Received: 19 June 2020 | Revised: 12 August 2021 | Accepted: 26 September 2021

DOI: 10.1002/vrc2.211

Vet Record CaseReports

CASE REPORT

Companion or pet animals

Feline acquired thymoma-associated myasthenia gravis managed with surgery and therapeutic plasma exchange

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Abstract

A 3-year-old cat with thymoma-associated myasthenia gravis underwent thymectomy. Skeletal muscle weakness deteriorated after surgery, and the cat became non-ambulatory tetraparetic. A higher dose of pyridostigmine failed to improve the weakness. Therapeutic plasma exchange was performed twice, 4 and 8 weeks after surgery, with continued pyridostigmine treatment. Improvement in clinical signs was noted after each therapeutic plasma exchange. Improvement was short-lasting after the first and long-lasting after the second session. The dose of pyridostigmine was reduced 4 and 21 months after thymectomy without relapse. Thirty-two months after thymectomy, the cat remained in clinical remission with continued pyridostigmine treatment, but serum anti-acetylcholine receptor antibody titre remained elevated (2.0 nmol/L). This is the first report of therapeutic plasma exchange in a cat with acquired thymoma-associated myasthenia gravis. Therapeutic plasma exchange may be considered an additional immune-modulating option to medical treatment in cats with acquired myasthenia gravis.

KEYWORDS

anti-acetylcholine receptor antibodies, feline, lower motor neuron, plasmapheresis, thymectomy

BACKGROUND

Myasthenia gravis is a neuromuscular transmission disorder. A characteristic clinical sign is muscle weakness, which worsens with activity.[1] In acquired myasthenia gravis, autoantibodies against postsynaptic acetylcholine receptors at the motor endplate of striated muscles cause failure in neuromuscular transmission.[1–4] Autoantibody binding decreases neuromuscular transmission by blocking the receptor, reducing the number of receptors and destroying the endplate architecture via activation of the complement cascade, resulting in a widened synaptic cleft.[4]

Different classifications exist in human and veterinary medicine depending on clinical signs.[4,5] In human medicine, myasthenia gravis is clinically subclassified into pure ocular myasthenia gravis and generalised myasthenia gravis with early or late onset.[4] In canine and feline species, the classification includes focal, generalised and acute fulminating presentations.[5] Focal myasthenia gravis is defined as weakness in one or more focal skeletal muscle groups not involving appendicular skeletal muscles, and generalised myasthenia gravis as appendicular skeletal muscle weakness with clinical signs of exercise-induced weakness with or without facial, oesophageal, pharyngeal or laryngeal skeletal muscle involvement. Respiratory distress can occur in severe cases of generalised myasthenia gravis.[2,3,5] The acute fulminating form presents with acute and rapidly progressive signs of generalised weakness.[5]

Neoplasia-associated myasthenia gravis is described secondary to thymoma in humans, dogs and cats.[1,3,4,6] In cats, this form accounts for up to 52% of all reported acquired feline myasthenia gravis cases.[1,3,7] The association between the thymic disease and myasthenia gravis is complex. The thymus gland contains specific cellular elements such as antigen-presenting cells, T cells and B cells, playing a central role in the myasthenic autoimmune process.[8] A T cell-mediated activation of B lymphocytes causes the synthesis of pathogenic high-affinity autoantibodies. Due to the antigenic similarity between the neurofilaments of the thymic myoid cells and the nicotinic acetylcholine receptors, these autoantibodies bind to the α-subunits of the acetylcholine receptors.[4,6]

Standard treatment approach in acquired thymoma-associated myasthenia gravis in cats, dogs and humans is thymectomy.[4,5,7,9,10] To remove circulating autoantibodies, plasmapheresis is recommended for human patients with myasthenic crisis (severe weakness with respiratory failure and the need for intubation and mechanical ventilation), prior to surgeries (including thymectomy) or prior to high-dosage corticosteroid pulse therapy.[4,11,12] Therapeutic plasma exchange (TPE) is one treatment modality of plasmapheresis, whereby the patient's entire plasma, containing circulating, pathogenic complement factors, immune complexes, toxins,

cytokines and autoantibodies, is replaced with substitution fluid, usually donor plasma combined with colloidal or crystalloid fluids.[4,13–16] In dogs, four cases of acquired myasthenia gravis and one case with primary myasthenia gravis managed with TPE are described, with a successful outcome in three dogs.[14,17,18] To the best of authors' knowledge, TPE has not been reported yet in cats with acquired thymoma-associated myasthenia gravis.

