VMA OF NYC NEWSLETTER

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VMA NYC
VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

PRESIDENTS MESSAGE

Katherine Quesenberry, DVM

The VMANYC had a tremendous success as a sponsor at the New York Vet Show, held on November 7-8, at the Jacob Javits Convention Center. Our sponsorship of five local speakers contributed significantly to the event's success. The speakers were outstanding, the audience was highly engaged, and the sessions were well attended. We extend our sincere thanks and congratulations to Tony Miele, George Korin, and Linda Chiaverini for their exceptional efforts in organizing VMA NYC's participation and curating a stellar lineup of speakers from the New York City area: Dr. Joseph Impellizeri from Veterinary Oncology Services NYC, Dr. Becky Telle from the Veterinary Eye Center of NYC, Dr. Katrina Cusack from Garden State Veterinary Specialists, Dr. Anthony Fischetti from the Schwarzman Animal Medical Center, and Dr. Stephanie Reabel from BluePearl Pet Hospital.

The entire conference was a success, featuring insightful lectures from both local and national speakers on a variety of important and practical clinical topics. As a reminder, one of the many benefits of being a VMANYC member is free registration to the event, as well as the opportunity to earn up to 16 hours of continuing education (CE) credit, in addition to the CE credits available from our regular in-person meetings.

Inside this issue

President's message continued …

We are also excited to announce our annual holiday party, which will take place at The Ricky at the Dream Hotel on December 3rd, immediately following our regular meeting and lecture. Drs. Sarah Stephan and Thao Vo will present a CE lecture on Neurology. Be sure to mark your calendars for this festive event and take advantage of the opportunity to network and celebrate with colleagues in the veterinary community.

In addition to offering excellent continuing education, the VMA NYC remains actively involved in monitoring local and national legislative and regulatory changes that impact our profession. One such recent development is the passage of Proposition 129 in Colorado, which establishes a new midlevel veterinary practitioner role, the Veterinary Professional Associate (VPA). While it will take time for the specifics of this position to be fully defined, it is likely that other states will follow Colorado's lead. Colorado State University has already launched a master's level program for this position, and Lincoln Memorial University offers a similar program targeting veterinary technicians. The creation of the VPA role could have significant implications for the veterinary field, potentially mirroring the role of physician assistants in human medicine. Local veterinary organizations such as VMA NYC and NYS VMS will play an important role in any potential legislative changes in New York.

It has been both an honor and a privilege to serve as President of the VMANYC for 2024. We have made significant strides in expanding our membership and advancing our mission of providing education, advocacy, and community engagement for the NYC veterinary profession. I look forward to continuing my involvement with the VMA NYC in 2025.

Wishing you all a joyful and restful holiday season!

Warm regards,

Katherine Quesenberry, DVM, MPH, DABVP

Call for Committee Chairs

Are you ready to lead and make a lasting impact? We are seeking passionate and dedicated individuals to serve as Committee Chairs for the VMANYC.

As a Committee Chair, you will have the opportunity to:

- **Lead with Purpose:** Guide your committee to achieve its goals and contribute to the VMANYC.
- **Shape the Future:** Play a key role in decision-making and project execution.
- **Build Connections:** Collaborate with fellow leaders and members, expanding your professional and personal network.

Ideal candidates are enthusiastic, organized, and eager to inspire and support their committee members. Prior experience is welcome but not required - we're here to support your success.

If you're interested in this leadership opportunity, please Linda Chiaverini at 212-246-0057 or by email at lchiaverini@vmanyc.org.

We can't wait to see the incredible work we can accomplish with your leadership!

Calendar of Events

Program Committee **- Megan McGlinn, VMD**

The schedule of the VMA of NYC Continuing Education meetings and events for the 2024 / 2025 calendar year is listed below, including the speakers and topics.

December 3, 2024 - 6:00 - 8:00 pm

Speakers: Sarah Stephan, DVM, DACVIM / Thao Vo, DVM, DACVIM
Topic: Walking Through Neurolocalization in the Spine: Case Based Approach to Common Spinal Diseases / Mapping the Mind: Brain Localization and Common Disorders
Location: Empire Steak House West

December 3, 2024 - 8:00 - 11:00 pm - Holiday Party

Location: The Rickey at Dream Hotel

January 8, 2025 - 7:00 - 9:00 pm

Speaker: Christie Cornelius DVM, EMBA
Location: STATE Grill and Bar

May 7, 2025 - 7:00 - 9:00 pm

Speaker: Andrea Minella, DVM, PhD, DACVO

September 10, 2025 - 7:00 - 9:00 pm

Speaker: TBD

October 1, 2025 - 7:00 - 9:00 pm

Speaker: Howard Seim DVM, DACVS

November 6 - 7, 2025 - New York VET Show

Location: Javits Convention Center

December 2, 2025 - 6:00 - 8:00 pm

Speaker: TBD

December 2, 2025 - 8:00—11:00 pm - Holiday Party

If you have any suggestion for a continuing education speaker, timely topic, or event, please email the VMANYC at info@vmanyc.org.

