



## CASE OF INTEREST

### Copper Toxicosis in a Mouse Deer.

By Richard Fox, Veterinary Pathologist  
Case Donated by Malcolm Silkstone, Veterinary Pathologist

One of two rare Mouse Deer presented with bloody nasal discharge. Clinical signs also included abdominal pain on palpation and lethargy. Bloating was not noted. The animal then suffered cardiac failure and was resuscitated but then died shortly afterwards.

Post mortem examination did not reveal any obvious gross abnormalities. Histological investigation was performed on submitted tissues including liver, spleen, lung, myocardium and kidney.

On histologic examination of the kidney congestion was noted. Several sections of kidney displayed tubular degeneration and necrosis with haemoglobin-stained granular casts and the presence of haemoglobin pigment within tubular epithelial cells. Note: Mouse Deer erythrocytes are approximately 1/10th of the size of mammalian erythrocytes.

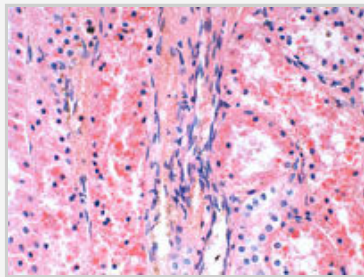


Figure 1. Histological section of the renal cortex displaying tubular necrosis with intra-tubular haemoglobin resorption droplets. HE Stain.

In the liver there was centrolobular to mid-zonal hepatocellular swelling with cytoplasmic vacuolation, the affected hepatocytes also contained abundant copper-like pigment. There were lesser amounts of lipofuscin-like pigment. Portal triads contain a few lymphocytes and plasma cells and pigmented macrophages.

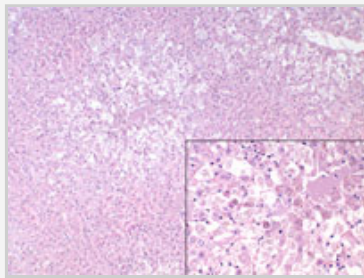


Figure 2. Histological section showing centrolobular to mid-zonal hepatocellular swelling with cytoplasmic vacuolation and affected hepatocytes containing abundant copper-like pigment. HE Stain.

Examination of urine revealed plentiful proteinaceous deposits and granular reddish casts consistent with haemoglobin casts.

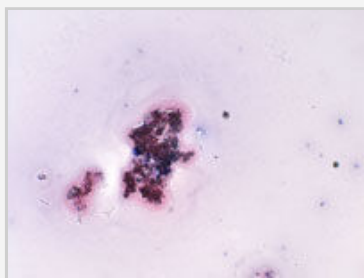


Figure 3. Photograph of urine sediment showing haemoglobin casts. HE Stain.

As a trace element, copper has a crucial role in mammalian metabolism, but it can be toxic in excess. The importance of a balanced copper homeostasis is illustrated by several copper-associated disorders in man and in a wide variety of animal models (eg, mice, dogs, and sheep). Proteins (copper transporters) involved in controlling copper metabolism have been well studied.

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## JOURNAL Articles(with e-links)

1. Kelly JM, Belding BA, Schaefer AK. Acanthomatous ameloblastoma in dogs treated with intralesional bleomycin. *Vet Comp Oncol.* 2010 Jun;8(2):81-6. [Link](#)

Acanthomatous ameloblastoma (AA) is a benign gingival tumour that often invades bone. This retrospective study evaluated the efficacy of intralesional (IL) bleomycin as a treatment for AA. Six dogs received weekly or bimonthly IL bleomycin injections (dose range, 10-20 U m(-2)). A seventh dog presented with advanced, nonresectable AA was treated palliatively. One to sixteen treatments were administered (median, 5). Six of the seven dogs had a complete response within 4 months from initial IL injection (median, 1.5 months), whereas the palliative case had approximately 25% decrease in tumour volume 14 days from initial injection. Local recurrence was not observed during the study period, with a median follow-up time of 842 days. Adverse effects were limited to wound formation with bone exposure (n = 4), mild tissue reactions (n = 3), local swelling (n = 2) and local infection (n = 1). The conclusions of this study show IL bleomycin is an effective treatment for canines with AA.

2. Leiva M, Peña T, Armengou L, Cesarini C, Monreal L. Uveal inflammation in septic newborn foals. *J Vet Intern Med.* 2010 Mar-Apr;24(2):391-7. [Link](#)

Septicemia in humans is described as a leading cause of uveitis, which eventually can induce blindness. Uveal inflammatory findings could be related to sepsis severity in newborn foals and might be used as an indirect indicator for survival. Seventy-four septic foals, 54 nonseptic foals, and 42 healthy foals. Prospective observational clinical study. A detailed blinded, ophthalmic examination was performed by boarded ophthalmologists on all admitted newborn foals. Foals were grouped as septic (when blood culture resulted positive or the sepsis score was > or =14), nonseptic, and controls. Based on blood culture results, the septic group was subdivided into bacteremic and nonbacteremic foals. Blood culture was performed in 62/74 septic foals, from which 35 (56%) were bacteremic and 27 (44%) were non-bacteremic. Anterior uveitis was diagnosed in a significantly (P < .005) higher number of septic/bacteremic foals (14/35, 40%) than in septic/nonbacteremic foals (5/27, 19%), nonseptic foals (4/54, 7%),

