Neurology
Case Log number: 3
Date of first treatment by applicant: 10/12/22
Patient ID number: 326364

Signalment

Canine, 326364, 1.58-year-old, male neutered, English Springer Spaniel

History

The patient was presented for evaluation of a 4wk progressive, generalized weakness despite ongoing therapy for previously presumptive diagnosis of myasthenia gravis (MG) and secondary megaesophagus (ME). The patient had a prior presumptive diagnosis of MG after a positive edrophonium chloride challenge test at 8mo old and was managed by the rDVM. He was placed on pyridostigmine – an acetylcholinesterase inhibitor at 30 mg (1.73 mg/kg) PO q12hr and within 1mo, his clinical signs resolved. Approximately 1mo prior to presentation, the patient began regurgitating and the pyridostigmine dose was increased to 45 mg (2.6 mg/kg) PO q12hr. Following this increase, the patient began to regurgitate more and developed a cough. The pyridostigmine dose was then decreased to 30 mg (1.73 mg/kg) PO q12hr. On 9/28/22 the patient was taken to the rDVM for evaluation of dyspnea and thoracic radiographs (CXR) were performed revealing signs of aspiration pneumonia (AsP) and ME. The rDVM prescribed amoxicillin and clavulanate potassium - a beta lactam antibiotic being used to treat the AsP at 250 mg (14.45 mg/kg) PO q12h. The patient was reevaluated by the rDVM on 10/12/22 where it was determined that the AsP had improved but the ME and generalized muscle weakness had worsened. The rDVM recommended immediate referral for additional diagnostics and supportive care. Since the initial presumptive diagnosis with MG, the patient had been fed in a Bailey chair, was up to date on vaccines, and received monthly flea, tick, and heartworm preventatives.

Patient status on presentation

On presentation, the patient was quiet, alert, and responsive, weighed 17.3 kg, had a rectal temperature of 101 F, and pulse rate of 180 bpm. Cardiac auscultation revealed no

murmurs or arrhythmias, and femoral pulses were bilaterally strong and synchronous. The respiration rate (RR) was increased at 48 breaths per minute, with a mild increase in respiratory effort and harsh lung sounds bilaterally. The patient regurgitated during the exam and a wet cough was noted. Oral mucous membranes were pale pink and moist with a capillary refill time of less than 2 seconds. The abdomen was soft and non-painful on palpation, with no masses or organomegaly detected. Palpable peripheral lymph nodes were soft, small, and symmetric. The eyes, ears, and nares were clean and free of discharge. The neurological examination revealed that the patient was quiet but mentally appropriate, weakly ambulatory tetraparetic (could only take a few steps before fatiguing) and had a fatigable palpebral reflex. Based on neurologic and physical evaluation, the patient's problem list at the time of presentation included diffuse neuromuscular disease due to prior presumptive diagnosis of MG (with a tetraparesis worse in the pelvic limbs), regurgitation, ME, hypersalivation, AsP, and tachypnea (with mildly increased respiratory effort).

Veterinarian's differential diagnosis

The attending DACVIM (Neurology) agreed with the presumptive diagnosis of MG and AsP based on clinical history and the radiographs performed with the rDVM. The attending DACVIM (Neurology) suspected acquired MG rather than paraneoplastic or congenital MG given the patient history. As the clinical course of MG in a patient can wax and wane, although another neuromuscular condition may have caused similar signs, these causes were thought to be less likely.

Veterinarian's initial assessment of prognosis

The prognosis for a patient with MG is guarded to poor (Dewey & da Costa, 2016) with patients who present with regurgitation, like this patient, being less likely to go into clinical

remission (Forgash et al., 2021). Generally, the goal of treatment is to support the patient's clinical signs until the patient goes into spontaneous clinical remission (Lahunta et al., 2021). However, even during clinical remission, ME may persist, and therefore episodes of regurgitation and AsP may continue to impact quality of life.

