Animal welfare, ethology and housing systems

Volume 9  Issue 3

Különszám/Special Issue

Gödöllő
2013
CAUSES LEADING TO DEATH OF CAPTIVE TRAGOPANS IN HUNGARIAN PRIVATE BREEDING COLONIES

Gál János, Mándoki Mira

Szent István University, Faculty of Veterinary Science, Department of Pathology, Division of Exotic and Wild Animal Medicine, Budapest 1078, István str. 2., Hungary
gal.janos@aotk.szie.hu

Abstract

The tragopans or horned pheasants are medium-sized pheasants whose natural habitats are to be found in the Himalayas, including North-Eastern India, Nepal, Burma, Northern Vietnam, China and Pakistan. Tragopans are among the rarest and most vulnerable species of the pheasant family. There are five species of tragopans described (Zheng et al., 1985, Zhang and Zheng, 2002). Among viral pathogens, corona viruses were confirmed to be found in pheasants by performing RT-PCR on oral and nasal swabs of birds showing respiratory signs (Cavanagh et al., 2002). The mortality of adult pheasants between 1995 and 1997 was investigated by Pennycott (2000) and nephritis due to pheasant corona virus infection was listed as the frequent cause of death. The susceptibility of Phasianiformes to infectious laryngotracheitis virus, an avian alpha herpes virus was examined. The infected pheasants suffered from severe haemorrhagic or fibrinous laryngotracheitis (Günther et al., 1997). The enteric syndrome, characterised by anorexia, diarrhoea, severe dehydration and increased mortality in 6-8-day-old pheasant chicks was investigated on a game farm in Italy (Legrottaglie et al., 1997). Three new pheasant rotavirus strains were detected in Hungary during 2008 (Ursu et al., 2009). The characteristics of Mycoplasma gallisepticum infection were investigated in backyard game bird operations in Slovenia (Bencina et al., 2003). The infection was associated with severe respiratory disease and they stated 20% mortality rate amongst pheasants. Hsieh (2009) diagnosed avian tuberculosis in a bird sanctuary in Taiwan in two of six Swinhoe’s pheasants (Lophura swinhoii) which pheasant is also close to extinction. Kearns (2003) outlines the clinical signs, diagnosis, epidemiology, treatment and control of avian mycobacteriosis. The isolated bacteria are Mycobacterium avium, M. genevense and M. tuberculosis. Most bird species show signs of this disease in the gastrointestinal tract and an example is given of a Cabot’s Tragopan with tuberculous granulomas in the intestine, cecum, liver and spleen. Salmonella Enterica serovar Agona was isolated from commercial pheasant flocks in 1995 and 2000 (Myoujin et al., 2003). Lal (1997) reported a large number of Heterakis tragopanis parasites in the intestines of a dead Satyr tragopan at a Zoological Garden in England in 1942. This report, in fact, outlines the discovery of that species of Heterakis. Analysing the causes of death in adult pheasants between 1995 and 1997, histomoniasis was diagnosed in six pheasants (Pennycott, 2000). Most of the affected birds were in good body condition, but had multiple coalescing yellow foci up to 1.5 cm in diameter on the surface and cut surfaces of the liver. Visvesvara (2010) investigated the sudden death of a five month old Temminck’s tragopan in a zoo in Ohio. Lesions included splenomegaly, diffuse 1mm white nodules throughout the liver, and both ceca were swollen and filled with a caseous material. Acanthamoeba spp. was observed in large numbers in the cultures from the liver. Unfortunately, there are not enough available data and reviews in literature about the diseases and fatal conditions in tragopans leading to death of these valuable animals.

Keywords: Fatal diseases, mortality, uricosis, tragopans, Tragopan sp.
Material and methods

Tragopan carcasses were collected on the territory of Hungary from 3 private pheasant keepers. The examination period lasted between 2004 and 2010. Tragopan carcasses originating from private breeders were dissected according to the regular dissection technique. After the dissection, tissue samples were taken and fixed in 8% neutral, buffered formaldehyde for histopathological examination, or stored at -80 °C for later PCR examinations. The samples were routinely processed, and embedded in paraffin. Five-micrometer sections were cut with a Reichert sled-type microtome (Vienna, Austria) and were stained with haematoxylin and eosin according to standard methods, for histological examination. The routine bacteriological examination was performed in aerob incubator on 37 °C from the degenerated organs. PCR tests for the identification of possible viral (infectious bronchitis and avian nephritis) and bacterial (Mycoplasma and Chlamydomphila) agents were run on the frozen samples as described previously by others (Cavanagh et al., 2002).