LEARNING POINTS/TAKE-HOME MESSAGES

- Therapeutic plasma exchange was associated with a pronounced, but short-lasting, improvement of weakness in a cat with myasthenia gravis.
- Therapeutic plasma exchange may be considered as emergency treatment
- Hypotension is a potential complication of therapeutic plasma exchange, especially in small patients such as cats.

CASE PRESENTATION

A 3-year-old female neutered domestic shorthair cat with clinical signs of acquired thymoma-associated myasthenia gravis was referred for thymectomy. The cat showed progressive generalised weakness, which started with lameness in one pelvic limb 3 months ago and progressed to generalised muscle weakness. Serum anti-acetylcholine receptor antibody (anti-AChR-Ab) concentration (Comparative Neuromuscular Laboratory, University of California, San Diego, CA, USA) was increased (5.98 nmol/L; reference interval <0.3 nmol/L, Figure 1). The cat had been treated orally with pyridostigmine (Mestinon; Meda Pharma) 1.3 mg/kg TID PO and prednisolone (Prednisolon; cp-pharma) 0.5 mg/kg SID PO for the previous 2 months without any relevant change in clinical signs.

At presentation, vital signs and mental status appeared unremarkable. The cat showed severe generalised weakness and would collapse after fewer than five steps. This was graded as severe weakness. Subsequently weakness was scored based on documentation in the medical records as shown in Table 1.

Neurologic examination revealed resting limb tremor, more severe in the pelvic limbs and sometimes affecting the whole body, ventroflexion of the head and neck and severely reduced withdrawal reflexes in all four limbs. Other spinal reflexes (patellar, cranial tibial, sciatic, extensor carpi radialis reflex) appeared normal to mildly decreased. The cat showed low head and neck carriage during wheelbarrowing, and no deficits in postural reactions and cranial nerve examination were present. Neuroanatomical localisation was generalised neuromuscular system.

INVESTIGATIONS

Results of complete blood cell count, serum biochemical profile, creatine kinase activity and venous blood gas analysis were within the reference range. Presurgical thoracic radiographs and computed tomography revealed a precardiac suprasternal mass (Figures 2 and 3).

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for the precardiac, suprasternal mass included thymoma, lymphoma, ectopic thyroid tissue, parathyroid or other neoplasia.

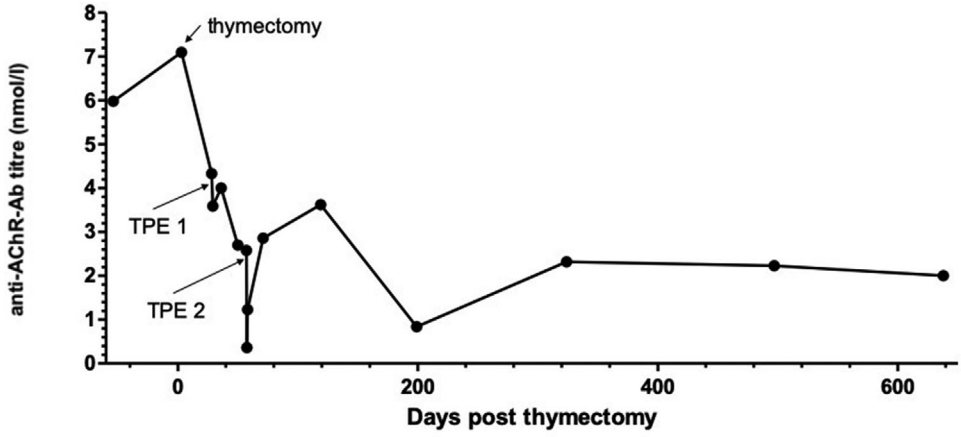

FIGURE 1 Anti-acetylcholine receptor antibody titre was monitored over a precise period of time in a cat with acquired thymoma-associated myasthenia gravis treated with thymectomy, therapeutic plasma exchange (TPE) and pyridostigmine. TPE 1: first therapeutic plasma exchange; TPE 2: second therapeutic plasma exchange