Interventions

The patient was hospitalized for supportive care and therapeutic plasma exchange (TPE) with the goal of TPE being to address the patient's worsening of neuromuscular signs as the patient was not clinically tolerant of increasing doses of pyridostigmine. Upon intake, day 1, due to the patient's tachypnea and increased respiratory effort, the applicant measured the patient's saturation of peripheral oxygen (SpO2) via a patient side pulse oximeter. The SpO2 level was 94%, lower than the recorded SpO2 measurements for healthy patients which should be 97% or higher (Bassert & Thomas, 2014), and the applicant administered supplemental O2 to the patient at 2 L/min via placement of bilateral nasal cannulas. This resulted in an improved SpO2 level of 98% and the patient remained on supplemental O2 throughout the course of treatment. The attending DACVIM (Neurology) ordered a venous blood gas which revealed a decreased ionized magnesium of 0.40 mmol/L (reference range [ref] 0.43- 0.58 mmol/L), elevated lactate at 4.8 mmol/L (ref 0.9- 4.2 mmol/L), and elevated Gap (K) of 19.2 mmol/L (ref 8.5- 19 mmol/L). The decrease in ionized magnesium was suspected to be due to the patient's hypersalivation. The elevated lactate a marker of poor perfusion - likely a result of the AsP and poor O2 exchange within the lungs, and the mildly elevated Gap (K), which indicates the anion gap, was suspected to be due to regurgitation causing a mild metabolic acidosis. The attending DACVIM (Neurology) also ordered CXR and acetylcholine receptor (AChR) Ab titers measured. The CXR confirmed the rDVM diagnosis of ME and AsP while also ruling out any neoplastic changes

such as a thymoma. The applicant collected 4 mL of whole blood in a plain, anticoagulant free tube so that the serum could be separated and submitted to the University of California, San Diego Comparative Neuromuscular Laboratory for AChR Ab titers. This test generally has a 5-7d turnaround time but was collected as a baseline sample prior to TPE in order to objectively assess patient response to therapy.

For the TPE protocol, the patient required placement of a central venous catheter (CVC). Typically, CVCs are placed in sedated patients but given this patient's increased risk for aspiration, the CVC was placed under general anesthesia (GA). While under GA, the attending DACVIM (Neurology) discussed performing electrodiagnostic testing, specifically single fiber EMG and repetitive nerve stimulation, to further support the diagnosis of MG.

Prior to GA and CVC placement the applicant clipped and aseptically prepared the right lateral saphenous vein with chlorhexidine and isopropyl alcohol and placed a 20-gauge intravenous catheter (IVC). The patient was started on a balanced crystalloid solution IV at 61 mL/kg/d (44 ml/H) and the attending DACVIM (Neurology) ordered maropitant - an antiemetic at 17 mg (0.98 mg/kg) IV q24hr, metoclopramide -a gastrointestinal prokinetic agent used as an antiemetic at 17.5 mg (1.01 mg/kg) IV once, pantoprazole - a proton pump inhibitor used to increase gastric secretion pH at 17 mg (0.98 mg/kg) IV once, ampicillin sulbactam- a beta lactam antibiotic used to treat the AsP at 519 mg (30 mg/kg) IV q8hr given slowly over 15min, and atropine -an anticholinergic used to reduce secretions at 0.16 mg (0.009 mg/kg) SQ, in preparation for GA.

The applicant calculated and administered butorphanol- an opiate partial agonist used for sedation at 5.2 mg (0.3 mg/kg) IV as part of the pre-anesthetic plan. Induction of anesthesia was with propofol-a non-barbiturate injectable anesthetic at 100 mg (5.78 mg/kg) IV. The patient was

intubated with a size 47 French [Fr] endotracheal (ET) tube without difficulty and placement of the ET tube was confirmed via capnography and the depth of the endotracheal tube placement was confirmed via prior measurement and palpation. Once placed, the ET tube cuff was inflated, and isoflurane, a general anesthetic inhalant, was started at 1.5% with 1 L/min of O2. The monitoring of the anesthetic event was then relinquished to another licensed veterinary technician (LVT) so that the applicant could place the CVC.

The applicant had gathered the materials needed for the CVC (sterile gloves, sterile 0.9% saline, sterile syringes, and sterile 7 Fr x 20 cm length, and a double-lumen catheter kit which included a guide wire, vessel dilator, catheter, and suture). The applicant positioned the patient in right lateral recumbency with the head in slight extension. The skin overlying the left jugular vein was clipped and aseptically prepared with applications of chlorhexidine and isopropyl alcohol. The applicant placed the double lumen central venous catheter using manufacturer's guidelines. The applicant used 3.0 nylon suture to secure the CVC in place. During CVC placement, the patient regurgitated, and suction was used to clear the oral cavity. Following the procedure, the applicant ensured that the patient was positioned in sternal recumbency in an O2 cage with 40% supplemental O2, and the head elevated to minimize further aspiration. The applicant assisted the attending DACVIM (Neurology) with the first of six TPE treatments while the patient recovered from anesthesia.

