and anaemia during the treatment. A critically ill patient would less likely be able to tolerate a significant drop in haematocrit, and priming of the extracorporeal circuit with type-matched blood products is recommended.

The prescription of TPE focuses on the calculation of how much plasma will be exchanged during the treatment. In this report we processed two plasma volumes (PVs) during a single TPE session. It is well recognised that the only fluid compartment accessible with TPE in terms of the removal of substances is the intravascular space. A total plasma exchange equivalent to two times the patient's PV is considered the best compromise between maximal solute removal and the required plasma replacement. The exchange of two PVs correlates with an 86% decrease in all solutes in plasma, which can be calculated using the following formula:

$$y = y_0 \cdot e^{-x}$$

Where y is the final concentration of the solute at end of treatment, y_0 is the initial concentration of the solute, e is the base of the natural log and x represents the number of times the patient's plasma volume is exchanged during TPE.[22]

Conclusions

We speculate that a combination of the timing of onset of neurological signs after PSS ligation, the absence of hyperammonaemia and the suggestive MRI findings confirm a diagnosis of PANS. In this cat, the MRI findings in the brain were similar to ones reported in dogs with PANS and also indicative of ODS.

Our patient showed a rapid response following TPE, with complete resolution of the seizure activity immediately after the session. The neurological signs also continued to improve over the course of a week, with no recurrence of any seizures. We cannot define a direct correlation between TPE and the resolution of all neurological signs with certainty. Because of the persistent refractoriness of the seizures, TPE was chosen as a last attempt to avoid euthanasia. Given the non-hyperammonaemic nature of PANS, it is impossible for us to prove which biochemical factors were possibly removed by TPE. Further investigation on the use of TPE in PANS in small animals could allow better definition of the clinical therapeutic recommendations.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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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 and/or this work involved the use of cadavers. 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). For any animals or people individually identifiable within this publication, informed consent (verbal or written) for their use in the publication was obtained from the people involved.

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