TABLE 1 Scoring sheet used for documentation of weakness in the cat with thymoma-associated myasthenia gravis

Severity	Walking	Playing	Jumping	Tremor	Cervical ventroflexion
Normal	>20 steps	>30 min/day	Yes	No	No
Mild	5–20 steps	1–10 min/day	Intermittent	Focal	Mild
Severe	<5 steps	No	No	Generalised	Profound

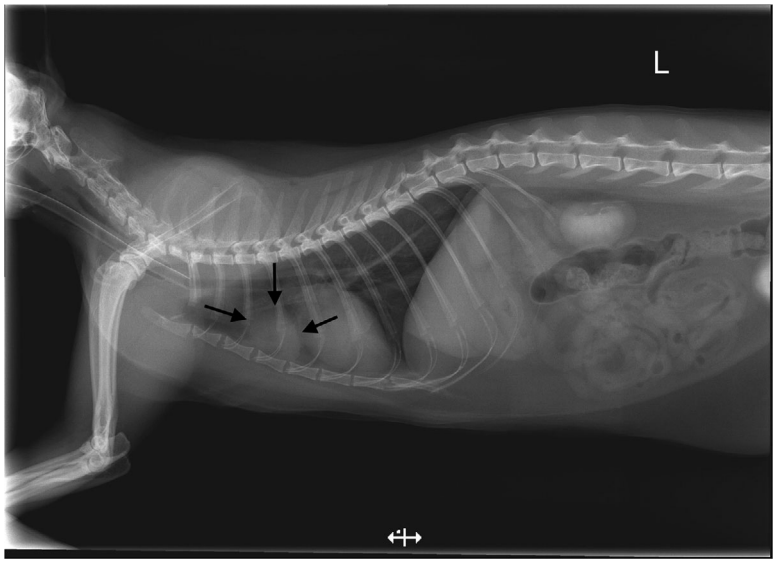

FIGURE 2 Thoracic radiographs of the cat with acquired thymoma-associated myasthenia gravis before thymectomy. The mass is indicated with arrows

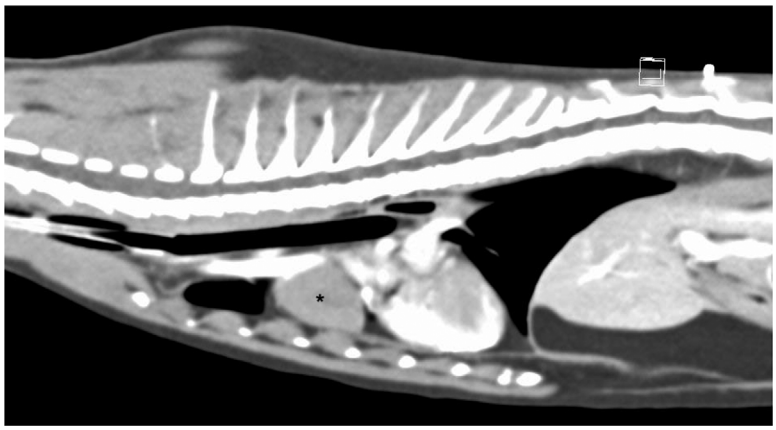

FIGURE 3 Computed tomography of the chest of the cat with acquired thymoma-associated myasthenia gravis prior to thymectomy, sagittal reconstruction. The mass is indicated with an asterisk

Differential diagnosis for generalised neuromuscular system disorders included polyneuropathy, polymyopathy and neuromuscular junction disorders. Acquired myasthenia gravis was diagnosed based on increased anti-AChR-Ab concentrations and the high specificity of this test.[3,5] There was no evidence for concurrent polymyositis, which has been described as a concomitant disorder in human and feline myasthenia gravis, since creatine kinase activity was within reference range.[2,19] However, muscle biopsies were not performed in this case; therefore, concurrent polymyositis could not be definitively excluded.