Case management

On day 2 the attending DACVIM (Neurology) updated the treatment sheet to include the following treatments q24hr: wt measurement, maropitant 17 mg (0.98 mg/kg) IV q24hr and continued NPO orders due to regurgitation. Additional treatments q12hr included inspection of the IVC to ensure patency, documentation of the total volume of infused balanced crystalloid

solution, passive range of motion (PROM), and documentation of BP as measured via doppler. Treatments q8hr included ampicillin sulbactam 519 mg (30 mg/kg) IV given slowly over 15min, hyoscyamine -an anticholinergic used to decrease oral secretions at 0.06 mg (0.003 mg/kg) PO, and ondansetron -an antiemetic/anti-nausea medication at 8.6 mg (0.5 mg/kg) IV. Treatments q6hr included mentation monitoring, walking or carting outside (with documentation of urination and defecation while also working on standing and walking therapy), and neostigmine -an acetylcholinesterase inhibitor that prolongs the effects of acetylcholine at 0.8 mg (0.05 mg/kg) IM. The patient's recumbency was rotated q4hr. Treatments q1hr included documentation of vomit/ regurgitation events and documentation of supplemental O2 percentage. The applicant monitored three plasma transfusions throughout the day as part of the TPE protocol and documented the total volume infused (120 mL at 30 mL/H). The patient's physical and neurological examination remained static throughout the day.

On day 3 there were no changes to the patient's ordered treatments. The patient's physical and neurological examination remained static and in anticipation of the reintroduction of feeding, the applicant calculated the patient's RER and documented caloric intake needs. To calculate the RER, the applicant used the following formula: (30 X 17.3 kg) + 70 = 589 kilocalories (kcal) per d. The applicant divided the feedings equally into 3 meals with each meal containing 196.33 kcal (589 kcal ÷ 3 meals/d). The commercial canned diet had 315 kcal/ can with each can containing 13 oz (315 kcal ÷ 13 oz= 24.23 kcal/oz). Therefore, the applicant planned to feed 8.10oz (196.33 kcal ÷ 24.23 kcal/oz = 8.10 oz) per meal to achieve RER.

On day 4, the supplemental O2 percentage gradually tapered throughout the day. Unfortunately, upon decreasing to 30%, an LVT noted an increase in respiratory effort. At that time, the patient's SpO2 level was 95% and the attending DACVIM (Neurology) ordered the O2

be increased back to 40%. Amoxicillin and clavulanate potassium, at 250 mg (14.45 mg/kg) PO q12h was started in the evening and the ampicillin sulbactam 519 mg (30 mg/kg) IV was discontinued.

On day 5 the fluid rate of the balanced crystalloid solution was increased to 73.1 mL/kg/d (46 mL/H) due to patient weight loss (wt at 15.1kg from 17.3kg). During the evening examination, the attending DACVIM (Neurology) found the patient to have improved minimally throughout the day in response to the completed TPE treatments and the patient was able to be transferred out of the O2 cage and maintain a normal SpO2 level (above 98%) with O2 provided by a nasal cannula at 3 L/min. As the patient was able to walk further before fatiguing and had fewer episodes of regurgitation, feeding was restarted using a Bailey chair. The hyoscyamine was increased to 0.09 mg (0.006 mg/kg) PO q8hr to decrease secretions and oral pyridostigmine was restarted at 15 mg (1 mg/kg) PO q8hr as the neostigmine was discontinued.

On day 6 the was offered food q6h but ate a minimal amount throughout the day. The attending DACVIM (Neurology) also attempted to decrease the nasal O2 flow rate, but when the O2 rate was decreased to 2 L/min, the applicant soon noted a mentation change so the O2 was increased back to 3 L/min. The patient was able to walk a further distance without tiring but was still regurgitating frequently. The applicant noted that the patient was coughing up mucus and suction had to be used to dislodge a mucus plug from the airway. The attending DACVIM (Neurology) ordered CXR, which the applicant completed, and the radiographs showed a persistent alveolar pattern consistent with AsP and ME.

Final outcome

On the evening of day 6, the patient unexpectedly experienced respiratory arrest and died - likely related to a suspected silent regurgitation event – and was unable to be resuscitated. Necropsy findings noted abundant mucus was present in the bronchioles, consistent with AsP.