Wellness Corner

Difficult Conversation with Coworkers

By Jennifer Tsung

Wouldn't life be so much easier if everyone at work got along all the time? Many of us spend so much time at work that our coworkers really are our work family. It is quite common to make solid friendships in the workplace and being happy around our work colleagues increases job satisfaction.

There will always be at least one person who we will have some degree of conflict with at work. This can annoy and distract us all the way to feeling eaten away by the toxic environment one or more people can cause. There are several types of difficult coworkers. I worked with a veterinarian in the past who reminded me of a beautiful animal called a sloth. I would get so annoyed as I was thinking that this person was purposely trying to not see cases or make phone calls so that I would end up taking up the slack when other staff members implored me to. I would think that she would go hide somewhere else in the hospital when the extremely ill animal or difficult client came in so that someone else would have to see that case. This went on for many months before I said anything to her. I had so much bottled-up frustration that I had to say something. I, like many, can find the stress of confrontation more painful than just doing the extra work. One thing that I have found over the years is that things are not always as they seem. It is always good to hear the other side. For this veterinarian, when we finally had a conversation, she told me about her severe anxiety. Minor things said to her would provoke these feelings where she ended up taking medication to try to calm her. I was then able to show more kindness to her after finding that out.

I feel that the hardest personality type to deal with at work is the gossiper. These are the staff members who are so personable and always chatting with everyone. The gossiper seems to enjoy turning innocent small talk into gossip which fuels uncomfortable situations at work. Even when we think this person is our friend, our little confidences are suddenly spun, and the entire hospital has a different version of the story. People who gossip can damage reputations and create a negative culture at work. One of the things I try to say to a gossiper is that I do not have an opinion on that topic. It is not easy to make gossipers accountable for what they say.

When we finally get to the point where we get over our fear of conflict and decide to have a difficult conversation, we should try to manage this skillfully. There are pointers to help us navigate these conversations.

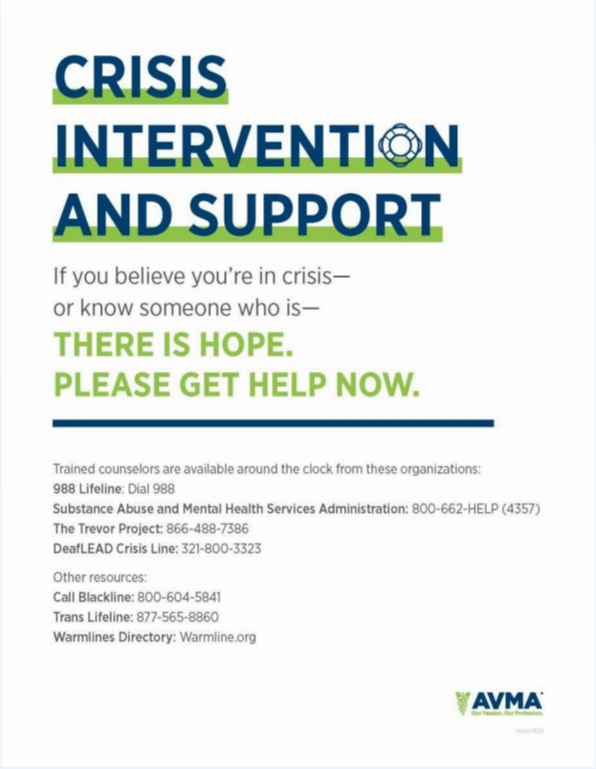
1. <u>Keep a professional mind frame and manage your emotions.</u> It is so easy to come in angry, scared, defensive, or hurt. We need to have some control before speaking.
2. <u>Gather the facts.</u> We need to share facts and examples of what we are concerned about.
3. <u>Do not put off a difficult conversation.</u> Over time, we will feel more anxious and not saying anything can make the other person feel that things are acceptable. I recently spoke to an assistant about his cat restraint. He was never scared to approach an aggressive cat but even with more gentle cats, he would at time scruff the cat a bit too roughly. Multiple staff members have noticed it, but no one wanted to talk to him about it. Unfortunately, it took me too much time to approach him. When I did, he asked me why I did not mention something much earlier.

Wellness Corner continued ...

4. Decide who should be involved in the conversation. Should it be one on one or should another team member be involved? One on one can seem less aggressive but there are times when it is good to have another person around.
5. Pick the right location. Do not have a tough conversation when clients or other staff members are able to walk nearby or hear what you are saying. The gossipers seem to want to be right outside the door to see if they can hear anything. Find a secluded location.
6. Use first person language. We want to communicate our perspective without seeming accusatory.
7. Listen actively with an open mind and be empathetic. Sometimes we hear things that we do not expect.
8. Speak your goal confidently. The point is to try to resolve an action or feelings. Is there any resolution that works for both sides?
9. Take a break. There are times when emotions escalate and there is no longer constructive conversation. When the conversation seems to keep repeating itself, it is time to take a break before we can not check our emotions.