TREATMENT

The cat was anaesthetised for thoracic surgery. The mass was completely resected via a median sternotomy. As the right cranial lung lobe had a strong connection to the mass, a partial lobectomy of this lung lobe was performed. Histopathology confirmed a thymoma with T-cells being the prominent cell type, resected with clear margins. The day after surgery, the cat was alert, but weakness was worse with the cat only able to crawl but unable to stand or walk. Pyridostigmine dosage was increased to 2.0 mg/kg TID PO but no relevant improvement was observed.

At the owner's request, the cat was discharged 6 days later, despite severe persistent weakness and was tapered off prednisolone as the cat developed gastrointestinal signs thought to be related to prednisolone administration. As there was no marked improvement over the following 2 weeks, TPE was suggested as an add-on treatment and performed twice, 4 weeks and 8 weeks after thymectomy.

At the day of the first TPE, and before performing TPE, the neurological status was unchanged compared to the situation prior to surgery, and the cat collapsed after less than five steps (severe weakness, Table 1). TPE was performed as membrane plasmapheresis using a veterinary plasmapheresis machine (VetSmart; Medica). In total, two and a half times the cat´s plasma volume was replaced in the first and the second TPE over 105 minutes and 75 minutes, respectively. This replaced plasma volume was higher than usually recommended in humans (factor 1.5–1.8) as there are currently no standard recommendations in cats, and we were aiming for an effect with less sessions than usually done in humans. In both sessions, hydroxyethyl starch and feline plasma were chosen as replacement fluids (50% each). The plasma exchange required processing of 1780 and 1970 ml of the cat's blood by the machine (Table 2). Replacement volume was slightly less than removed plasma volume during the first TPE as additional fluids (sodium citrate and diluted calcium gluconate continuous rate infusion) were given for anticoagulation. During the second TPE, heparin was used as anticoagulant. Further information is listed in Table 2.

TABLE 2 Protocol of therapeutic plasma exchanges in the cat with thymoma-associated myasthenia gravis

	First therapeutic plasma exchange	Second therapeutic plasma exchange
Time point	**Four weeks after thymectomy**	**Eight weeks after thymectomy**
Sedation	Butorphanol[a] 0.2 mg/kg, IV	
Venous access	Central venous catheter[c] (7 French, 20 cm) right jugular vein	Central venous catheter[c] (7 French, 20 cm) left jugular vein
Machine and materials	VetSmart machine[b], Tubing and filter system (KIT PEX 200[b]) with extracorporeal blood volume: 53 ml	VetSmart machine[b], Tubing and filter system (KIT PEX 100[b]) with extracorporeal blood volume: 48 ml
Patient plasma volume	(0.06 × kg × [1 – haematocrit]) × 1000 = 150 ml	
Anti-coagulation	Sodium citrate 3.13%[d] - at the arterial port of the central venous catheter - ml/h = 3 × blood flow (ml/min) for 30 minutes followed by - ml/h = 1.5–2 × blood flow (ml/min) Calcium gluconate 10%[c] - at the peripheral venous catheter - diluted 1:10 in 0.9 % NaCl - ml/h = flow sodium citrate (ml/h) × 0.5	Heparin 25.000/5 ml[c] - bolus 50 IU/kg - followed by 50 IU/kg/h
Planned plasma volume to be removed	2.5-fold plasma volume (= 375 ml)	
Plasma volume removed	370 ml	400 ml
Blood volume processed	1780 ml	1970 ml
Blood flow	Start 5 ml/min, gradually increased to 30 ml/min within 15 minutes	
Duration of the TPE	105 minutes	75 minutes
Replacement fluids	166 ml hydroxyethyl starch (HAES 6%) 134 ml feline fresh frozen plasma	214 ml hydroxyethyl starch (HAES 6%) 186 ml fresh frozen plasma
Monitoring	Blood gases (ionized calcium) q30min in extracorporal (target concentration <0.4 mmol/L) and patient plasma (target concentration >0.8 mmol/L) Clinical monitoring Electrocardiography Oscillometric non-invasive blood pressure	Activated partial thromboplastin time q30min (target patient aPTT: 150%–200% increase) Clinical monitoring Electrocardiography Oscillometric non-invasive blood pressure
Side effects	Hypotension: Mean arterial pressure decreased from 140 to 56 mmHg after 30 minutes, treated with dopamine continuous rate infusion	No hypotension Mild salivation (treated with maropitant)

[a]Butorgesic; CP-Pharma, Germany.
[b]Medica S., Medolla, Italy.
[c]Certofix Duo 720, B. Braun, Germany.
[d]Eifelfango, Bad Neuenahr-Ahrweiler, Germany.

