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Case Report

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Acute respiratory distress syndrome and septic shock in a cat with disseminated toxoplasmosis

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Abstract

Objective – To describe acute respiratory distress syndrome (ARDS) and septic shock in a cat with disseminated toxoplasmosis.

Case Summary – A 2-year-old neutered male domestic shorthair cat was presented for acute respiratory distress. At the time of presentation it had been receiving cyclosporine for treatment of eosinophilic dermatitis. Thoracic radiographs revealed severe mixed nodular interstitial and alveolar patterns. An endotracheal wash was performed, which confirmed a diagnosis of pulmonary toxoplasmosis. Despite initial treatment with oxygen supplementation and intravenous clindamycin, the cat developed refractory hypoxemia and hypotension requiring mechanical ventilation and vasopressor support within 24 hours of hospital admission. Cardiac arrest occurred 56 hours after admission. Necropsy was performed and histopathology revealed protozoal organisms disseminated throughout the heart, lungs, liver, and brain.

New or Unique Information Provided – The clinical and necropsy findings presented here are consistent with ARDS secondary to disseminated toxoplasmosis in a cat. This is the first detailed report of ARDS in a cat. Toxoplasma titer testing and antimicrobial prophylaxis should be considered in cats prior to immunosuppressive treatment with cyclosporine.

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Keywords: acute lung injury, ARDS, feline, mechanical ventilation, Toxoplasma

Abbreviations

ABG	arterial blood gas
ALI	acute lung injury
ARDS	acute respiratory distress syndrome
ETW	endotracheal wash
P:F	PaO_2/FiO_2
PEEP	positive end-expiratory pressure
S:F	SpO2/FiO2
ScvO2	central venous oxygen saturation
VetALI	veterinary acute lung injury
VetARDS	veterinary acute respiratory distress syndrome

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Offprints will not be available from the authors.

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Introduction

Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are uncommonly reported in the veterinary literature. In 2007, the Dorothy Russell Havemeyer working group defined veterinary ALI and ARDS.[1] Criteria for the diagnosis of VetALI/VetARDS include: acute onset of respiratory signs, known risk factors (such as inflammation or infection), evidence of pulmonary capillary leak, and evidence of inefficient gas exchange (such as decreased PaO_2:FiO_2 [P:F] ratio). Optimally, evidence of diffuse inflammation is also demonstrated, but fulfillment of this criterion is not required for clinical diagnosis.[1] VetALI and VetARDS are distinguished by a difference in severity of hypoxemia as described by the P:F ratio (< 300 and < 200, respectively), which has been adopted from the human literature. While there have been sporadic reports of

ARDS in dogs,[2] ARDS has not been described in cats to date.

In critically ill people, sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Septic shock occurs in patients with sepsis who exhibit persistent hypotension and increased plasma lactate despite adequate volume resuscitation, requiring the use of vasopressors.[3] Although there is no written consensus defining criteria for septic shock in dogs or cats, veterinary studies have adopted the definition used to describe human patients.[4]

Bacterial infections are the most common cause of sepsis in veterinary patients, though viral, fungal, and protozoal sepsis may also occur.[5] The most common cause of feline systemic protozoal infection is the organism *Toxoplasma gondii*, with an overall seroprevalence of 31.6–34% in domestic cats in the United States.[6,7] These cats may never develop overt signs of disease related to *Toxoplasma gondii*, as the organism remains enclosed within cysts in the central nervous system, muscles, and visceral organs.[8] Reactivation of *Toxoplasma gondii* has been documented in cats treated with immunosuppressive therapy. In particular, the use of cyclosporine has been associated with recrudescent toxoplasmosis in both dogs and cats.[9–11] In this manuscript, the authors report VetARDS and septic shock secondary to disseminated toxoplasmosis that occurred in a cat receiving cyclosporine.