Results and discussion

21 tragopan carcasses from four private pheasant collectors throughout Hungary were examined. Temminck’s Tragopan and Satyr Tragopan made up 57% and 43% of the 21 Tragopans dissected, respectively. Blyth’s, Western and Cabot’s Tragopan were not included in this study. The genders were almost equally represented, with 52% males and 48% females. The mean age of the Tragopans was 3.5 years (1 year old was 14%, 2 years 29%, 3 years 33%, 4 years 14%, 5 years 5%, 6 or more years 5%). In this study 42% of Tragopans were found to have kidney lesions and visceral uricosis. 67% of the 9 birds diagnosed with uricosis were male. After checking the keeping conditions for non-infectious causes of gout (cold, fasting, lack of water, hypoxia, vitamin A-deficiency, mycotoxicosis) the samples were screened for pathogens known to induce uricosis in poultry. All samples were negative for both the avian corona (IBV) and the avian nephritis virus (ANV) by using PCR method, so the presence of viral (IBV or ANV) infection was not proven. The explanation of this can be either a viral infection in early age of the birds causing degenerative changes and leading to slow destruction of the kidney parenchyma or other non-infectious causes resulting in nephrosis such as former mycotoxicosis, vitamin A deficiency or thirst due to lack of water. According to this result the background of the important cause of death in Tragopans, the uricosis remains to be unidentified. Further investigations, such as infectious experiments would be needed to clarify the exact background, but due to the specific reproduction strategy (low number (3-5) of layed eggs per nest) it is difficult and expensive to set up statistically relevant experimental conditions. Obstruction of the gastro-intestinal tract was shown in one bird. Dissection of the female Satyr Tragopan revealed a partially decomposed mouse obstructing the isthmus between the proventriculus and the gizzard. In one case, obesity and pathological simple fatty degeneration of the liver was observed in a female Temminck’s Tragopan. The male Tragopans have strong sexual activity in the breeding season, and terrorize the immature females. In our cases all females were in inactive reproduction cycle and the females were not accepting the Tragopan cock. Cannibalism was the cause of death in 9% of the cases and 100% (50% Temminck’s and 50% Satyr) of affected Tragopans were female. Pneumonia and air sacculturitis were detected in 5% of the cases. In other samples taken from the affected parts of the lung of a male Satyr Tragopan Chlamydomphila psittaci was found to be the causative agent. Lung and air sac mycosis were also accounted for 5% of the cases. Tragopans are very sensitive for noises and foreign sounds. These effects cause disorientation of
the birds and flustered flying in the cage, especially at night. The result is severe trauma and massive bleeding in an unfortunate case. Haematoma and posttraumatic haemorrhagic anaemia was shown in 5% of the animals as in a male Temminck’s Tragopan dissection revealed a large haematoma between the superficial and deep pectoral muscles. Reproductive tract disease was observed in 5% of the dissected birds. Dissection of a female Temminck’s Tragopan showed an enlarged, misshapen oviduct with greenish-grey discoulouration, and chronic fibrinous inflammation. The surrounding peritoneum also showed chronic fibrinous inflammation. The enlarged oviduct was opened, pseudo-concretion from an egg accounted for the secondary coelomitis. Acute serous rhinitis was described in 5% of the dissected birds. Transparent mucoid exudate was found in the naso- and oropharyngeal cavity of the examined male Satyr Tragopan. In samples taken for bacterial culture and PCR test Mycoplasma sp. was found to be the causative agent. Enteritis and dehydration were accounted for 5% of results. In samples taken from a female Satyr Tragopan Escherichia coli was detected as the causative agent. Tuberculosis was diagnosed in 9% of the dissected Tragopans and was caused by Mycobacterium avium. All of the affected birds were male Temminck’s Tragopans. Kearns (2003) studied avian mycobacteriosis in birds with tubercules in the intestine, ceca, liver and spleen and proved M. avium, M. genevense and M. tuberculosis in several avian species including a Cabot’s Tragopan.

The collection of these results draws the attention to the diverse pathological conditions leading to death in Tragopans. The role of veterinarians in informing the breeders on the prevention is very important to save these expensive and endangered birds. Some of the causes are environmental and can easily be avoided by proper breeding and keeping conditions, but the importance of screening for infectious agents and quarantine of the new birds is also highlighted considering the numerous pathogens causing the mortality of captive Tragopans.

References