The cat experienced a severe hypotensive episode after 30 minutes, with a decrease in mean arterial blood pressure from 140 to 56 mmHg during the first TPE. Therefore, continuous rate infusion of dopamine was started (10 μg/kg/min CRI, Dopamine Fresenius 50 mg/ml; Fresenius Kabi) and mean arterial blood pressure increased to 80 mmHg. In the first hours after the first TPE, the cat appeared weaker than before TPE. However, 1 day later, the cat showed no more signs of weakness, was able to walk and play for more than 30 minutes and was able to jump on chairs (normal, Table 1). Pyridostigmine was reduced to 1.3 mg/kg BID PO, and the cat was discharged. The owner was provided with a scoring sheet to document clinical signs and severity of weakness 1 hour after and 3 hours before application of pyridostigmine (Table 1). In the evening after discharge, 30 minutes after pyridostigmine treatment, the cat showed salivation and resting tremor. Possible side effects of pyridostigmine overdosing were considered, and pyridostigmine dose was further reduced to 0.66 mg/kg BID. A second TPE after 48 hours, according to human protocols, was suggested but declined by the owner.

Over the following 3 days, the owner observed no signs of weakness, the cat could walk and play continuously and appeared as healthy as it had been prior to onset of clinical signs (normal, Table 1). On day 4, the cat was allowed outdoors for several hours and, on its return, the cat again showed mild ventroflexion and pelvic limb weakness and was able to walk only up to 10 to 20 steps (mild, Table 1). Pyridostigmine was gradually increased to 2.0 mg/kg TID PO within the following 2 weeks. No side effects appeared but the degree of weakness remained unchanged.

Due to a lack of further improvement (mild weakness, Table 1), the pet owner agreed to a second TPE 4 weeks after the first TPE (Table 2). Mild salivation occurred during this session, which resolved with maropitant treatment (1 mg/kg IV, Prevomax; Dechra). Mean arterial blood pressure remained stable (>60 mmHg). One day after the second TPE, the cat displayed no signs of weakness and was able to walk and jumped on chairs (normal, Table 1). Pyridostigmine treatment was continued at 2.0 mg/kg TID PO. No side effects associated with medication were observed.

During the next week, signs of intermittent weakness and walking for only 5–10 steps were evident 3 hours before the next medication. Pyridostigmine was increased to 2.6 mg/kg TID PO. In the following 2 weeks, two short episodes of mild pelvic limb lameness and tremor lasting less than 1 hour were reported. Thereafter, the cat was asymptomatic without any signs of weakness. Clinical remission was maintained with continued pyridostigmine treatment at follow-up after 3 years. Four months after thymectomy, pyridostigmine was reduced to 2.6 mg/kg PO BID and 21 months after thymectomy to 1.9 mg/kg PO BID without any recurrence of weakness.

Anti-AChR-Ab concentration was monitored over the entire clinical course (Figure 1). Three days after surgery, a mild increase in anti-AChR-Ab concentration from 5.98 to 7.10 nmol/L was noted. A mild decrease in anti-AChR-Ab concentration occurred after the first TPE (17%) and a marked decrease after the second TPE (86%, Figure 1), but this was followed by a rebound increase. Thereafter, anti-AChR-Ab concentration appeared to decrease continuously, but immune remission was not achieved (2.0 nmol/L, Figure 1).

OUTCOME AND FOLLOW-UP

Thirty-two months after thymectomy, the cat shows no weakness with continued pyridostigmine therapy.

DISCUSSION

This is the first report of TPE use for managing acquired thymoma-associated myasthenia gravis in a cat. TPE in combination with pyridostigmine treatment was associated with long-term clinical remission in this cat without the use of corticosteroids; however, immune remission was not achieved.