Case Description

A 2-year-old neutered male domestic shorthair cat weighing 5.1 kilograms was presented for urgent evaluation of respiratory distress, lethargy, and vomiting. The cat had been obtained from a shelter 7 months prior. While at the shelter the cat had developed dermatitis that was unresponsive to treatment with antimicrobials or prednisolone. Following adoption, the cat was referred to see a dermatologist and was diagnosed with presumptive eosinophilic dermatitis. Initial therapy consisted of an antifungal agent[a] and a hypoallergenic diet trial. When there was no improvement with these therapies, the cat was started on cyclosporine (6.3 mg/kg PO q 12 h)[b] and the owner was instructed to continue feeding the hypoallergenic diet. The dermatitis improved; however, the cat developed inappetence and intermittent vomiting. At a recheck dermatology appointment 5 days prior to emergency presentation, the total dose of cyclosporine was reduced. Because the clinician did not acknowledge a recent weight loss of 1.7 kg (24% of body weight), the mg/kg dose of cyclosporine was unchanged from the dose originally prescribed. The owners reported that the cat had neither improved nor deteriorated in the first 4 days after the dermatology recheck; however, the cat experienced a sudden increase in respiratory rate and effort beginning 20 hours prior to presentation.

Upon presentation to the emergency service, the cat was obtunded, tachypneic (70 breaths per minute), and dyspneic. Thoracic auscultation revealed bilateral pulmonary crackles and did not identify a cardiac murmur or gallop sound. Temperature and pulse rate were 100.9°F (39.3°C) and 180/min, respectively. The cat was placed in an oxygen cage with FiO_2 set to 0.6 and 0.2 mg/kg of butorphanol[c] was administered intramuscularly to relieve distress and facilitate diagnostic testing. A blood pressure measurement was not obtained due to the cat's fragile condition. Thoracic and abdominal point of care ultrasound did not reveal pleural, pericardial, or peritoneal effusion. Dorsoventral and right lateral radiographs of the thorax were performed, and revealed a severe, diffuse, nodular interstitial pattern with areas of patchy alveolar infiltrate (Figure 1).

Blood was collected for biochemical and hematologic analysis. Major abnormalities on the serum biochemistry

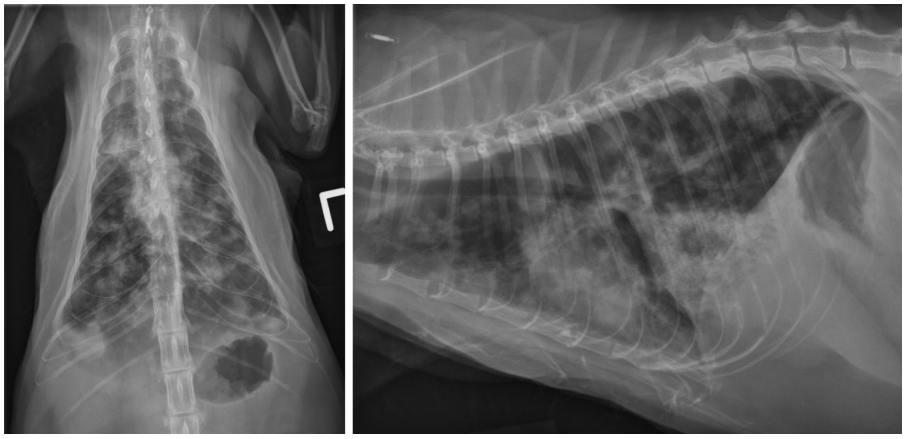

Figure 1: Dorsoventral and right lateral radiographs of the thorax taken upon presentation.

Table 1: Clinicopathologic data for the duration of hospitalization in a cat with ARDS secondary to toxoplasmosis