Discussion

Myasthenia gravis is a neuromuscular junction condition that can be acquired or congenital. As this patient was older at the time of onset than patients typically seen with congenital MG and of appropriate age for onset of acquired MG, the latter was suspected (Mignan et al., 2020). Acquired MG is most commonly due to an autoimmune disorder, where autoantibodies are produced and attach to the postsynaptic acetylcholine receptors, causing them to recede, lessening the number of available receptors. However, it can also be due to a paraneoplastic syndrome, most commonly a thymoma. As this patient had an extensive work up to evaluate for paraneoplastic causes, the more common autoimmune form was suspected.

Patients with MG may exhibit clinical signs of focal or generalized weakness. Patients who experience focal MG typically exhibit weakness in one or more focal skeletal muscle groups (such as facial, esophageal, pharyngeal, and laryngeal) without exhibiting signs of appendicular skeletal muscle weakness (Mignan et al., 2020). Given the generalized appendicular weakness of this patient, focal MG was ruled out.

Diagnosing MG is multifaceted with the following factors acting as integral roles: the patient's signalment, a neurologic examination consistent with diffuse neuromuscular dysfunction, regurgitation, increased salivation, and positive diagnostic tests including a positive edrophonium chloride challenge test, elevated AChR Ab titers, and/or electrodiagnostic changes (single fiber EMG and repetitive nerve stimulation). Edrophonium chloride is an ultra-short-acting-drug that blocks acetylcholinesterase, resulting in increased acetylcholine at the

neuromuscular junction. Elevated AChR Ab titers, ref greater than 0.6 nanomoles (nmol)/ L are considered positive serum titers for MG. If GA is safe for the patient, electrodiagnostic examination can also help diagnose MG. Results of single fiber EMG consistent with MG are characterized by an increased variation in the time from stimulation to the action potential (known as jitter). A decrementing reduction in wave amplitude of repetitive nerve stimulation tests may also support the presumptive diagnosis of MG (Dewey & da Costa, 2016). This patient's signalment, neurologic examination, prior positive edrophonium chloride challenge test, and documented elevated AChR Ab titers all supported the diagnosis of MG. While the patient was under GA to place the CVC, the attending DACVIM (Neurology) elected to not proceed with electrodiagnostic testing due to patient safety given the amount of regurgitation the patient was exhibiting.

From a patient care perspective, MG is a labor-intensive disease. Given the severity of clinical signs in this patient, constant monitoring was required including CVC care for TPE administration, special dietary needs regarding both caloric needs and physical assistance while eating/walking, and physical rehabilitation. The applicant was able to use advanced skills to monitor and assess the patient to recognize mentation changes and respiratory changes, resulting in changing the O2 supplementation and improving the patient's status. Due to the patient's ME, the patient needed to be positioned upright in a Bailey chair while eating or drinking and left in that position for at least 15min afterward to allow gravity to assist in emptying the esophageal contents into the stomach. The applicant was also responsible for calculating and ensuring the patient was fed their RER as knowing the RER was important given the secondary AsP (increasing energy demands) and overall depression the patient exhibited, preventing the patient from having a normal appetite. The applicant was able to use additional knowledge and

experience working with patients who require physical rehabilitation to assist in this patient's care, including rotating q4h to prevent decubital ulcers as well as PROM to prevent muscle contraction and changes to the ligaments, tendons, and joint capsules (Thomovsky, 2021).

The applicant played a vital role in the TPE treatments as well. The applicant was able to place and maintain a CVC, which was used to facilitate the six TPE treatments as well as other injectable treatments. TPE is used as an adjunct therapy for patients who are not responding to conventional medical treatment with an acetylcholinesterase inhibitor such as pyridostigmine and neostigmine. TPE was utilized in this case in order to decrease the immunoglobin levels (which are plasma bound) faster than the patient would be able to do with the assistance of acetylcholinesterase inhibitors or immunosuppressive medications alone (Vitalo et al., 2020). In some cases, immunosuppressive medications are prescribed in attempts to control clinical signs when acetylcholinesterase therapy is not enough, however, given the severity of the AsP this patient exhibited, further immunosuppression was contraindicated (Vitalo et al., 2020). As expected, the TPE therapy did decrease the AChR Ab titer results for this patient from 0.93 nmol/L to 0.62 nmol/L, (ref greater than 0.6 nmol/L is positive serum titer). Unfortunately, despite the applicant's advanced nursing skills and the decreasing AChR Ab titers following TPE treatments, the patient died in hospital. The applicant's advanced knowledge and skills were utilized throughout hospitalization; however, it did not change the outcome of this case given the poor prognosis. AsP is the most common MG related disease that causes 48.5% of patients to die or be euthanized (Forgash et al., 2021).