TPE is considered an effective immunomodulatory treatment option with a rapid onset of action.[20] Given the evidence to support the efficacy of TPE in human literature and previous case reports in dogs with myasthenia gravis, TPE was considered in the present case for its potential to accelerate clinical improvement.[14,15,17,18] In humans, TPE has been shown to be effective in Guillain-Barré syndrome, demyelinating polyneuropathy and acquired myasthenia gravis.[15,16] TPE is mainly performed prior to thymectomy in people and, as recently reported, in dogs with thymoma-associated myasthenia gravis to decrease the risk of postoperative complications,[4,18] while in this cat TPE was performed after thymectomy because of its potential to rapidly alleviate weakness which appeared exacerbated by thymectomy.

In the present case, TPE was performed twice within a 4-week interval. In human medicine, guidelines recommend three to six treatments of TPE over 10–14 days until clinical remission occurs combined with glucocorticoids for further antibody reduction.[4,15,16] The aim is to exchange 1–1.5 times the patient's plasma volume to remove anti-acetylcholine receptor antibodies.[4,15,16] Various protocols exist. Some units perform TPE every other day, and in other hospitals, TPE is performed daily for 5 days followed by every other day.[21] In veterinary medicine, five dogs with myasthenia gravis which were treated with TPE are published.[14,17,18] Of the five canine cases, four had acquired myasthenia gravis and one a myasthenia-like syndrome. Neoplasia was identified as the underlying cause (thymic carcinoma, thymoma and hemangiosarcoma with invasive lymphoma) in three of these dogs.[14,17,18] In each dog, TPE was performed sequentially, 24 hours, 48 hours or 72 hours apart[14,17,18] and was combined with immunosuppressive drugs, such as corticosteroids,[17,18] mycophenolate mofetil[18] or a combination of leflunomide and dexamethasone,[14] which were given prior or simultaneously to TPE.[14,17,18] Three of these dogs achieved clinical remission.[17,18] There are no reports on use of TPE in cats with myasthenia gravis in the veterinary literature.[22] In cats with acquired myasthenia gravis, the use of corticosteroids is recommended.[5,10,23] Treatment with corticosteroids or other immunosuppressants was not considered in this cat due to gastrointestinal side effects and the difficulties in administration of oral medication.

In the present case, clinical improvement occurred 12–24 hours after the first TPE, but this effect lasted only for 3 days. Thereafter, the cat worsened again despite continued treatment with pyridostigmine. In human and canine cases of myasthenia gravis which went into clinical remission, first signs of clinical improvement appeared within 24–72 hours after TPE, and improvement lasted for several weeks or months.[17,18,24] This long-lasting effect was not observed after the first TPE in the present case. Possible reasons are the lack of repeated application of TPE within a 48-hour time-interval or the discontinuation of corticosteroids, which is different from current recommendations in human medicine.[4,25] Following the second TPE, the cat improved again within 12–24 hours, and long-term clinical remission was achieved with continued pyridostigmine treatment.

TPE is an invasive procedure and transient complications may occur, most of these depend on the type of replacement fluids. In human medicine, hypotension is reported as a rare side effect of TPE besides fever, urticaria and hypocalcemia.[26] In our cat, hypotension occurred during the first TPE, while salivation, which was treated with maropitant, was the only side effect which occurred during the second TPE. The same replacement fluids were used in both TPEs, thus it is possible that hypotension was related to the exchange of large plasma volumes during the first TPE. Hypotension was successfully managed with hydroxyethyl starch bolus and dopamine continuous rate infusion. Worsening weakness for a short period after the first TPE may have been caused by hypotension. Blood pressure should therefore be monitored during TPE, and a decrease in blood pressure should be treated aggressively. Other possible causes for the transient worsening of weakness in this cat include the longer duration of the first TPE, hypovolemia, hypothermia, electrolyte imbalances and effects of sedatives. Sedatives may potentially contribute to weakness, but this cat was only sedated with a single bolus of butorphanol prior to both sessions, and no other sedatives or repeated injections were used. Therefore, it is unlikely that butorphanol caused the worsening of weakness after the first TPE. Hypothermia was not observed in the present case, and no electrolyte imbalances were evident.

