

# NEWS FROM THE WORLD OF ONCOLOGY

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## PARANEOPLASTIC SYNDROMES

The treatment of cancer patients has many aspects to consider beyond the tumour biology, appropriate treatment options and pharmacodynamics of chemotherapeutic agents. Often there are other complicating factors that also must be addressed. A common factor, which can influence cancer care for a patient, is the development of paraneoplastic syndromes (PNS). A PNS is a cancer related alteration in bodily structure or function that occurs at a site distant to the original tumour.

The causes of PNS are varied but often result in serious medical conditions requiring treatment prior to or at least concurrent with the inciting cancer. Once the underlying cause of the PNS has been addressed, the PNS will typically resolve. Interestingly, the PNS will often return in advance of clinical signs of cancer recurrence or end of remission. In this sense the PNS will often function as a sentinel for disease. There are many PNS. However, one of the most common PNS is hypercalcemia. This month I will discuss hypercalcemia of malignancy and next month continue the discussion with hypoglycemia and other PNS.



Hypercalcemia of malignancy is associated with many common tumours (lymphoma (LSA), anal sac apocrine gland adenocarcinoma (ACA), multiple myeloma (MM)). The etiology of the hypercalcemia is not uniform across all cancers. Hypercalcemia of malignancy is seen in about 25% of dogs with LSA, 25% of dogs with ACA and 20% of dogs with MM. The cause of hypercalcemia in both LSA and ACA is the tumour-associated production of parathyroid hormone related peptide (PTHrp). PTHrp differs by only a few amino acids from parathyroid hormone (PTH). The body is unable to differentiate between PTH and PTHrp and therefore all physiologic processes usually stimulated by PTH are induced. This includes increased absorption of calcium in the kidneys and from the GI tract, increased mobilization of calcium from bone and increased renal excretion of phosphorous. The result will be an animal with hypercalcemia and all associated physiologic and clinical complications (renal failure, CNS deficiency, PU/PD).

Multiple myeloma produces a molecule called the osteoclast-activating factor. This factor up regulates the function of osteoclasts, the cell response for the mobilization of calcium from bone. The result of increased levels of osteoclast activation factor is an elevation in circulating calcium levels. Other mechanisms of hypercalcemia of malignancy can include direct extension of tumour into bone (such as with osteosarcoma) or renal failure associated with direct tumour action or the development of glomerular nephritis.

In many cases the hypercalcemia is severe and must be immediately addressed prior to or while instituting treatment for the underlying cancer. Initial treatments for hypercalcemia include IV fluid diuresis with 0.9% NaCl and administration of calcitonin. Other treatments may include the use of furosemide and the use of prednisone. Furosemide should be used with caution and never given to a hypercalcemic patient who is also dehydrated. Prednisone should never be given to a patient who is suspected as having cancer but has not yet had a diagnosis made. This is because prednisone will cause a short-term remission of certain cancers such as LSA and MM. Once the hypercalcemia has been dealt with in the short term, long-term control will be achieved with appropriate cancer therapy. As previously mentioned, the return of a PNS is often the first indication that the cancer has come out of remission.

***Dr. Kevin Finora is a board certified Oncologist and Small Animal Internist. He sees patients Wednesday (including evenings) to Saturday at VEC/RC South. Please do not hesitate to contact Dr. Finora if you have any cancer related questions.***

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