	Arrival	24 hours	48 hours	Reference interval
Glucose	253	N/A	N/A	75–134 mg/dL
	14.05			4.2–7.4 mmol/L
BUN	37	N/A	N/A	15–35 mg/dL
	13.2			5.4–12.5 mmol/L
Creatinine	1.7	1.6	1.8	0.9–2.3 mg/dL
	150.2	141.4	159.1	79.5–203.3 μmol/L
Total protein	5.4	4.1	4	5.5–7.7 g/dL
	54	41	40	55–77 g/L
Albumin	2.3	1.6	1.6	2.7–3.9 g/dL
	23	16	16	27–39 g/L
AST	114	144	105	6–44 U/L
ALT	75	N/A	N/A	20–108 U/L
ALP	< 20	N/A	N/A	23–107 U/L
GGT	< 10	N/A	N/A	0–10 U/L
Total bilirubin	1.0	1.2	1.4	0.1–0.4 mg/dL
	17.1	20.5	23.9	1.7–6.8 μmol/L
Lactate	N/A	2.8	3.0	0.5–2.0 mmol/L
$ScvO_2$	N/A	76.7%	75.5%	> 65%*
PCV	51	42	N/A	31–51%
	0.51	0.42		0.31–0.51 L/L
PLT	255	209	N/A	195–624 × 10^3/μL
	255	209		195–624 × 10^9/L
WBC	4.9	13.9	N/A	3.4–13.5 × 10^3/μL
	4.9	13.9		3.4–13.5 × 10^9/L
Segmented neutrophils	3.6	10.7	N/A	1.5–9.6 × 10^3/μL
	3.6	10.7		1.5–9.6 × 10^9/L
Band neutrophils	0.6	2.4	N/A	0.0–0.1 × 10^3/μL
	0.6	2.4		0.0–0.1 × 10^9/L

ALP, alkaline phosphatase activity; ALT, alanine aminotransferase activity; AST, aspartate aminotransferase activity; ARDS, acute respiratory distress syndrome; GGT, gamma-glutamyl transferase activity; PLT, platelet count; $ScvO_2$, central venous oxygen saturation; N/A, data point not available.
*, reference value from Prittie J. Optimal endpoints of resuscitation and early goal-directed therapy. J Vet Emerg Crit Care 2006; 16(4):329–339.

panel included hypoalbuminemia, azotemia, and hyperbilirubinemia, and the CBC revealed hemoconcentration and a left shift despite a normal leukocyte count (Table 1). Based on the history and initial diagnostic test results the attending clinician considered toxoplasmosis to be the most likely differential diagnosis. Other possibilities considered were fungal pneumonia and severe diffuse bacterial pneumonia. The decision was made to perform an endotracheal wash (ETW). Following intramuscular premedication with 0.02 mg/kg terbutaline,[d] anesthesia was induced with propofol[e] and 5 mL of sterile 0.9% saline was administered via a 3.5-Fr red rubber catheter through a sterile endotracheal tube. Following this procedure approximately 15 mL of serosanguinous fluid was recovered from the endotracheal tube. Total protein of this fluid measured via refractometry was 7.0 g/dL (70 g/L). The fluid recovered during the ETW was submitted for cytologic analysis and aerobic bacterial culture and susceptibility testing. Cytology revealed large numbers of non-degenerate neutrophils with fewer enlarged foamy macrophages. Moderate numbers of intracellular and extracellular *Toxoplasma* tachyzoites (crescent-shaped organisms with central nucleus) were observed (Figure 2). Heavy growth of *Pasturella multocida* was documented on final culture results.

The cat recovered well from anesthesia and was replaced in an oxygen cage with FiO_2 of 0.6. Due to absent dorsal pedal pulses, poor femoral pulses, and cool extremities noted on physical examination, a 10 mL/kg bolus of crystalloid fluid[f] was given IV over 30 minutes. Physical examination parameters improved with this therapy and IV fluids were continued at 33 mL/h for 12 hours to correct dehydration, then reduced to 50 mL/kg/day. Clindamycin[g] (15 mg/kg IV q 12 h) was started in addition to enrofloxacin[h] (5 mg/kg IV q 24 h). Tachypnea (80–100/min) persisted despite supplemental oxygen therapy with pulse oximetry readings (SpO_2) ranging between 96–98%. Approximately 12 hours following recovery from anesthesia, the patient developed a marked increase in respiratory effort with paradoxical abdominal movement and SpO_2 dropped to 88%. Repeat thoracic point of care ultrasound revealed no evidence of pleural effusion or pneumothorax; however, hepatized lung with intermittent areas of gas echogenicity were observed. Given the patient's hypoxemia and respiratory fatigue, mechanical ventilation was instituted with client consent. Anesthesia was