In a case series of cats with hyperviscosity syndrome and congestive heart failure managed with TPE, no adverse events were mentioned,[22] but cardiac and respiratory arrest have been described in human medicine on rare occasions.[27] In 40 dogs treated with TPE for other causes than myasthenia gravis,

only two dogs with pre-existing severe dyspnea and pulmonary haemorrhage had a fatal outcome, which the authors did not attribute to the TPE itself. Other reported complications included vomiting, sneezing, urticaria, laryngeal edema, chemosis, systemic clotting and technical problems due to low blood flow rates.[28] A fatal outcome was reported in two of the four published canine cases with acquired myasthenia gravis managed with TPE.[14,18] One of these dog was euthanised after the second TPE due to oliguric acute kidney failure after cardiac arrest and resuscitation.[14] The other dog was diagnosed with hemangiosarcoma within the spleen and invasive thymoma and was euthanised due to multiorgan failure.[18]

Immune remission was not achieved in this cat, and anti-AChR-Ab concentration remained elevated. However, immune remission is generally rare in cats with thymoma-associated myasthenia gravis and even after surgery life-long-term treatment with immune-modulating drugs besides pyridostigmine is usually necessary. In a previous study, only one cat achieved immune remission after thymectomy.[7] Evidence suggests that a complete resection of the thymoma is necessary to achieve immune remission, and outcome appears less favourable with myasthenia gravis associated with thymoma.[5,10] Therefore, remaining thymomatous tissue could be another reason for the failure of immune remission.

The underlying cause for temporal clinical deterioration after thymectomy remains undefined in this cat. In dogs, a worsening of weakness has been attributed to high dosages of acetylcholinesterase inhibitors or high dosages of prednisolone, but dosages were not considered excessive in this cat.[5,11,10] General anaesthesia can also cause an exacerbation of clinical signs or myasthenic crisis.[29,30] In humans with myasthenia gravis, peripheral nerve blocks and epidural anaesthesia are the preferred anaesthetic management.[29] Neuromuscular blocking agents, which may also worsen clinical signs, were not used in this case.[29] In the present case, worsening of weakness after surgery was accompanied by an increase in anti-AChR-Ab concentration (from 5.98 prior to 7.10 nmol/L after surgery). In another cat with acquired thymoma-associated myasthenia gravis, deterioration of neurological signs after thymectomy with concomitant increase in anti-AChR-Ab concentration (from 2.75 nmol/L prior to 6.12 nmol/L 4 weeks after surgery) has also been described.[1] Anti-AChR-Ab concentration returned to pre-treatment levels after 465 days, and clinical signs resolved.[1] Other studies showed no relevant change in anti-AChR-Ab concentration in cats with acquired thymoma-associated myasthenia gravis immediately after surgery.[7] Surgical procedures, particularly thymectomies, causing surgical stress are a well-known risk factor for immune stimulation, excessive antibody production, worsening of weakness and myasthenic crisis in humans.[5,31–33] In general, in a given animal, changes in anti-AChR-Ab concentrations correlate well with the clinical course and are negatively associated with survival time, but there is poor correlation of absolute anti-AChR-Ab concentration with severity of weakness.[7,34]

The aim of the present case report was to describe the use of TPE in a cat with acquired thymoma-associated myasthenia gravis and the subsequent clinical course. Long-term clinical remission occurred after two TPEs and was maintained with continued pyridostigmine therapy without the use of corticosteroids, but anti-AChR-Ab concentration remained elevated. In conclusion, TPE was fairly well tolerated by the cat, and subsequent improvement of weakness was observed. TPE may also be considered in cats if corticosteroids are not a suitable option. General efficacy cannot be proven with a single case report, and it is possible that the cat's clinical signs waxed and waned independently of the TPE. Further studies are needed to demonstrate the efficacy of TPE in the treatment of acquired myasthenia gravis. Furthermore, the high costs and possible complications of TPE need to be considered when suggesting this treatment modality.

ACKNOWLEDGEMENTS

Open access funding enabled and organized by Projekt DEAL.

FUNDING

The authors received no specific funding for this work.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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How to cite this article: Dörfelt S, Fischer A, Meyer-Lindenberg A, Dörfelt R. Feline acquired thymoma-associated myasthenia gravis managed with surgery and therapeutic plasma exchange. Vet Rec Case Rep. 2021;9:e211. https://doi.org/10.1002/vrc2.211