Copper storage disorders have also been observed in several dog breeds, such as Bedlington Terriers, Labrador Retrievers, West Highland White Terriers, and Doberman Pinschers. However, for some breeds it is still unclear whether the affected hepatic copper metabolism is the primary cause of hepatitis. The autosomal recessive disorder of copper toxicosis in Bedlington Terriers is at present the best-characterised copper storage disease. Copper toxicosis is caused by defective biliary copper excretion resulting in hepatic copper accumulation after chronic progressive hepatitis and cirrhosis. The onset is between 2 and 6 y of age and the clinical phenotype resembles Wilson disease, although neurologic defects are absent and affected animals have normal serum ceruloplasmin concentrations. The mutation in dogs affected with copper toxicosis is found in the *COMMD1* gene, has been identified, although some Bedlington Terriers with copper toxicosis in the United Kingdom and Australia were identified without this homozygous deletion. No other *COMMD1* mutations have been seen in these dogs, which suggests that other, as yet unknown, genetic defects are involved in copper toxicity in Bedlington Terriers without a *COMMD1* deletion.

Generally speaking, during the accumulation or pre-hemolytic phase, animals may be clinically normal, even with liver copper concentrations of 1,000 ppm, so long as increasing mitotic rate produces enough new hepatocytes to take up the copper released by dying cells. However, liver damage does occur during this period as indicated by increased levels of lactic dehydrogenase and AST. The second phase, or hemolytic crisis, lasts from hours to days and is characterised by the sudden onset of severe intravascular hemolysis and hemoglobinemia associated with increased blood copper levels, with resulting liver, kidney, and brain damage. The sudden increase in blood copper concentrations causes formation of methemoglobin and Heinz bodies as well as lipid peroxidation of erythrocyte membranes and intravascular hemolysis of damaged erythrocytes.

In this case it was not clear as to whether these Mouse Deer were susceptible to copper toxicosis due to an underlying abnormality in copper metabolism and uptake or if their diet contained excess copper or they ingested copper containing compounds.

References:

1. Fuentealba IC, Aburto EM. Animal models of copper-associated liver disease. *Comp Hepatol.* 2003 Apr 3;2(1):5. [Link](#)
2. Vonk WI, Wijmenga C, van de Sluis B. Relevance of animal models for understanding mammalian copper homeostasis. *Am J Clin Nutr.* 2008 Sep;88(3):840S-5S. [Link](#)

and control foals (0%). Anterior chamber fibrin was only observed in 4/14 (29%) septic/bacteremic foals with anterior uveitis. Anterior uveitis was also associated with posterior uveitis in 6/35 (19%) septic/bacteremic foals. The diagnosis of uveitis was related to nonsurvival ( $P = .001$ , odds ratio = 6.2, 95% confidence interval = 2.1-18.2). Anterior uveitis is highly prevalent in septic newborn foals, especially in those with a positive blood culture, and it should be considered as a survival prognostic factor.

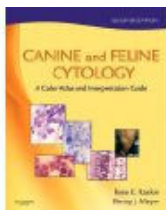
3. F. J. Ollivier, R. F. Sanchez, S. J. Monclin. *Equine glaucomas: A review* Equine Veterinary Education Volume 21, Issue 5, Date: May 2009, P 232-235. [Link](#) (Free PDF)

Glaucomas are diseases that lead to the destruction of retinal ganglion cells (RGC) and their axons via a number of mechanisms such as direct pressure damage, hypoxic and toxic injuries (Wilcock et al. 1991; Wilkie and Gilger 2004; Lassaline and Brooks 2005). These diseases have been recognised in several domestic species, including the horse, and clinical presentations vary widely between them. The glaucomas in horses are usually of slow progression with discreet signs of pain and insidious loss of vision; their estimated incidence in the United States is 0.07% (Miller et al. 1997; Wilkie et al. 2001; Brooks and Mathews 2007).

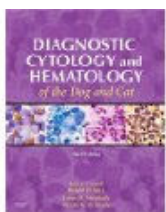
## SIDE STORY

### Two Clinical Veterinary Cytology Books

We have been asked recently what cytology reference book we have and use for our diagnostics. The following are an improvement on the previous editions with more images (picture spotting is always a good way to begin learning cytology) but also (as one gets more proficient at cytology) expanded texts on detailed cytological descriptions and disease characteristics.



Canine and Feline Cytology: A Color Atlas and Interpretation Guide by Rose E. Raskin and Denny Meyer - 2009



Diagnostic Cytology and Hematology of the Dog and Cat by Rick L. Cowell, Ronald D. Tyler, James H. Meinkoth, and Dennis B. DeNicola - 2007

## LATEST NEWS

### AVS - Diagnostic Clinical Microbiology Service (ACP)

Abbey Veterinary Services is pleased to announce the introduction of a full in-house diagnostic microbiology service by our new sister company Abbey Clinical Pathology.

We are now pleased be able to receive samples for microbiology, culture and sensitivity and faecal parasite examination for all species. This aspect of our work is the responsibility of Stephen Steen MSc, FIBMS, CHS (ABHI) who has extensive experience in all aspects of laboratory work and is a very well respected and very experienced microbiologist.



He is involved in on-site teaching of undergraduate students and helping in the development of treatment strategies with our clients.

Over the past 5 years he has obtained a very high reputation for the quality of his work and the clinical value of his comments on results. In the next few months, we hope to gradually introduce other tests and will soon be able to provide a full clinical pathology service to our clients. Please contact us for a **price list** within this newsletter.

## OUR DETAILS

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