Difficult conversations are difficult. The goal is to come out with a resolution that works for both sides. There are times when feelings will be hurt and these emotions should be checked on later. My ideal goal after a difficult conversation is for both sides to leave with dignity. There is a reason why most veterinarians prefer to deal with animals. Humans are complicated.

"The single biggest problem in communication is the illusion that it has taken place."
George Bernard Shaw



Paraneoplastic Disorders in Small Animals

By Brooke Britton, DVM, DACVIM (Oncology)

OVERVIEW OF PARANEOPLASTIC DISORDERS IN SMALL ANIMALS

Paraneoplastic syndromes (PNS) are changes in bodily structure or function that are not directly caused by cancer itself; rather, they arise secondary to tumor production of small molecules (such as hormones, cytokines, or peptides), tumor depletion of small molecules, or the host immune response to the cancer. PNS may precede a diagnosis of cancer as the first sign of disease, and, when recurrent, are typically a harbinger of relapse. While many PNS resolve with successful treatment of the underlying cancer, some syndromes persist despite tumor response to therapy, and may cause significant ongoing patient morbidity.

Broadly, PNS in small animals can be grouped into the following categories: gastrointestinal, endocrinologic, hematologic, cutaneous, neurologic, and miscellaneous (hypertrophic osteopathy, fever). Importantly, specific PNS are associated with relatively few cancer types, which allows the clinician to narrow the list of differentials when presented with these patients.

GASTROINTESTINAL PNS

Anorexia and cachexia

Anorexia and cachexia are common and debilitating PNS. Glycolysis and pyruvate production are critical to neoplastic cell proliferation - energy costly processes which contribute to increases in resting energy requirements and other metabolic changes in cancer patients, though alterations in resting energy and consumption are not uniform across the spectrum of malignancies. For example, alterations in glucose metabolism, increased protein turnover, and urinary protein loss in dogs with osteosarcoma; alterations in carbohydrate metabolism suggesting insulin resistance in dogs with lymphoma; and mild alterations in lipid metabolism in dogs with lymphoma, have been documented.[1,2]

Anorexia is often secondary to tumor-associated inflammation, and the production of cytokines (IL-1, IL-6) which interfere with bodily signaling involved in the regulation of appetite, resulting in appetite suppression.[3,4] Additionally, there may be pain associated with eating (ie, with oral or head/neck tumors), or dysfunctional gastrointestinal transit or obstruction (ie, in the case of GI cancers) which contributes to decreased intake, malabsorption, and/or loss of nutrients through vomiting or diarrhea. Loss of appetite may be compounded by chemotherapy-related side effects, including alteration or loss of taste (dysgeusia/ageusia) and smell (anosmia), which may persist for several weeks to months even after completion of a chemotherapy protocol.

Cachexia is characterized by severe metabolic derangement and profound muscle wasting in the face of adequate nutrient intake. TNF-alpha, IL-1 and IL-6 have been implicated in driving cancer cachexia by promoting insulin resistance, extensive lipolysis, and proteolysis of tissue stores.[3,4] Excessive cytokine stimulation induces anorexia, increases energy metabolism, and accelerates muscle wasting, producing an inexorable cycle of decline. The diagnostic criteria for cachexia in dogs and cats with cancer are not well-defined. True cachexia appears uncommon in dogs with non-hematopoietic cancer. However, while only approximately 4% of dogs are classified as emaciated (BCS 3/9 or lower) at diagnosis, muscle wasting is present in ~35%.[5] Additionally, ~14% of dogs will have lost 5-10% of their body weight and 23% of dogs >10% of their body weight in the 12 months prior to a cancer diagnosis.[5] Cats more commonly exhibit 'classic' cachexia with excessive lean muscle wasting in the advanced stages of their cancer. ~45-55% of cats with lymphoma or other (solid/non-hematopoietic) tumors have a BCS 5/9 or lower at diagnosis; muscle wasting is present in 93% of cats, including in 72% of cats with an 'adequate' BCS (5 or higher).[6] The majority of cachectic cats are those with diagnoses of GI lymphoma or oral squamous cell carcinoma.[a] Low BCS and low body weight are also negative

prognostic indicators in cats - across tumor types - portending a less durable response to treatment and lower overall survival times.[6]