References

Bassert, J. M., & Thomas, J. A. (2014). *McCurnin's Clinical Textbook for Veterinary Technicians* (Eight Edition). Elsevier.

Dewey, C. W., & da Costa, R. C. (2016). *Practical Guide to Canine and Feline Neurology* (Third Edition). John Wiley & Sons, Inc.

Forgash, J. T., Chang, Y., Mittelman, N. S., Petesch, S., Benedicenti, L., Galban, E., Hammond, J. J., Glass, E. N., Barker, J. R., Shelton, G. D., Luo, J., & Garden, O. A. (2021). Clinical features and outcome of acquired myasthenia gravis in 94 dogs. *Journal of Veterinary Internal Medicine*, *35*(5), 2315–2326. https://doi.org/10.1111/jvim.16223

Lahunta, A. de, Glass, E., & Kent, M. (2021). *De Lahunta's veterinary neuroanatomy and clinical neurology*. Elsevier.

Mignan, T., Targett, M., & Lowrie, M. (2020). Classification of myasthenia gravis and congenital myasthenic syndromes in dogs and cats. *Journal of Veterinary Internal Medicine*, *34*(5), 1707–1717. https://doi.org/10.1111/jvim.15855

Thomovsky, S. A. (2021). The physiology associated with "bed rest" and inactivity and how it may relate to the veterinary patient with spinal cord injury and physical rehabilitation. F*rontiers in Veterinary Science*, *8*. https://doi.org/10.3389/fvets.2021.601914

Vitalo, A., Buckley, G., & Londoño, L. (2020). Therapeutic Plasma Exchange as adjunct therapy in 3 dogs with myasthenia gravis and myasthenia-like syndrome. *Journal of Veterinary Emergency and Critical Care*, *31*(1), 106–111. https://doi.org/10.1111/vec.13022

[redacted]

Owner:
[redacted]

RDVM:
[redacted]

Clinic:
[redacted]

Accession Number: C22-022110
Reference Number: 1724118
Received: Oct 12, 2022
Finalized: Oct 13, 2022
Species: Canine
Breed: Springer Spaniel
Sex: Male Neutered
Age: 1 Y
Animal ID: [redacted]
Case Ref #: 326364
Specimen: Blood

Nova

NOVA CCX Full panel

Specimen type	VENOUS				
pH	7.416			7.299 - 7.439	10/13/2022 10:46 AM
pCO2	27.2		mmHg	23.7 - 43.9	10/13/2022 10:46 AM
pO2	88.6		mmHg	48.1 - 235.3	10/13/2022 10:46 AM
SO2%	97.4			95.3 - 99.9	10/13/2022 10:46 AM
Hct	51		%	39 - 54	10/13/2022 10:46 AM
Hgb	17.1		g/dL	12.9 - 18.1	10/13/2022 10:46 AM
Sodium	149.5		mmol/L	143.0 - 151.1	10/13/2022 10:46 AM
Potassium	3.84		mmol/L	3.77 - 4.80	10/13/2022 10:46 AM
Chloride	116.5		nmol/L	110.5 - 118.8	10/13/2022 10:46 AM
Ionized Calcium	1.27		mmol/L	1.17 - 1.43	10/13/2022 10:46 AM
Ionized Magnesium	0.40	L	mmol/L	0.43 - 0.58	10/13/2022 10:46 AM
Glucose	114		mg/dl	70 - 114	10/13/2022 10:46 AM
Lac	4.8	H	mmol/L	0.9 - 4.2	10/13/2022 10:46 AM
BUN	13		mg/dl	9 - 27	10/13/2022 10:46 AM
Creatinine	0.9		mg/dl	0.7 - 1.5	10/13/2022 10:46 AM
TCO2	18.5		mmol/L	15.2 - 24.4	10/13/2022 10:46 AM
Gap(K)	19.2	H	mmol/L	8.5 - 19.0	10/13/2022 10:46 AM
BUN/Creat	13.4			9.6 - 21.5	10/13/2022 10:46 AM
BE-ecf	-7.1		mmol/L	-9.5 - -2.9	10/13/2022 10:46 AM
HCO3	17.6		mmol/L	14.5 - 23.1	10/13/2022 10:46 AM
O2Cap	23.8		mL/dL	17.9 - 25.1	10/13/2022 10:46 AM
Alveolar O2	113.6		mmHg	92.9 - 119.8	10/13/2022 10:46 AM

NOVA comment

Analyzed by Hospital personnel [redacted]

