There are 3 general categories of oncological emergencies:

- Problems primarily caused by the tumor itself
- Paraneoplastic problems
- Treatment-induced issues

Problems stemming from the tumor itself are not as common as treatment-induced or paraneoplastic issues. Primary problems that could occur are:

- Renal failure secondary to renal lymphoma or carcinoma
- Liver failure secondary to diffuse hepatic carcinoma
- Seizures secondary to intracranial masses
- Anemia or thrombocytopenia secondary to bone marrow neoplasia
- GI obstruction or bleeding secondary to GI adenocarcinoma or lymphoma
- Hemoabdomen secondary to ruptured hemangiosarcoma
- Fracture secondary to osteosarcoma
- Pneumothorax secondary to a ruptured bronchial carcinoma
- Epistaxis secondary to nasal tumors

Paraneoplastic syndromes are due to remote effects of the tumor and are often the first sign of cancer that the patient demonstrates. Paraneoplastic disorders are unrelated to the size of the tumor or presence of metastasis. They are usually a result of tumor production of hormones, hormonelike substances, or polypeptides. Paraneoplastic syndromes can often have a higher morbidity in the primary tumor itself.

*Hypercalcemia* is one of the most common paraneoplastic syndromes seen. It is caused by hematological neoplasias such as lymphoma (most common), leukemia, multiple myeloma, and bone marrow neoplasia. Non-hematological neoplastic causes include adenocarcinoma, (especially anal sac apocrine gland adenocarcinoma), mammary carcinoma, fibrosarcoma, and pancreatic neoplasia.

Hypercalcemia results from either a paracrine or humoral induced mechanism. In paracrine-mediated hypercalcemia the tumor cells in the bone release cytokines and growth factors, which act on the osteoblasts and osteoclasts to increase bone resorption.

Humoral mediated hypercalcemia is due to tumor cells releasing Parathyroid Hormone Related Proteins (PTHrP), which act on the bones to increase resorption as well as act in the kidney to increase renal tubular calcium reabsorption.

Clinical signs of hypercalcemia include PU/PD, nausea, vomiting, dehydration, and confusion, stupor, or coma.

Treatment initially consists of saline diuresis was 0.9% saline. This is used to increase the GFR as well as increase calcium excretion. Furosemide can be added to cause a calciuresis. It also inhibits calcium reabsorption in the ascending loop. However, do not use furosemide in a volume-depleted patient.

Calcitonin (4 to 6 units/kg sq q.8 hours) can be used to inhibit bone resorption. It also decreases intestinal calcium absorption and increases renal calcium excretion. However, this drug is used in the acute setting only, because the hypercalcemia can be refractory to this treatment after 12 to 48 hours.

Corticosteroids will inhibit prostaglandin E and osteoclast activating factor as well as induce calciuresis. The use of steroids may mask an accurate diagnosis and should not be used unless absolute necessary until a definitive diagnosis has been made.
The bisphosphonates will inhibit bone resorption by acting on the osteoclasts. They are most effective for the treatment of multiple myeloma, but are also good for solid tumors with bony metastasis. They are less effective for humoral mediated hypercalcemia (i.e., PTHrP mediated) disease, as they have no effect on renal tubular calcium resorption. They also can take 1–2 days to be effective so are not appropriate for treatment of an acute crisis. The most common bisphosphonate used in veterinary medicine is pamidronate, which can achieve normal calcium in 90% of patients.

There are multiple tumor types that may manifest with hypoglycemia. The most commonly thought of is an insulinoma; however, leiomyosarcomas are also implicated in causing hypoglycemia. Other possible tumor types seen with hypoglycemia include lymphoma, hemangiosarcoma, hepatocellular carcinoma, and oral melanoma. Hypoglycemia can also be seen in patients that have sepsis or liver failure secondary to neoplasia.

The mechanism of hypoglycemia in neoplastic conditions is speculated to be a combination of multiple factors. These include increased tumor glucose use, decreased liver glyconeogenesis or gluconeogenesis, increased insulin effects due to insulin-like growth factor, or the tumor may make glucagon suppression factor.