Gastrointestinal ulceration

Mast cell tumor (MCT) is the most common cause of paraneoplastic gastrointestinal ulceration, and is secondary to tumor-associated hyperhistaminemia. Histamine release may be spontaneous, triggered by tumor manipulation, or as a result of degranulation induced by chemotherapy or radiation. Histamine stimulates gastric acid secretion via binding to gastric parietal cell H2 receptors, and exerts direct effects on the gastric mucosa causing increased vascular permeability and mucosal blood flow, in addition to protein exudation.[7] Plasma histamine concentrations are typically elevated in dogs with macroscopic MCT, however are not predictive of clinical signs of ulceration; therefore, it is possible that subclinical ulceration exists in a fair proportion of these patients. Cutaneous MCT in feline patients is more likely to behave benignly, as compared with the dog, where a wider spectrum of biologic behavior is noted. Cats are however more likely to have visceral involvement of their MCT (eg, spleen, liver, GI tract). Significant degranulation of cutaneous MCT in cats causing stomach upset and GI ulceration is rare in the author's experience, therefore cats presenting in GI distress with minimal cutaneous disease burden should be thoroughly staged with abdominal ultrasound, buffy coat, and liver and splenic aspirates to interrogate for evidence of visceral/systemic disease.

Gastrinoma, a gastrin-secreting neuroendocrine pancreatic tumor, is a rare additional cause of paraneoplastic gastroduodenal ulceration in both dogs and cats. These tumors are rare in the dog, and exceptionally rare in the cat. Gastrinoma almost always arise from the islet D-cells of the pancreas; rarely, they have been reported to arise within the duodenum. Zollinger-Ellison syndrome refers to the triad of a non-beta-cell neuroendocrine tumor, hypergastrinemia, and GI ulceration.[8] Gastrinomas are highly metastatic, with extra-pancreatic involvement appreciable in ~85% of dogs and cats at diagnosis.[8]

Treatment with gastroprotectants (H2 blockers, proton-pump inhibitors, sucralfate, and misoprostol) is warranted in dogs and cats as a preventative measure against ulceration, or to treat known or suspected paraneoplastic GI ulceration. Commonly utilized drugs and their dosages are detailed below.

H1 blockers (PRN, may be continued long-term if gross disease is present):
- Diphenhydramine: 2-4 mg/kg PO BID
- Chlorpheniramine: 0.22 – 0.5 mg/kg PO TID
- Cyproheptadine (cats): 1-2 mg per cat PO SID to BID

H2 blockers:
- Famotidine: 0.5-1 mg/kg PO SID to BID
- Cimetidine: 4-5.5 mg/kg PO TID

Proton pump inhibitors:
- Omeprazole: 0.5-1 mg/kg PO SID to BID, or IV SID

Gastroprotectants:
- Sucralfate: 250-1000mg PO TID
- Misoprostol: 2-5 mcg/kg PO BID to TID

ENDOCRINOLOGIC PNS

Hypercalcemia

Cancer is the most common cause of hypercalcemia in the dog, and the second most common cause in the cat. Malignancies most frequently associated with hypercalcemia include lymphoma (notably 35-55% of T-cell

ENDOCRINOLOGIC PNS

Hypercalcemia

lymphoma patients), leukemia, multiple myeloma, thymoma, carcinomas (primarily anal sac adenocarcinoma in dogs and squamous cell carcinoma in cats), and parathyroid tumors.[9] Paraneoplastic mechanisms of hypercalcemia include tumor production and release of soluble mediators (such as parathyroid hormone-related protein (PTHrp), IL-1, IL-6, and calcitriol) by tumor cells into circulation; release of osteoclast-activating factors (OAFs) by bony metastatic lesions; excessive production of 1,25-dihydroxyvitamin D; and excessive production of PTH.[9] Affected patients may demonstrate evidence of renal compromise or failure (PU/PD, isosthenuria or hyposthenuria on urine testing), in addition to a host of other non-specific signs (anorexia or other GI distress, lethargy, neurologic deficits, arrhythmias, etc). Overt evidence of cancer may be apparent on physical exam (eg, palpable anal sac mass, marked organomegaly, peripheral lymph node enlargement, skin lesions, oral mass in a cat).[b] Additional bloodwork abnormalities, such as a marked lymphocytosis, visible/circulating lymphoblasts on a blood smear, hyperglobulinemia, cytopenias, azotemia, and/or liver enzyme elevations may be present. Imaging may reveal a cranial mediastinal mass or metastatic pulmonary nodules, organomegaly, nodal or bony metastases, or neoplastic effusion.

