PENICILLIUM GLABRUM GRANULOMA PRESENTING AS A HEART BASE MASS IN A CAT

L. Bosseler[*], S. Marynissen[†], P. Van Rooij[*], R. Ducatelle[*] and K. Chiers[*]

[*]*Department of Pathology, Bacteriology and Poultry Diseases, University of Ghent and* [†]*Department of Medicine and Clinical Biology of Small Animals, University of Ghent, Belgium*

Introduction: A 12-year-old cat was presented with a 3-week cough and tachypnoea. A CT-scan revealed the presence of a cranial mediastinal mass. The cat was humanely destroyed because of a suspected heart base tumour.

Materials and Methods: At necropsy examination, tissue samples were collected and processed. Sections were stained with HE. DNA was extracted from the heart base mass, amplified with an ITS1-4 primer and the resulting DNA fragments were sequenced.

Results: A firm, white mass (3 cm in diameter) was present at the base of the heart and attached to the right atrium and the right cranial lung lobe. Randomly dispersed white nodules, varying between 3 mm and 3 cm, were spread throughout the lungs. Histologically, the mass consisted of large areas of necrosis containing lightly basophilic, septate, 8 μm wide hyphae, surrounded by an eosinophilic granulomatous inflammation. At the periphery of the mass, several large bronchi with transmural necrosis and inflammation were seen. Both in these bronchi and in other lung sections, large numbers of embryonated nematode eggs and fewer larvae, morphologically consistent with *Aelurostrongylus abstrusus*, were present. DNA sequencing allowed identification of the fungus as *Penicillium glabrum*.

Conclusions: This is the first report of infection of an animal or human with *P. glabrum*, which is normally known as a food contaminant. The route of infection of *P. glabrum* remains elusive. However, the involvement of the lung in the granuloma and the extensive lungworm infestation suggests primary pulmonary damage due to lungworms after which the fungus found a portal of entry.

ATRIOVENTRICULAR VALVE DYSPLASIA IN COMMON FOXES (*VULPES VULPES*) WITH CARDIORESPIRATORY SYNDROME

T. Piasecki[*], M. Nowak[†], I. Janus[†], U. Pasławska[‡] and A. Noszczyk-Nowak[‡]

[*]*Department of Epizootiology with Clinic of Birds and Exotic Animals,* [†]*Department of Pathology and* [‡]*Department of Internal Medicine and Diseases with Clinic for Horses, Dogs and Cats, Wroclaw University of Environmental and Life Sciences, Wroclaw, Poland*

Introduction: Cardiorespiratory syndrome (also called lung–heart syndrome) among common foxes was described for the first time in the mid-1980s in Poland and Canada and has been reported as a cause of significant losses in fox farms. The aetiology of the disease still remains elusive. Our previous study suggests the possible role of heart defects in the aetiology of the disease.

Materials and Methods: The study was conducted on 50 foxes that died with signs of acute cardiorespiratory failure at a fox farm in the years 2012 and 2013. Special emphasis was put on detailed postmortem examination of the animals' hearts.

Results: The pathological examination of the hearts revealed dilation of the right atrioventricular opening and abnormal structure of the tricuspid valve in all cases. The septal cusp was shortened, thickened and immobilized. The chordae tendinae of that cusp were completely absent in 29 hearts (58%) or significantly shortened in another 21 cases (42%). Moreover, in 32% of animals abnormal structure of the mitral valve was noted.

Conclusions: The presence of tricuspid valve dysplasia in all examined animals suggests its role in the aetiopathogenesis of cardiorespiratory syndrome.