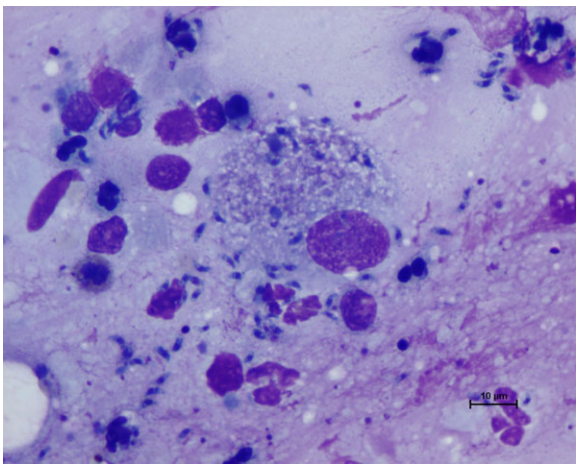

Figure 2: Endotracheal wash cytology demonstrating presence of *Toxoplasma* tachyzoites. 100× magnification.

Table 2: Trends in parameters throughout ventilation in a cat with ARDS secondary to toxoplasmosis

Time (hours)	0	6	12	18	24	30
Ventilator mode	SIMV-PC	SIMV-PC	SIMV-PC	SIMV-PC	SIMV-PC	SIMV-PC
SpO_2 (%)	86	92	89	97	93	97
FiO_2 (%)	100	85	90	70	80	90
SpO_2:FiO_2 ratio	86	108.3	98.9	138.6	116.25	107.8
$PvCO_2$ (mmHg)	N/A	44.5	N/A	N/A	44.3	54.9
$ETCO_2$ (mmHg)	30	45	38	35	44	51
RR (breaths/min)	12	18	20	20	20	25
PEEP (cmH_2O)	10	8	11	11	11	11
I:E ratio	1:2.1	1:1.2	1:1.3	1:1.3	1:1.3	1:1.5
Peak inspiratory pressure (cmH_2O)	21	20	24	23	25	27
MV (mL/kg/min)	126.6	114.6	123.6	145.5	141.8	218.2

$ETCO_2$, end tidal carbon dioxide; FiO_2, fraction of inspired oxygen; I:E ratio, ratio of inspiratory time to expiratory time; MV, minute ventilation; N/A, data point not available; PC, pressure control; PEEP, positive end expiratory pressure; RR, respiratory rate; SIMV, synchronized intermittent mandatory ventilation; SpO_2, oxygen saturation as measured by pulse oximetry; SpO_2:FiO_2 ratio, ratio of percentage of oxygen saturation to fraction of inspired oxygen.