When evaluating hypercalcemia in the dog and cat, it is vital to obtain an ionized calcium value in addition to the serum (total) calcium, as the ionized calcium more precisely characterizes the significance of the hypercalcemia. Ionized calcium values >1.4-1.5 mmol/L are considered in the hypercalcemic range. A hypercalcemia panel measuring PTH, vitamin D, and PTHrp levels, should be considered in any patient where physical examination, bloodwork and imaging findings are unrewarding in determining an immediate cause. PTH levels are typically low in the face of an elevated ionized calcium when cancer is present and renal function is normal. In the case of hypercalcemia secondary to parathyroid tumor (primary hyperparathyroidism), PTH levels are within the normal to high range; these results should prompt cervical ultrasound, if available, to investigate for a parathyroid mass. Elevated PTHrp levels are typically indicative of underlying cancer, though levels are usually non-detectable in the majority of patients in the author's experience, even when cancer is present.[10]

Treatment of paraneoplastic hypercalcemia typically centers on correction of hydration deficits, diuresis, antineoplastic chemotherapy where appropriate, and the use of steroids if warranted, +/- bisphosphonate therapy (eg, pamidronate, zoledronate). Fluid diuresis specifically with 0.9% NaCl should be performed, as the relatively high sodium content of this fluid competes with calcium for renal tubular absorption, therefore enhancing calciuresis. Bisphosphonates potently inhibit osteoclastic activity, and often result in rapid, marked reduction of calcium levels. This treatment is administered as an IV infusion, and is typically reserved for patients with moderate to severe hypercalcemia and good renal function. Steroids such as dexamethasone or prednisone should be reserved for cases of moderate to severe hypercalcemia where a definitive diagnosis of cancer has already been made, as the use of these medications may confound the ability to interpret diagnostic tests and confirm a diagnosis; steroids may also induce resistance to certain chemotherapy agents when used prematurely or inappropriately.

Suggestions for treatment of paraneoplastic hypercalcemia:
Mild hypercalcemia with minimal clinical signs

- Fluid diuresis with 0.9% NaCl – ideally IV, though subcutaneous fluids can be given if hospitalization is not feasible

Moderate to severe hypercalcemia +/- clinical signs

- Aggressive in-patient fluid diuresis with 0.9% NaCl IV (1.5-2x maintenance after correction of existing hydration deficits)

- Bisphosphonates:
  - ◊ *Zoledronate:* 0.1-0.25 mg/kg diluted in 60ml 0.9% NaCl IV over 15 minutes q2-4 weeks, not to exceed 4mg total dose per infusion); 100-1000x more potent than pamidronate and therefore preferred due to increased efficacy and far shorter infusion time[11]
- +/- Furosemide: 2-4 mg/kg IV, SC or PO q8-12 hours (in fully hydrated, renally-competent patients with refractory hypercalcemia)
- +/- Steroid therapy:
  - ◊ Prednisone: 1-2 mg/kg PO daily
  - ◊ Dexamethasone SP: 0.1-0.2 mg/kg IV daily (inpatient setting)

Hypoglycemia

The most common cause of paraneoplastic hypoglycemia in the dog is insulinoma, which arises from pancreatic beta cells. Functional pancreatic beta-cell tumors release excessive amounts of insulin, and may also release IGF-1 and IGF-2. The diagnosis of insulinoma is typically supported by identification of persistent, marked hypoglycemia on bloodwork (BG < 60 mg/dL), as well as an inappropriately normal or elevated serum insulin level in the presence of hypoglycemia as measured via insulin:glucose pairing. Abdominal imaging (ultrasound or CT scan) may reveal a pancreatic mass, however this is inconsistent, with <50% of canine insulinomas being readily identifiable on ultrasound;[12] very small or non-visible tumors are nevertheless capable of producing profound hypoglycemia. Insulinomas are highly metastatic tumors, with many patients having at least microscopic spread of disease at the time of diagnosis. Affected patients usually present with hypoglycemic seizures, lethargy/weakness, weight loss, or other non-specific signs referable to their tumor, though some patients are remarkably able to compensate for their hypoglycemia, particularly if longstanding, and may demonstrate only subtle if any clinical signs. Treatment of insulinoma involves surgical removal of the pancreatic mass if visualized, or exploratory laparotomy with partial pancreatectomy even if a mass is not seen. If surgery is not feasible (ie, in the case of widespread metastasis at diagnosis), or if hypoglycemia persists or recurs after surgical management, medical treatment of hypoglycemia with prednisone, diazoxide, octreotide, or the chemotherapeutic streptozotocin, can be pursued. Prednisone mitigates hypoglycemia by decreasing insulin sensitivity and increasing endogenous hepatic glucose production.[13] Diazoxide suppresses insulin release from beta cells, stimulates hepatic gluconeogenesis and glycogenolysis, and inhibits cellular uptake of glucose; response rates approach 70% in patients with insulinoma.[13] Octreotide inhibits synthesis and secretion of insulin by pancreatic beta cells, and resolves hypoglycemia in approximately 50% of dogs.[13] Streptozotocin requires intensive saline diuresis during administration due to its nephrotoxicity; additionally, it is a highly emetic drug, and can cause liver enzyme elevations and myelosuppression.[13] Given the potential for significant side effects and questionable long-term benefit of this therapy, streptozotocin is rarely used in current practice.