The clinical signs of hypoglycemia include weakness or lethargy, ataxia, shaking, progressing to seizures, and drooling, which is seen more frequently cats.

The most important treatment for hypoglycemia is to find and treat the underlying cause if possible. If there is no obvious cause, such as an insulinoma or leiomyosarcoma, a thorough search needs to be made for a septic focus. Liver failure must be strongly considered for all unexplained causes of hypoglycemia.

Frequent meals are very helpful for keeping the blood glucose levels elevated. Prednisone can be given to increase insulin resistance. Certainly dextrose supplementation may be necessary for the treatment of hypoglycemia, especially critically low glucose levels. However, extreme caution must be used with dextrose supplementation in a patient suspected to have an insulinoma. Aggressive supplementation of dextrose can induce the insulinoma to produce even higher levels of insulin, resulting in a drop in the blood glucose that may be even more severe than the initial hypoglycemia. Therefore, in a suspected insulinoma patient, it is advised to only give enough dextrose to prevent clinical signs of hypoglycemia and not to try to achieve euglycemia.

Bleeding problems in a neoplastic patient can also be split into primary bleeding versus secondary bleeding. Primary bleeding is seen when the tumor causes direct invasion of the vasculature. This includes GI bleeding secondary to gastrointestinal tumors, hemoabdomen secondary to a ruptured hemangiosarcoma, pericardial effusion secondary to heart base masses, or epistaxis secondary to nasal or pulmonary tumors. Secondary bleeding is due to DIC, thrombocytopenia, or liver failure.

Coagulopathies should be suspected if there is excessive bleeding from the venipuncture, or if petechia or ecchymosis is noted. Epistaxis, hematuria, or melena/hematochezia should prompt further investigation as well. Diagnostics include a CBC with a manual platelet count, and a PT/PTT. D-dimers are helpful in the diagnosis of DIC because they indicate the breakdown of an actual clot in the body. The disadvantage of D-dimers is that unless you are in a university setting, the results can take 3 to 5 days to get back, which is too long to be of any clinical usefulness. Bone marrow aspirates may be indicated in animals with thrombocytopenia and in which DIC has been ruled out.

Treatment of bleeding disorders depends on whether or not there is a clotting factor deficiency, or a platelet deficiency. In clotting factor deficiencies fresh frozen plasma or whole blood is indicated. Platelets cannot be successfully transfused using whole blood or standard plasma products. There is platelet concentrate available in some geographical areas; however, that is a very expensive product. GI bleeding can sometimes be reduced with the use of sucralfate and/or H2 blockers such as ranitidine or famotidine. Proton pump inhibitors such as omeprazole may be superior to the histamine blockers.

Treatment-induced complications that may result in an emergency room visit or hospitalization include acute tumor lysis syndrome, anaphylaxis, and neutropenia and/or sepsis.
Acute tumor lysis syndrome (ATLS) is due to rapid and massive cell death secondary to chemotherapy. It can also be seen in rapidly growing tumors that underwent spontaneous cell death. Additional risk factors include hypovolemia and diminished renal and liver function.

ATLS is due to release of intracellular potassium and phosphorus. Malignant lymphocytes have 4 times the normal phosphorus levels as well as an increased ATP content, which is why lymphocytic-based tumors are at higher risk for ATLS.

ATLS causes hyperkalemia, hyperphosphatemia, and hypocalcemia. Nephrocalcinosis can result in acute renal failure. Hyperkalemia can cause atrial standstill; therefore, patients need to be monitored for bradycardia, and an ECG should be run if there is any suspicion of atrial standstill. Hyperkalemia can be treated with saline diuresis, dextrose/insulin, and sodium bicarb. Aggressive fluid therapy should be instituted to try to prevent acute renal failure. Furosemide and mannitol therapy may be necessary if the renal failure becomes oliguric or anuric.

Prevention of ATLS is the best strategy. Recognize the patient at risk for ATLS, know which chemotherapy protocols are more likely to result in an ATLS crisis, and perform diligent monitoring. Any fluid deficits should be corrected prior to chemotherapy.