induced with 2 mg/kg propofol[e] IV and maintained with total intravenous anesthesia using midazolam[i] (0.1–0.5 mg/kg/h), ketamine[j] (10–40 μg/kg/min), and fentanyl[k] (2–10 μg/kg/h). A synchronized intermittent mandatory ventilation with pressure control protocol was chosen with initial settings for FiO_2 of 1.0, inspiratory pressure of 10 cm H_2O, and positive end-expiratory pressure (PEEP) of 3 cm H_2O.[l] PEEP was increased to 10 cm H_2O within 15 minutes of initiating mechanical ventilation in order to achieve SpO_2 > 85%. With this approach to ventilation, tidal volumes of 6–9 mL/kg were achieved.[l] Collection of an arterial blood sample for arterial blood gas (ABG) analysis was attempted but was unsuccessful. Despite these ventilator settings, SpO_2 measurements remained between 86–89%, and worsened rapidly (SpO_2 < 82%) if FiO_2 or PEEP were reduced. Central venous and urethral catheters were placed to facilitate blood sampling and urine output monitoring. Upon placement of the urinary catheter 30 mL of urine was obtained with a urine specific gravity of 1.028. Additional therapies with trimethoprim-sulfonamide[m] (15 mg/kg IV q 12 h) for additional activity against *Toxoplasma* spp., pantoprazole[n] (1 mg/kg IV q 24 h) for stress ulcer prophylaxis, and parenteral nutrition[o] (1/3 resting energy requirement) were commenced. Persistent progressive hypotension (Doppler blood pressure[p] < 100 mm Hg) was documented despite presumably adequate fluid resuscitation. With concern that *Toxoplasma*-associated myocarditis could be contributing to hypotension, and without immediate access to formal echocardiography, dobutamine[q] was started at 3 μg/kg/min and increased to 8 μg/kg/min. Norepinephrine[r] (0.05 μg/kg/min, gradually increased to 0.5 μg/kg/min) was added when there was failure to resolve hypotension (Doppler BP < 90 mm Hg). When these measures failed to resolve hypotension, hydrocortisone[s] (2 mg/kg IV q 12 h) was added to the therapeutic regimen for possible critical illness-related corticosteroid insufficiency. The cat was ventilated for a total of 30 hours. Ventilator settings were selected with goals of maintaining SpO_2 90–92% and end-tidal PCO_2 35–45 mm Hg (Table2). Despite vasopressor support and presumably adequate IV fluid resuscitation (100 mL/kg of IV fluid therapy given over 24 hours, 0.6 kg increase in body weight), the patient remained markedly hypotensive and was found to be oliguric (urine output 0.7 mL/kg/h) then anuric. Doppler blood pressure readings were 40–65 mm Hg during the last 10 hours of hospitalization. During the last 4 hours of hospitalization there was no blood pressure reading obtainable, which was consistent with hypotension given the patient's weak femoral pulse quality and absent dorsal pedal pulses. Cardiac arrest occurred 30 hours after mechanical ventilation was started. Cardiopulmonary resuscitation was not attempted.

Necropsy revealed a small volume of pleural effusion (59 mL) with no evidence of pneumothorax. There was a large volume of fluid present in the trachea and mainstem bronchi. There were multiple nodules present throughout the lung lobes measuring from 2 × 2 × 2 mm to 20 × 10 × 20 mm. The heart weighed 23.2 g, which was considered normal for a cat of this size.[12] There was a 5 × 7 × 5 mm white nodule located 4 mm to the left of the coronary groove and 9 mm proximal to the apex of the heart. Two more white nodules were noted in the left ventricle on a papillary muscle 7 mm proximal to the apex of the ventricle, and extending into the lumen on the right auricle from the interatrial septal wall. Histopathology revealed the presence of *Toxoplasma* bradyzoites in the lungs, liver, brain, and myocardium. Pulmonary changes included severe locally extensive

necrosis extending from areas of hemorrhage characterized by wide regions of alveolar eosinophilic globular material and karyorrhectic and pyknotic debris, accompanied by scattered lymphocytes, plasma cells, foamy macrophages, and fewer neutrophils. The bronchiolar epithelium was diffusely and subtotally sloughed. Final diagnoses included severe multifocal granulomatous bronchopneumonia, local granulomatous myocarditis, marked multifocal cerebral necrosis with encephalitis, and hepatic lipidosis and necrosis.