Suggestions for treatment of paraneoplastic hypoglycemia due to insulinoma:

- *Prednisone:* 0.25 mg/kg BID, escalating to 1 mg/kg/day if needed
- *Diazoxide:* starting dose of 5 mg/kg PO BID, increased gradually to 30 mg/kg/day if needed
- *Octreotide:* 10-50 ug SQ BID to TID

Finally, dietary modification can be employed to attempt to mitigate hypoglycemia. Balanced diets Including complex carbohydrates should be fed in small amounts frequently throughout the day; simple sugars are to be avoided.

Liver tumors (hepatocellular adenocarcinoma/adenoma) and smooth muscle tumors (leiomyosarcoma/ leiomyoma) arising from the stomach, spleen, liver, or intestinal tract cause hypoglycemia primarily via tumor cell

production of IGF-2.[14] Most non-islet-cell tumors causing hypoglycemia are larger and readily identifiable on abdominal palpation or imaging.[c] Surgery to remove the tumor (+/- chemotherapy post-operatively if warranted, as in the case of high-grade tumors), typically resolves the hypoglycemia.

Hyperestrogenism

Hyperestrogenism is most commonly associated with Sertoli cell tumors, occurring in 25-50% of dogs with this disease; approximately half of canine Sertoli cell tumors develop in cryptorchid testes.[15] This PNS has not been reported in cats. Other testicular tumors (seminoma, interstitial cell tumor) and ovarian granulosa cell tumor, can also cause hyperestrogenism. Hyperestrogenism occurs via direct estrogen production by neoplastic cells.[15] Clinical signs of hyperestrogenism may include symmetrical alopecia, epidermal thinning, gynecomastia, hyperpigmentation, penile atrophy, pendulous prepuce, galactorrhea, and prostatic atrophy or prostatomegaly.[d] Increased estrogen levels may also cause bone marrow toxicosis, characterized by progressive bone marrow hypoplasia or aplasia/pancytopenia, as increased estrogen levels significantly affect bone marrow cell differentiation and function. Clinical signs of hyperestrogenism typically resolve with tumor removal, however resolution may be gradual. For those dogs with severe myelotoxicosis at diagnosis leading to aplastic anemia, GI bleeding, weakness, tachycardia, epistaxis and infection or sepsis can occur, and mortality rates are high.

In dogs where signs of hyperestrogenism are present, Sertoli cell tumor must be a differential, regardless of whether a testicular mass is present, and imaging is essential to investigate for a cryptorchid testis. Hormone profiles in dogs may reveal significant elevations in plasma estradiol-17B levels; a low testosterone/estradiol ratio; low expression of 5-alpha-reductase type I; and/or low plasma testosterone.[16]

Treatment consists of surgical removal of the primary tumor, +/- additional anti-neoplastic therapy if residual disease or metastases are present. Clinical signs of hyperestrogenism typically improve or resolve with removal of the primary tumor, however this may take several months. Morbidity and mortality rates are high in dogs developing myelotoxicity.

Acromegaly

In cats, acromegaly is caused by excessive growth hormone (GH) secretion by a functional pituitary adenoma of the pars distalis.[17] It is more common in male cats, and there is no breed predisposition. Canine acromegaly by contrast is commonly caused by progestin-induced GH secretion by mammary ductal epithelium.[17] Mammary tumors and pituitary tumors are rare additional causes in the dog. Acromegaly is rare in canine patients. GH reduces insulin receptors and interferes with post-receptor signaling and processing; chronic exposure to GH results in overgrowth of connective tissues, bone and viscera.[17]

The majority of cats with acromegaly initially present with diabetes mellitus and insulin resistance, and over time, develop overgrowth of connective tissue, bone and viscera and organ dysfunction.[e] Many effects of GH are mediated by IGF-1; measurement of this hormone remains the most practical way to confirm feline hypersomatotropism in conjunction with physical examination and clinical findings. Long-term prognosis in cats with this disease is poor, however treatment options include in-home glucose monitoring and insulin therapy, radiation therapy, somatostatin analogs, and feeding a low-carbohydrate diet.