Reported By

Mcelreath MLT(ASCP), George

Report Date

October 13, 2022

UNIVERSITY of CALIFORNIA, SAN DIEGO
SCHOOL OF MEDICINE

Lab Director: G. Diane Shelton, DVM, Ph.D.
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Customer Information

[redacted]
Account No. 2129
[redacted]
[redacted]
[redacted]
[redacted]
[redacted]
Date Reported: 10/20/2022
[redacted]

Acetylcholine Receptor Ab-Myasthenia Gravis (905)

UCSD Number	358345 - 22
Owner	[redacted]
Patient	[redacted]
ID Number	326364
Lab Result (in nmol/l)	**0.93**

Reference/Interpretation

Canine Reference Values:	
< 0.6 nmol/L	Normal serum titer.
> 0.6 nmol/L	Positive serum titer, diagnostic of acquired myasthenia gravis.

Feline Reference Values:	
< 0.3 nmol/L	Normal serum titer.
> 0.3 nmol/L	Positive serum titer, diagnostic of acquired myasthenia gravis.

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Customer Information

[redacted]
Account No. 2129
[redacted]
[redacted]
[redacted]
[redacted]
[redacted]
Date Reported: 10/24/2022
[redacted]

Acetylcholine Receptor Ab-Myasthenia Gravis (905)

UCSD Number	358392 - 22
Owner	[redacted]
Patient	[redacted]
ID Number	326364
Lab Result (in nmol/l)	**0.62**

Reference/Interpretation

Canine ReferenceValues:	
< 0.6 nmol/L	Normal serum titer.
> 0.6 nmol/L	Positive serum titer, diagnostic of acquired myasthenia gravis.
Feline Reference Values:	
< 0.3 nmol/L	Normal serum titer.
> 0.3 nmol/L	Positive serum titer, diagnostic of acquired myasthenia gravis.

[redacted]

[redacted]

LABORATORY TEST REPORT

Owner:
[redacted]

RDVM:
[redacted]

Clinic:
[redacted]

Accession Number: A23-15323
Reference Number: 1726816
Case Coordinator: Dan R. Rissi
Received: Oct 19, 2022
Finalized: Oct 28, 2022
Species: Canine
Breed: Springer Spaniel
Sex: Male Neutered
Age: 1 Y
Animal ID: [redacted]
Case Ref #: 326364
Specimen: DOA

This report supersedes any previous reports issued for this case prior to 08/11/2023 at 3:09 PM

Pathology

Necropsy - Companion & Exotic Animals

Gross Pathology

A 1-year-old castrated male white and brown Springer spaniel dog was necropsied October 19th, 2022. The carcass weighed 14.8 kg and was mildly autolyzed.

External examination:
The body condition score was 2/5; the muscle mass index was 1/3. A microchip (#AVID 604-548-541) was scannable between the shoulder blades on dorsal midline. The antebrachia, crura, ventral neck, and lateral thorax were clipped.

Internal examination:
Thorax: All lung lobes were mottled red to dark red with the cranioventral lung lobes markedly firm. The trachea contained a moderate amount of red, viscous, opaque fluid.

No other significant changes were observed.

Histopathology

Lungs: Filling bronchioles and extensive areas of alveoli, obscuring interstitial architecture, are numerous neutrophils, epithelioid macrophages, and fewer multinucleated giant cells. Bronchioles also contain abundant mucous and occasional foreign material. In the background of alveoli is scant necrosis, edema, hemorrhage, and basophilic fragmented material (dystrophic mineralization). Few gram-positive cocci, occasionally arranged in pairs or chains, are found scattered intracellularly or extracellularly. In less affected lungs, alveolar spaces frequently contain edema and are variably collapsed (atelectasis).

There are no pathologic changes in the other tissue sections.

Diagnosis

1) Lungs: Subacute, marked, cranioventral, pyogranulomatous bronchopneumonia with few gram-positive cocci.

2) Whole body: Chronic, moderate, diffuse, muscle wasting.

Comments

Necropsy reveals a cranioventral centric pneumonia most consistent with aspiration pneumonia, likely secondary to the reported myasthenia gravis, which can cause regurgitation and aspiration of gastric contents.

Megaesophagus was not observed, but esophageal motility cannot be assessed postmortem. The muscle wasting is also consistent with the antemortem diagnosis of myasthenia gravis.

Reported By

Jesse Riker, DVM
Anatomic Pathology Resident

Reported By

Dan R. Rissi, DVM, MS, PhD, DACVP
Associate Professor