CHANGES IN IRON STATUS IN PERIPHERAL BLOOD AND LIVERS OF PIGS WITH TACHYCARDIA-INDUCED CARDIOMYOPATHY

J. Kiczak[*,‖], A. Orłowska[*], A. Tomaszek[†,‖], P. Kuropka[‡], J. Bania[§,‖], M. Zacharski[*,‖], I. Rybinska[‖], A. Janiszewski[¶,‖], A. Noszczyk-Nowak[¶,‖], U. Paslawska[¶,‖], M. Kasztura[#,‖], E.A. Jankowska[#,‖] and P. Ponikowski[#,‖]

[*]*Department of Biochemistry, Pharmacology and Toxicology,* [†]*Department of Pathology,* [‡]*Department of Animal Physiology and Biostructure,* [§]*Department of Food Hygiene and Consumer Health Protection,* [¶]*Department of Internal Diseases and Clinic of Diseases of Horses, Dogs and Cats, Faculty of Veterinary Medicine, Wroclaw University of Environmental and Life Sciences,* [‖]*Regional Specialist Hospital in Wroclaw, Research and Development Centre and* [#]*Wroclaw Medical University, Department of Heart Diseases, Poland*

Introduction: Changes in iron status occurring during the natural history of heart failure (HF) and related mechanisms remain unknown. We examined iron status using a porcine model of tachycardia-induced cardiomyopathy.

Materials and Methods: Homogeneous male siblings of large white pigs ($n = 12$) underwent continuous right ventricular pacing (RV) at 170 bpm, and four sham-operated subjects served as controls. All pigs were killed at subsequent stages of the disease (i.e. mild, moderate and severe HF). We analyzed indices of iron status in the peripheral blood, Fe^{3+} in liver sections (Perl's Prussian blue staining) and hepatic expression of a ubiquitous intracellular protein that stores iron and releases it in a controlled fashion (i.e. ferritin light [FTL] and heavy [FTH] chains) by immunohistochemistry.

Results: Local passive congestion was seen in the liver in pigs with mild HF, while in moderate and severe HF marked passive congestion with areas of necrosis was observed. The progression of HF was accompanied by a gradual decrease of Fe^{3+} in hepatocytes ($R = -0.921$, $P = 0.00015$). HF was correlated inversely with transferrin saturation (TSAT) ($R = -0.5699$, $P = 0.01$), a marker of biological iron availability, when TSAT was associated with hepatic Fe^{3+} content ($R = 0.833$, $P = 0.005$). Ferritin (both FTL and HTL) in hepatocytes was decreased in severe HF.

Conclusions: The development of HF due to RV pacing is accompanied by decreased hepatic Fe^{3+} content linked with lower biological iron availability as well as reduced hepatic iron storing ability (in severe HF).

CARDIAC TRUNCUS ARTERIOSUS IN AN EASTERN BLACK RHINOCEROS (*DICEROS BICORNIS MICHAELI*)

J.M. Monné Rodríguez[*], J. Chantrey[*], S. Unwin[†] and R. Verin[*]

[*]*Section of Veterinary Pathology, School of Veterinary Science, University of Liverpool and* [†]*North of England Zoological Society, Chester Zoo, UK*

Introduction: A 1-month-old female Eastern black rhinoceros (*Diceros bicornis michaeli*), born in captivity at Chester Zoo (UK), was presented at 1 month old but below the average expected weight for an animal at birth. She was reported with sudden onset lethargy and laboured breathing. This rapidly progressed to cardiorespiratory arrest with no response to resuscitation attempts.

Materials and Methods: The carcass was subjected to post-mortem examination and a full set of tissues was submitted for histopathological examination.

Results: Post-mortem examination showed severe enlargement and rounding of the cardiac profile, moderate hydrothorax and severe pulmonary congestion. The heart had a single arterial trunk originating from the right ventricle and giving origin to the aorta. A large 5 × 4 cm irregular defect of the interventricular septum was also observed. The base of this arterial trunk exhibited a single valve characterized by three irregular and thickened leaflets from which a narrow fibrous sheet was continuous with the mitral valve through the septal defect. Approximately 3 cm distal from the valve, the left and right pulmonary arteries arose independently from either side of the trunk. Histological examination revealed marked myocardial degeneration and chronic pulmonary congestion with abundant alveolar haemosiderophages ('heart failure cells').

Conclusions: To our knowledge this is the first report describing cardiac malformation in an Eastern black rhinoceros. The morphological features of this malformation closely resemble type III truncus arteriosus in man, which is associated with an early embryological defect on the outflow tract division of the fetal heart.