HEMATOLOGIC PNS

Anemia

Anemia is common in cancer patients, and may result from the cancer itself (eg, bone marrow/leukemic involvement of lymphoma), anemia of chronic disease, immune-mediated red blood cell destruction, hemorrhage from the tumor, or cumulative bone marrow suppression secondary to long-term chemotherapy. It is most commonly seen as a paraneoplastic syndrome in patients with lymphoma, leukemia, hemangiosarcoma, disseminated mast cell tumor, and histocytic sarcoma, however any patient with cancer may develop anemia. Anemia may be regenerative or non-regenerative at diagnosis, and the nature of the anemia may change over the course of the disease. Treatment of anemias range from proactive monitoring if the anemia is mild in nature, to aggressive hemodynamic support in the face of acute hemorrhage. Anemias secondary to chemotherapy and anemias of chronic disease are typically mild, while immune-mediated anemia, anemia due to hemorrhage or tumor involvement within the bone marrow, and anemia due to cumulative myelosuppression are typically moderate to severe. Patients may exhibit a wide variety of clinical signs referable to the severity of their anemia, including pale or icteric mucous membranes, tachycardia, tachypnea, weakness or collapse, anorexia, arrhythmia or heart murmur, palpable organomegaly, hematochezia, hematemesis, hematuria, or epistaxis.[f] Diagnostic testing including a blood smear, Coombs test, bone marrow aspiration, and feline retroviral testing may be helpful in further characterizing anemias. Treatment is typically with supportive care and blood products if transfusion is warranted if and until the underlying cancer can be successfully treated.

Thrombocytopenia

The most common malignancies associated with thrombocytopenia include hematologic cancers (ie lymphoma, leukemia, myeloma), mammary adenocarcinoma, nasal adenocarcinoma, mast cell tumor, hemangiosarcoma, fibrosarcoma, and histocytic sarcoma.[18] Thrombocytopenia in cancer patients can be due to decreased platelet production, increased destruction or consumption, sequestration, and loss by hemorrhage. Rarely, cytotoxic myelosuppression can also occur with chronic anti-tumor therapy. Patients with paraneoplastic thrombocytopenia may exhibit bruising and bleeding from and around tumors, petechiae or ecchymoses (usually at platelet counts <50 k/ul), or spontaneous bleeding (usually at platelet counts below 25k/ul).[g] Exercise intolerance and mentation abnormalities may also be evident in those patients with clinically significant bleeding due to thrombocytopenia. Patients with marked thrombocytopenia should be handled with care, minimizing blood draws and avoiding jugular sampling and intramuscular injections. NSAID medications should also be discontinued. Transfusions of platelet-rich or whole blood products may be considered in severe cases of thrombocytopenia, however the benefits of transfusion are short-lived (days) if the underlying cancer is not adequately addressed.

The goal of therapy in the thrombocytopenic cancer patient is to restore the platelet count to the threshold of a 'non-clinical' state, though thrombocytopenia may never fully resolve, particularly in the face of hematologic malignancies where there is marrow involvement or where an immune-mediated component secondary to the cancer is present.

Hyperglobulinemia

Hyperglobulinemia is commonly seen with multiple myeloma and myeloma-related disorders (MRD), plasma cell tumor, and lymphoma/leukemia, whereby tumor cells produce an overabundance of a single type or component of immunoglobulin (M component). This typically results in a monoclonal gammopathy, which can be confirmed via serum protein electrophoresis.[19] M components interfere with coagulation by coating platelets

and inhibiting aggregation, and can lead to hyperviscosity syndrome, whereby 'sludging' of blood in small vessels, ineffective oxygen and nutrient delivery to tissues, and coagulation abnormalities can occur.[19] The degree of hyperglobulinemia can be mild to severe. Severely affected patients may present with interstitial nephritis and signs of renal failure, mentation changes, seizures, retinal hemorrhage or detachment, blindness, heart enlargement or failure, or bleeding diathesis. Diagnostic testing including serum protein electrophoresis (SPE) and/or urine testing for Bence-Jones proteins (excretion of free immunoglobulin light chain detected in the urine) are useful in further characterizing hyperglobulinemia and supporting an underlying neoplastic process. Treatment of paraneoplastic hyperglobulinemia centers around treatment of the patient's cancer, however plasmapheresis may be considered in patients with hyperviscosity syndrome for more Immediate relief.

CUTANEOUS PNS

Nodular dermatofibrosis (ND)

Nodular dermatofibrosis (ND) is a rare but highly specific PNS associated with multiple slowly-growing cutaneous collagenous nodules, predominantly on the limbs, in association with bilateral renal cystadenocarcinomas or cystadenomas, and almost exclusively in German Shepherd dogs with a likely autosomal dominant inheritance. ND is associated with genetic mutation in the BHD gene on chromosome 5, which encodes for the protein folliculin.[20]

Patients with ND present with multifocal to diffuse, small, non-pruritic cutaneous nodules which on biopsy are consistent with well-differentiated dense collagen bundles in the dermis and subcutis.h There is a predominant limb distribution, with head, neck and trunk involvement developing in more advanced stages of the condition. ND typically precedes systemic signs of illness from tumor-induced renal failure or metastasis by months to years. Affected females typically have concurrent uterine leiomyomas. There is no known effective treatment for ND, and the mean time from development to death is ~2.5 years.[20]

Superficial necrolytic dermatitis (SND)

