Idiopathic hypercalcemia in domestic cats: a case report

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Introduction: Hypercalcemia can be due to several medical conditions⁽¹⁾. Recently, however, idiopathic hypercalcemia has become the most common diagnosis by exclusion, particularly in longhaired hypercalcemic cats⁽²⁾.

Animals, Materials and Methods: A neutered five-year old domestic longhair cat was presented with signs of partial anorexia, weight loss, and lethargy. Physical examination, blood and urine sampling, thoracic radiographs, abdominal ultrasound, as well as aspiration and cytology of the peripheral lymph nodes were performed. A complete blood count (CBC), a biochemistry panel (Chem), and analyses of ionized calcium (iCa), parathyroid hormone (PTH), PTH-related protein (PTH-rp), vitamin D, and a routine urinalysis were done. After 4, 10, and 16w, CBC and Chem were repeated, whereas analysis of iCa was repeated after 4, 13 and 22w.

Results and Discussion: At presentation, clinical examination was normal, apart from a body condition score (BCS) of 3 on a 9-point scale⁽³⁾. Serum total calcium (tCa) was moderately increased, whereas a mild azotemia, hypophosphatemia, and neutropenia were present. An elevated iCa (1.60mmol/l) coincided with low-normal PTH, PTH-rp, and vitamin D concentrations. Urinalysis, radiography, and lymph node cytology did not show any abnormalities, whereas ultrasound revealed bilateral renal abnormalities. Idiopathic hypercalcemia was diagnosed by exclusion of other known causes, and a calcium restricted diet was recommended. The cat had been on a dry diet, containing a high calcium (Ca) content (1.8% on dry matter basis (DM)). Due to the coinciding mild azotemia, the cat was gradually transitioned to a dry renal diet (0.73% DM Ca). An evaluation of the dietary intervention after 4w, revealed no changes in CBC, Chem, and iCa (1.59mmol/l). Therefore, 1.25mg/kg sid prednisolone therapy was instituted. After 10w of dietary change and 6w on prednisolone, the tCa normalized, neutropenia was absent, but a mild azotemia remained. Additionally, the water source was changed from tap to bottled, since tCa neared the upper limit of the reference interval. Thirtheen weeks after the initial diagnosis, iCa (1.41mmol/l) showed a major decrease, but was still slightly above the normal upper limit. At 16w post diagnosis, the tCa was mildly elevated, a mild neutropenia was present, however, no azotemia was observed. Finally, after 12w on bottled water, 18w on prednisolone, and 22w on renal diet, the iCa (1.28mmol/l) decreased, and was within the normal reference interval. Therefore, the dosage of prednisolone was decreased to 0.75mg/kg sid.

Conclusions: Restriction of dietary Ca intake (0.5g/d vs. 1.3g/d) for 4w did not decrease tCa or iCa, and short-term dietary intervention was not able to normalize azotemia. However, it has been suggested to evaluate dietary changes only after 6 to 8 weeks⁽²⁾. Furthermore, the calcium restriction in the diet might not have been sufficient, and feeding a diet lower in calcium might have been more appropriate at that time. However, due to the coexisting azotemia, a renal diet was necessary. A combination of dietary and medical intervention normalized serum Ca levels and resolved the azotemia. Furthermore, the calcium might be a beneficial additional strategy in controlling the blood calcium level in hypercalcemic cats.

⁽¹⁾Midkiff et al., 2000, J Vet Int Med 14, 619-626. ⁽²⁾Chew and Schenck, 2008. In: Kirk's current veterinary therapy, 14th ed., 236-240. ⁽³⁾Laflamme, 1997, Fel pract, 25, 13-17.