Discussion

This report describes the first case of feline VetARDS, according to diagnostic criteria established in a veterinary consensus statement.[1] Although the cat demonstrated a month history of weight loss, physical examination 5 days prior to emergency presentation did not reveal any respiratory abnormalities. Also, the owner did not report any respiratory signs until 20 hours prior to the final hospital visit. Thoracic radiographs demonstrated bilateral pulmonary infiltrates, but did not suggest congestive heart failure since neither cardiomegaly nor pulmonary venous distention was present. Although necropsy demonstrated the presence of myocarditis, there was no structural evidence of diffuse cardiomyopathy or left atrial dilation to support a diagnosis of left-sided congestive heart failure as an explanation for poor lung function. Echocardiography or NTproBNP concentrations may have also assisted in ruling out the presence of cardiac disease; however, neither of these tests were immediately available at the time. While it is a useful clinical tool to direct treatment, echocardiography is not required as a diagnostic test in the diagnosis of VetARDS. There was evidence of airway inflammation and capillary leak based on documentation of proteinaceous material within the conducting airways found ante mortem via ETW. Due to technical difficulties in arterial sampling in a hypotensive cat, an ABG could not be performed in this case and SpO_2 was used to estimate the patient's oxygenation. In dogs, SpO_2/FiO_2 (S:F) ratios have been shown to correlate with P:F ratios.[13] Two studies in people have investigated the usefulness of the S:F ratio as a surrogate for the P:F ratio in the diagnosis of ALI and ARDS.[14,15] The first was a study in adults that found the S:F ratio threshold values of 235 (85% sensitivity, 85% specificity) and 315 (91% sensitivity, 56% specificity) resulted in P:F ratios of 200 and 300, respectively.[14] A second study of 383 pediatric human patients where > 2 ABG readings were obtained and compared to SpO_2, the S:F ratio performed well as a surrogate marker for P:F ratio in infants and children. S:F values of 263 and 201 corresponded with P:F ratio criteria for ALI and ARDS, respectively.[15] While the use of this technique has yet to be fully evaluated in cats, this patient's S:F ratio ranged 86–156, which is consistent with a diagnosis of ARDS using either the pediatric or adult human reference interval. Pulse oximetry may be unreliable in patients with poor perfusion, thus ABG analysis should ideally be performed.[16]

Histopathology of lung tissue in this case revealed severe, locally extensive necrosis extending from areas of hemorrhage, sloughing of the bronchiolar epithelium, and inflammation. Histologic changes in ARDS vary depending on the stage of ARDS and the etiology of the inciting cause. Acute respiratory distress syndrome is divided into 3 clinical stages: an acute or exudative phase lasting up to 6 days, followed by a proliferative phase, and a final fibrotic phase.[17] Although ARDS is a clinical and not a histopathologic diagnosis, the most common histologic changes observed in people with acute phase ALI/ARDS is diffuse alveolar damage.[18] This damage is characterized by interstitial and alveolar edema with mild to moderate accumulation of inflammatory cells. There is also evidence of both endothelial and epithelial injury, often with denuding of the alveolar epithelium and intra-alveolar hemorrhage.[17,18] The histopathologic changes observed in this case are consistent with a diagnosis of ARDS.

Acute respiratory distress syndrome has been described in people with toxoplasmosis secondary to immunosuppression associated with bone marrow transplantation.[19] All 3 patients reported in this series died. All patients were seropositive for *Toxoplasma gondii* prior to immunosuppression. Prophylaxis for seropositive patients with trimethoprim-sulfa antimicrobials is now the standard of care in human medicine.[20] Toxoplasmosis secondary to immunosuppression with cyclosporine in cats has been described on multiple occasions.[9,10] In veterinary medicine there is currently no consensus supporting routine measurement of *Toxoplasma* titers or routine prophylactic treatment for cats with immune-mediated disease prior to initiating therapy with cyclosporine. A recent study of cats with experimental *Toxoplasma* infection found that clindamycin prophylaxis successfully prevented recrudescence of oocyte shedding in cats immunosuppressed with dexamethasone, while the group not given prophylaxis had recurrence.[21] A serological survey of > 12,000 clinically ill cats in the United States found a seroprevalence of 31.6% in this population of cats that had clinical signs referable to organ systems affected by toxoplasmosis.[6] Given the high seroprevalence, serological evaluation and prophylaxis for seropositive cats undergoing immunosuppression should be considered. Cats receiving immunosuppressive treatment with cyclosporine following kidney transplantation are routinely screened for *Toxoplasma*, and depending upon

clinician preference those seropositive are provided antimicrobial prophylaxis.[22] When cats treated with cyclosporine demonstrate new signs of illness, clinicians should have a high degree of suspicion for infectious disease secondary to immunosuppression.