Anaphylaxis can be seen with manipulation of mast cell tumors as well after asparaginase, doxorubicin, and epirubicin therapy. Anaphylaxis is a type I hypersensitive reaction. There is histamine-mediated vasodilation and bronchoconstriction. There is also prostaglandin and leukotriene release, with resulting leaky capillaries and smooth muscle constriction. In cats anaphylaxis is usually manifested by severe dyspnea from bronchoconstriction and airway edema. In dogs there is an acute splanchnic congestion resulting in gastrointestinal signs. Because there is so much blood pooling in the splanchnic vasculature, these dogs will develop hypotensive shock with the classic signs of hypotension, pale mm, tachycardia, poor pulses, and collapse.

The treatment of anaphylaxis includes immediate vascular access for fluid therapy consisting of colloids and crystalloids. Oxygen therapy is indicated in cats with bronchoconstriction and airway edema. A bronchodilator such as terbutaline should be administered to cats as well. Epinephrine (0.1 ml/kg of 1:10,000) is necessary in severe anaphylactic reactions. Diphenhydramine (0.2–2.0 mg/kg not to exceed 50 mg IM) should be given. Dexamethasone SP 0.25–0.5 mg/kg IV) can be helpful if there is continued mediator release but is of questionable benefit in the acute crisis. THE MOST IMPORTANT thing to recognize is that patients with anaphylaxis die from hypotensive shock. Aggressive fluid therapy is critical to a positive outcome.

Neutropenia is defined by a neutrophil count less than 3000 µl/L. Sepsis is usually not seen unless counts drop below 1000 µl/L. Neutropenia is often seen 5 to 10 days after chemotherapy treatment. The agents that are usually implicated in causing neutropenia include cyclophosphamide, doxorubicin, mitoxantrone, and methotrexate.

The most common sites of infection that are seen secondary to neutropenia include gastrointestinal, urogenital, and respiratory. It is important to note that overt signs of inflammation may not be present. The most common signs of inflammation, such as redness, heat, and swelling, are neutrophil derived and without the presence of neutrophils these symptoms may not be evident.

Clinical signs depend on the site of the infection. Pneumonia results in dyspnea or coughing. Patients with a urinary tract infection may show hematuria or stranguria. GI infections manifest with vomiting or diarrhea. Nonspecific signs include fever, depression, and anorexia.

It is very important to differentiate a localized mild infection versus a patient that may be septic. The diagnosis of sepsis can be difficult without serial blood cultures, but there are clinical findings that make one suspicious of sepsis (see Table 1).
In a patient suspected to be septic, the minimum database includes a CBC and blood smear, PCV/TP, glucose, BUN, PT/PTT, and a urinalysis as well as urine culture. Complete diagnostics include a chemistry profile, thoracic radiographs, abdominal radiographs, or ultrasound. Blood pressures need to be done to monitor for hypotension. Blood cultures are needed for a definitive diagnosis and can help guide antibiotic selection. But they often take 3 to 5 days to get the results, too long to wait in a potentially septic patient.

Treatment of neutropenia depends on the severity of the neutropenia and clinical signs of the patient. Treatment needs to be tailored for each individual patient, but some general recommendations are shown in Table 2:

Antibiotics selected for neutropenic patient’s need to have a broad-spectrum coverage. Initial antibiotics can consist of either a penicillin or cefazolin combined with Baytril. An aminoglycoside can be substituted for the Baytril in a well-hydrated patient. In a patient suspected be septic that is taking prednisone, the prednisone may need to be reduced to physiologic dose of 0.1 to 0.2 mg/kg /day. Strict aseptic technique is extremely important in neutropenic patients. Sepsis can cause a profound hypotension; therefore aggressive fluid therapy may be needed. Septic patients are also at great risk for DIC, so PT/PTT need to be monitored and plasma may be indicated.

The goal is to have these patients at home as soon as possible, where there is much less risk of exposure to infectious diseases.