Superficial necrolytic dermatitis (SND) is seen most commonly in dogs with glucagon-secreting tumors of the liver and pancreas.[21] Dermatologic findings include erosions and ulcerations with alopecia, exudation from pressure points, feet, the flank, perineal area, ventrum, and oral cavity/muzzle, crusting, and hyperkeratosis of footpads (otherwise known as 'hepatocutaneous syndrome'). Footpad hyperkeratosis is the hallmark of this PNS and occurs in all patients. Lesions can be very painful and pruritic. Sustained gluconeogenesis and amino acid catabolism in patients with these tumors is thought to lead to hypoaminoacidemia, which in turn leads to epidermal protein depletion and keratinocyte necrolysis.[21] Most dogs present with metastatic disease, however resolution may occur with resection of solitary tumors. Amino acid infusions and somatostatin analogs have been reported for palliative treatment, to limited effect.

Feline paraneoplastic alopecia/feline exfoliative dermatitis

Feline paraneoplastic alopecia is a non-pruritic, symmetric, progressive alopecia reported in cats with pancreatic carcinoma and biliary carcinoma. Hair epilates easily, and skin is shiny and thin in appearance; affectation of the footpads is also common.[i] Feline exfoliative dermatitis has been reported as a PNS in cats with thymoma, and is characterized by non-pruritic scaling, crusting, and alopecia, eventually progressing to the neck, trunk and limbs with accumulation of keratosebaceous debris between the digits, within the nail beds, and in the ear canals. The underlying mechanism may be immune-mediated. Resolution of paraneoplastic alopecia is rare, given the aggressive and often metastatic nature of the underlying cancer. Exfoliative dermatitis may resolve with thymoma resection.

NEUROLOGIC PNS

Myasthenia gravis (MG)

Myasthenia gravis (MG) is an immune-mediated PNS resulting from antibody production against nicotinic acetylcholine (ACh) receptors within the neuromuscular junction, most commonly seen in dogs and cats with thymoma.[22, j] Other tumors reported to cause this PNS include osteosarcoma, oral sarcomas, cholangiocellular carcinoma, and non-epitheliotropic lymphoma. Generalized MG is usually associated with exercise-induced muscular weakness with concurrent weakness of the muscles of the esophagus, face, and pharynx/larynx. Definitive diagnosis is made via testing for circulating antibodies against the acetylcholine receptor. A positive edrophonium test is also supportive of the diagnosis.[22]

Surgical removal of thymoma or the underlying tumor is recommended, however post-operative Improvement is inconsistent. Anticholinesterase therapy with pyridostigmine bromide and immunosuppressive therapy are recommended prior to surgery where possible to reduce the risk of perioperative aspiration pneumonia.[22] Significant morbidity and mortality occur in patients with megaesophagus secondary to MG, even if the underlying tumor is removed.

MISCELLANEOUS PNS

Hypertrophic osteopathy (HO)

Hypertrophic osteopathy (HO) may occur with multiple different tumor types, however is most commonly associated with primary lung tumors. HO can also be seen with metastatic lung tumors (particularly from osteosarcoma), urogenital tumors (renal carcinomas, botryoid rhabdomyosarcoma of the bladder, and nephroblastoma), malignant schwannoma, and other carcinomas (eg esophageal, pancreatic). The pathophysiology of HO is not well-understood. It is exceptionally rare in the cat.

HO is characterized by an exuberant periosteal reaction which can be observed on radiographs, and is considered a painful condition; it is often associated with soft tissue swelling along the limbs in addition to lameness (sometimes shifting). The appearance of HO on radiographs is very unique, and, when recognized, should always prompt thoracic radiographs.k Treatment is centered on pain management and treatment of the underlying cancer; NSAIDS/opioids, gabapentin, amantadine, and potentially bisphosphonate therapy are typically employed to treat HO-associated pain.

Fever

Fever is a non-specific development that occurs in many veterinary cancer patients during the course of their disease. Paraneoplastic fever typically results from activation of the innate immune response against tumor antigens, or in response to tumor necrosis, and is mediated primarily by inflammatory cytokines that activate cellular cascades which ultimately impact the thermoregulatory center within the hypothalamus.[23] Concurrent infection of a tumor, perforation of gastrointestinal masses causing septic peritonitis, and leukopenia due to myelophthisis (secondary to involvement of hematologic cancer within the bone marrow) can also cause fever. These causes must be differentiated from fever that develops as a complication of cancer treatment (ie febrile neutropenia secondary to chemotherapy treatment, or urinary or respiratory tract infections secondary to myelosuppression from cancer treatment). Paraneoplastic fever is typically treated with specific antitumor therapy, such as chemotherapy, to address the underlying cancer. In the author's experience, paraneoplastic fevers in the absence of infection rarely exceed 103.5 degrees F, and typically wax and wane, such that specific medications aimed at fever reduction (eg NSAID therapy) are not additionally required.

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