

NUTRITIONAL PATHOLOGY 101: COMMON POST MORTEM LESIONS OF NUTRITIONAL DEFICIENCIES AND EXCESS ACROSS TAXA

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ABSTRACT

The wide variety of lifestyle, digestive tract, and physiologic adaptations that exist across taxa result in an expansive array of nutritional needs and sensitivities, which can result in inadvertent essential nutrient excesses or deficiencies that result in disease (Barboza et al., 2009; Dierenfeld, 1997). While the incidence of nutrition related disease has seen a steady decline in veterinary pathology with the advent of specialists in comparative animal nutrition, these processes are seen with sufficient frequency that a basic knowledge of their post-mortem appearance is valuable in the evaluation of individual patients or in patterns of morbidity and mortality in larger groups of animals. Although primary deficiencies and excesses due to errors in dietary formulation, storage related degradation or intentional over supplementation often receive the most focus, secondary causes of nutrient deficiencies and excess, such as underlying diseases that disrupt intestinal absorption, systemic distribution, tissue storage or metabolic conversion can also be contributory (Kumar, et al., 2010). A review of all nutrition associated gross lesions across taxa is well beyond the scope of this presentation, therefore characteristic lesions and common organ system targets for nutrition related diseases have been selected. The focus of the discussion is the basic gross and histologic lesions consistent with potential nutrition related disease processes. Similarities and differences in lesion presentation across taxa (mammal, avian, reptile, amphibian and fish) will be highlighted. The selected lesions to be discussed include:

- Skeletal deformities due to deficiency in vitamin A, vitamin C, copper, or manganese and excess of vitamin A, vitamin D, or fluorine. The nutritional osteodystrophies (metabolic bone diseases) that result from vitamin D deficiency, calcium deficiency, calcium: phosphorus imbalance and nutritional secondary hyperparathyroidism (rickets, osteomalacia, osteopenia, fibrous osteodystrophy) will be reviewed with a view toward the common features demonstrated across taxa (Boyer, 2006; Edwards, 2000; Hoby, et al., 2010; Klasing and Austic, 2003; Tacon, 1992; Thompson, 2007; Ullrey, 2003; Wright, 2001).
- Nutritional myopathies due to vitamin E and selenium deficiency including the gross lesions which are common across all taxa as well as age related and taxon specific muscle targets (Dierenfeld, 1994; Gabor, 2005; Klasing and Austic, 2003; Tacon, 1992; Ullrey, 2003).
- Skin diseases including altered pigmentation of adnexal structures (hair, feathers, scales) and steatitis associated with fatty acid deficiencies, vitamin deficiencies (A, B complex, C,E), or deficiencies of copper and zinc (Ginn, et al., Hargis and Ginn, 2007; Hensel, 2010; Melinda and Jan, 2010; Miller, et al., 2001; Soto, et al., 2010; Tacon, 1992).

- Cardiovascular lesions associated with Vitamin E and selenium deficiency, hypervitaminosis D or hypercholesterolemia (Dierenfeld, 1994; Klasing and Austic, 2003; Van Vleet and Ferrans, 2007).
- Digestive system lesions, including odontodystrophies due to fluorine toxicosis, vitamin A deficiency, calcium deficiency, phosphorus deficiency or hepatic changes associated with excess dietary iron, copper or deficiencies of choline, cobalt and sulfur containing amino acids (Lowenstein and Munson, 1999; Wright and Whitaker, 2001).
- Neurologic and neurodegenerative diseases due to Vitamin A deficiency, Vitamin B1 (thiamin) deficiency, Vitamin E deficiency or copper deficiency (Boyer, 2006; Dierenfeld, 1994; Holz, et al., 2002; Klasing and Austic, 2003; Maxie and Youssef, 2007; Schaeffer and Waters, 1996; Summers, et al., 1995; Tacon, 1992; Zachary, 2007).
- The ocular effects of hypercholesterolemia (Clode, et al., 2002; Russel, et al., 1990; Wilcock, 2007; Wright and Whitaker, 2001).
- The systemic effects of hypovitaminosis A on specialized epithelial surfaces, including the sites of squamous metaplasia and the variation in affected sites across taxa (Boyer, 2006; Miller, et al., 2001; Tacon, 1992; Wright and Whitaker, 2001).

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