CASE OF INTEREST

Herpesvirus in Tortoises

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Herpesviruses have been detected in a wide range of vertebrates. In chelonians Herpesvirus infections have been identified in at least seven genera (Gopherus, Chelonia, Geochelone, Testudo, Graptemys, Clemmys, Chrysemys) and in five types of tortoises: Greek (Testudo graeca), Horsfield (Testudo horsfieldi), Hermann’s (Testudo hermanni), desert (Gopherus agassizii), and Argentine tortoises (Geochelone chilensis).

Herpesvirus in chelonia is caused by Chelonid Herpesvirus (ChHV). Common terms include "stomatitis-rhinitis" characterised by necrotizing lesions of the oral mucosa (dipheritic plaques), with a yellow-white pseudomembrane extending often from the caudal half of the tongue to the caudal pharynx and epiglottal area. These lesions impair food prehension and swallowing. These plaques often have a clyclical appereance and resolution. Rhinitis and conjunctivitis are also common features seen in Herpesvirus. A clear serous nasal discharge is present which often progresses to a mucopurulent discharge. Eyelids are often swollen with serous to mucoid ocular discharge.

Diagnosis of herpesviral infection is generally based on the presence of intranuclear inclusion bodies, electron microscopic identification of viral particles, isolation of the virus, and/or immunohistochemical confirmation using anti-herpesvirus antibodies.

At necropsy, in addition to the oropharyngeal lesions, enlargement and ecchymosis of the liver can be observed as well as pseudomembrane formation in the stomach.

Histopathology of the mucosal epithelium from the tongue to the pharyngolaryngeal region reveal diffuse areas of degeneration and necrosis, with an accumulation of necrotic cellular debris and fibrin on the surface (a pseudomembrane) (Fig 1). The mucosa is usually infiltrated by mixed inflammatory cells. Polymorphic eosinophilic or amphophilic inclusion bodies are visible in the nuclei of mucosal epithelial cells, in some cases occupying the entire nucleus (Figs. 2, 4).

Figure 1. Histological section of the dorsal hard palate. Note the ulceration and the presence of surface necrotic cell debris (Pseudomembrane) (x5 obj.). HE Stain.

Figure 2. Histological section of the dorsal hard palate. Note the intranuclear amphophilic inclusion bodies in the nuclei of sloughed glandular and squamous epithelial cells. (x65 obj.). HE Stain.

Similar lesions are also present in the liver, spleen, esophagus, stomach, cerebrum, and lungs (Fig 3 & 4), adrenal glands, kidneys, duodenum, jejunum, colon, and pancreas of tortoise.

Figure 3. Histological section of the lung with plentiful intra-lumen granulocytic and necrotic exudate (x2 obj.). HE Stain.
Tortoises that survive the primary infecton are likely to host the virus for life and consideration of the overall health status of the tortoise is important. The presence of serum neutralizing antibodies does not block the appearance of clinical signs. Therefore separation of exposed/infected animals from unexposed individuals is important and effective environmental cleaning is also paramount.

References:
1. Y. Une; K. Uemura; Y. Nakano; J. Kamile; T. Ishibashi; Y. Nomura. Herpesvirus infection in tortoises (Malacochersus tornieri and Testudo horsfieldii). Vet Pathol 1999 36: 624-627

LATEST NEWS
Gene Therapy Improves Survival And Quality Of Life Of Dogs With Cancer

The therapy uses a nonviral DNA molecule, called a plasmid, which encodes for growth hormone-releasing hormone (GHRH). This stimulates the endogenous growth hormone and another growth substance, insulin-like growth factor-1 (IGF-1), which have anabolic effects.

The therapeutic process involves injecting the DNA fragment into a muscle and applying electroporation—short, mild, controlled electric fields—in the area of the injection. It opens the cell membrane pores and traps the DNA inside the cells, which allows the production of GHRH. This thwarts the body’s natural process of eliminating a foreign body, in this case the DNA molecule.

The researchers tested the gene therapy in 55 companion dogs that had cancer and anemia and were receiving cancer treatment. Three months after the injection, 54 percent of the dogs had responded to gene therapy, as apparent on blood testing. Dogs that responded to therapy survived 84 percent longer, compared with dogs that did not respond to gene therapy and untreated control dogs that received a placebo injection. Although the response rate dropped to 47 percent at 4 months, it was still 22 percent higher than in control dogs.

Additional info: External Link

SIDE STORY
Metillin-resistant Staphylococcus aureus in a veterinary orthopaedic referral hospital: staff nasal colonisation and incidence of clinical cases.

Nasal bacterial swabs were collected from veterinary staff and environmental surfaces swabbed at six monthly intervals for metillin-resistant Staphylococcus aureus monitoring over an 18 month period. The incidence of metillin-resistant Staphylococcus aureus-associated postoperative wound complications of two veterinary orthopaedic surgeons was reviewed for a period when one was positive for nasal metillin-resistant Staphylococcus aureus.

Metillin-resistant Staphylococcus aureus was isolated from a maximum of two out of 10 staff on each occasion. The persistently infected clinician was primary surgeon in 180 cases, of which four veterinary patients were reviewed by a single individual, and another surgeon developed metillin-resistant Staphylococcus aureus-associated complications. None of 141 operations led by the other surgeon developed metillin-resistant Staphylococcus aureus-associated complications.

Veterinary workers are at increased risk for metillin-resistant Staphylococcus aureus colonisation, so it is likely that many veterinary patients are treated by metillin-resistant Staphylococcus aureus-positive staff. Nasal colonisation of veterinary surgeons with metillin-resistant Staphylococcus aureus appears to present only a small risk to their patients when appropriate infection control procedures are followed.

Read More: External Link

BIOPSY TIPS
Liver Cytology
- Cytology of the liver is not suitable for any disease in which the histological structure is required for proper judgement. This applies to the vast majority of liver diseases. FNA is useful only if it is possible for the diagnosis to be made on single isolated cells. This applies to the identification of tumour cells from a local lesion, or changes which are diffusely present in all hepatocytes such as steatosis (fatty liver) of steroid hepatopathy. Possible underlying liver pathology will, however, not be identified*

This is also applicable to most cases in which visceral pathology is suspected including aspiration of the kidney, intestine, spleen as well as the lung.