Little is reported in the literature about cats and outcomes of mechanical ventilation. The largest study to date examined outcomes in 53 cats, and found an overall survival rate of 15%.[23] Hopper and others also reported on outcomes of cats and dogs, with only 20% of cats surviving to discharge in comparison to 29% of dogs.[24] A common finding between these studies was that those ventilated for hypoxemia tended to have a worse outcome than those ventilated for primary hypoventilation due to neurologic disease. There have been sporadic reports of successful outcomes in dogs mechanically ventilated for the treatment of ARDS.[25]

The cat in this report demonstrated sepsis-induced hypotension despite adequate volume resuscitation and required vasopressor support, thus meeting criteria for septic shock. Mortality rates in people with septic shock are in excess of 40%.[3] While the reported overall mortality rate in cats with septic peritonitis is high (54%),[26] there are no studies that report survival data specifically for cats with septic shock. Given the poor survival associated with both ARDS and septic shock in people, a fatal outcome was not surprising in this cat. In hindsight, there were several aspects of this case that could have been managed differently. A level of suspicion for reactivated toxoplasmosis at the recheck appointment may have resulted in earlier identification and treatment of illness, and possibly a different outcome. The use of higher doses of norepinephrine and the addition of vasopressin at the point of pressor-refractory septic shock could have been considered. Dobutamine was used in this case due to concern for reduced systolic function secondary to possible *Toxoplasma*-associated myocarditis and sepsis. Dobutamine may have contributed to systemic hypotension due to its vasodilatory properties related to agonism of the peripheral arteriolar β_2 adrenergic receptor. Given that the patient's blood pressure improved with dobutamine administration, the authors do not suspect that the drug contributed to the hypotension documented here. Higher doses of dobutamine were not used due to concern for possible adverse effects such as seizure activity.

One of the challenges in this case was assessing the intravascular volume status of the patient. Incomplete intravenous volume resuscitation may have contributed to the hypotension observed. Positive pressure ventilation and PEEP may complicate the interpretation of central venous pressures, and thus that monitoring technique was not used to estimate intravascular volume status. The cat's plasma lactate concentration was increased, consistent with inadequate cellular energy production. Hyperlactatemia, in light of the cat's normal central venous oxygen saturation, suggests that its cells may not have been able to utilize delivered oxygen adequately.

An essential technique to maintain adequate oxygenation while using a lung-protective ventilatory strategy is the use of PEEP to maintain recruitment of lung units. Positive end-expiratory pressure increases intrathoracic pressure, leading to a reduction in cardiac output by reducing venous return to the heart.[27] The use of high levels of PEEP was unavoidable in this case to maintain conservative oxygenation goals of 88–95% SpO_2 while limiting FiO_2.[28] The authors recognize that these settings may have contributed to the severe hypotension observed in this case.

As highlighted in the case presented here, reactivated toxoplasmosis is a risk associated with immunosuppression in cats. Clinicians should be aware of potential complications including ARDS, hepatitis, myocarditis, encephalitis, and death. Prior to treatment with cyclosporine, *Toxoplasma* IgM and IgG titer testing and prophylaxis should be considered in cats.

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Footnotes

[a] Ketoconazole, Mylan, Rockford, IL.
[b] Atopica, Novartis, Basel, Switzerland.
[c] Butorphanol tatrate, Hospira, Lake Forest, IL.
[d] Terbutaline sulfate, WestWard Pharmaceuticals, Eatontown, NJ.
[e] Propofol, Hospira.
[f] Plasmalyte A, Baxter, Deerfield, IL.
[g] Clindamycin injection, Hospira.
[h] Baytril, Bayer HealthCare LLC, Shawnee, KS.
[i] Midazolam injection, WestWard Pharmaceuticals.
[j] Ketamine injection, Phoenix Pharmaceuticals, Mannheim, Germany.
[k] Fentanyl injection, Hospira.
[l] Engström Carestation, GE, Milwaukee, WI.
[m] Sulfamethoxazole and trimethoprim injection, Teva Parenteral Medicines, Inc, North Wales, PA.
[n] Protonix, Pfizer, NY.
[o] Intralipid 20%, Baxter; Travasol 10%, Baxter; 50% dextrose, Hospira.
[p] Model 811-B Doppler unit, Parks Medical Electronics Inc., Aloha, OR.
[q] Dobutamine injection, Hospira.
[r] Norepinephrine injection, Hospira.
[s] Solu-cortef, Pfizer.

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