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ENTERIC, CARDIOPULMONARY AND MIXED FORMS OF CANINE PARVOVIROSIS. HISTOPATHOLOGICAL STUDY AND ANALYTICAL DETERMINATIONS

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The relationship between canine parvovirus (CPV-2) and the gastroenteric syndrome in dogs seems quite clear (Fernández et al., 1987). Cases of myocarditis caused by parvovirus have also been widely reported (Dotta and Guarda, 1980). Little information is available, however, concerning the mixed form of parvovirosis (Cammarata et al., 1981). The present study aims to provide further information on this third type of canine parvovirosis.

Material and methods

57 dogs (Dobermann, Cocker spaniel and Alsatian) ranging in age from 40 to 120 days were used for this study. A rapid diagnosis of virosis was made using electron microscopy for faecal analysis by the negative staining technique described by Fernandez et al. (1989). Once the diagnosis was confirmed, blood and urine samples were taken for analysis within 24 hours ante mortem.

Results

Physiopathological findings

	Control	Enteric	Myocardial	Mixed
Hemochromometric analysis				
Erythro. (10^6 mm)	7±0.4	4.5±0.3	6.3±0.3	5±0.4
Leuko. (10^3 mm)	11±0.5	3.3±0.5	5.3±0.5	4.2±0.4
Platelet (10^3 mm)	340±8.9	290±6.1	313±10	299±8
Hematocrit (%)	40.2±1.4	30.1±3.4	36.2±3.4	32.5±3.6
Hemogl. (g/dl)	15±2.2	9.8±1.2	12±0.8	11±1.1
Liver function tests				
Ttl.bil. (mg/dl)	0.40±0.09	0.65±0.04	0.46±0.05	0.54±0.07
Dir.Bil. (mg/dl)	0.12±0.02	0.22±0.08	0.14±0.03	0.18±0.02
GOT (IU)	30±2558.4	88±6.5	40±2.2	69±4.3
GPT (IU)	32±6.3	72±5.7	38±4.2	61±5.9
Alk.Phosph. (UI)	98±11.2	145±13.6	109±8.9	125±10.2
Arginase (UI)	0.03±0.01	0.2±0.04	0.09±0.03	0.15±0.03
Ttl.prot. (g/dl)	6.1±0.3	4.5±0.5	5.4±0.3	5.1±0.2
Kidney function tests				
pH	6.6±0.2	5.1±0.3	6±0.2	5.6±0.3
Density	1.035±0.4	1.051±0.2	1.038±0.2	1.046±0.2
Non-pr.nit. (g/l)	25±5	40±3.4	28±4.3	34±3.5
Nitro.urea (g/l)	16±4	30±4.2	19±3.1	26±2.5

Histopathological findings:

	Enteric	Myocardial	Mixed
Digestive System	Hemorrhagic gastroenteritis	Hyperemia in submucosa	Catarrhal gastroenteritis
Liver	Parenchymatous hepatitis	Centrolobular stasis	Stasis Apoptosis
Kidney	Serous G-nephritis Tubular necrosis		Tubulonephrosis
Mesenteric Lymph nod.	Lymphocytolysis in Germ. centres		Lymphocytolysis
Spleen		Hyperplastic Splenitis	Follicular Hyperplasia
Myocardium	Cell necrosis	Non-purulent	Non-purulent
	Interfasc.edema	myocarditis Intranuclear inclusions	myocarditis + Intense fibre destruction
Respirat. System		Acute Alveolar Edema	

Discussion

The specific histopathology of the enteric form (necrotic-hemorrhagic gastroenteritis), the myocardial form (non-purulent myocarditis with intranuclear inclusion corpuscles) and the mixed form (catarrhal enteritis and non-purulent myocarditis) in correlation with blood and urine tests allows three different clinical and pathological forms to be defined within canine parvovirosis.

References

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PATHOLOGY OF FIV-INFECTION

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The number of publications on FIV-infection in cats is already high although the virus is known only for 3 years. Detailed pathological and histological studies of alterations in FIV-infected animals, however, have only been published as case reports of very few cats and such an investigation in an at least somewhat more relevant number has not been published yet. Most papers which mention pathological and histological alterations do not give an estimation of the frequency of lesions and therefore give no clue which alterations might be interpreted as a hint for FIV-infection of cats at necropsy. The interpretation of findings in FIV-positive cats is furthermore complicated by the fact that due to the long asymptomatic carrier period

alterations in experimentally infected animals are not well known yet either. Additionally, experimentally infected cats usually do not have the same burden of opportunistic infections or other environmental stresses present in free roaming spontaneously infected animals.
Thus, at the moment only associations between FIV-infection and lesions frequently occurring in these animals can be described. Since FIV-positive cats are usually older than the average cat population at necropsy it is also difficult to differentiate between age-associated and FIV-associated findings. FIV-positive cats are often destroyed when the diagnosis «FIV infection» is made. Statistical evaluation of FIV-associated lesions is blurred by the fact that false positive as well as false negative results occur in ELISA. Furthermore, the cats are destroyed or die when being in different phases of the infection (acute phase, asymptomatic carried, ARC, AIDS) which may result in completely different alterations (e.g. follicular hyperplasia and dysplasia in lymph nodes versus hypocellular follicles and nodes).
We had the opportunity to investigate carcasses and/or tissues of 25 cats for whom FIV-infection was diagnosed by ELISA and which were sent to the Department of Veterinary Pathology, School of Veterinary Medicine, University of Giessen. The following findings appear to be circumstantial evidence for an FIV-infection.
Cats older than 6 years of age and male or male neutered animals predominate at post mortem investigation as also reported from clinical studies. The case history most often includes stomatitis, diarrhea, pyrexia, emaciation, rhinitis or cellulitis.
There are no specific alterations at necropsy in FIV-infected cats. Anemia, emaciation, stomatitis and rhinitis, lymphoid hyperplasia and nephritis are found most often.
Histologically the most consistent and striking fact is hypercellularity of bone marrow which even in old animals is easily diagnosed in the marrow of the femur. The majority of hypercellular bone marrows also display a predominance of immature nuclear forms consistent with myelodysplasia. In the brain there is often a mild to moderate localized encephalitis and/or meningitis. The encephalitis preferentially occurs in the cerebral cortex. The kidneys exhibit in the vast majority of FIV-positive cats interstitial lesions ranging from chronic focal interstitial nephritis to nephrosclerosis. Additionally, there is a usually focal and segmental but sometimes also panglomerular and diffuse glomerulonephritis found in most cases. The lymphatic tissues of spleen and lymph nodes often show proteinaceous and/or fibrillar material with or without clefts covered by endothelial-like cells in follicles. Often follicles are hypocellular or inconspicuous but sometimes there is hyperplasia of follicles and follicles are found in the medulla of lymph nodes. Lymph nodes often show proliferation of activated macrophages in the sinus. Fat storage in Ito cells and hepatocytes, cholangitis and pericholangitis as well as bile duct proliferation is often present in the liver. Bilirubin storage in hepatocytes also occurs frequently. In the intestine moderate to severe cellular infiltration of the mucosa, mild depletion and dilatation of crypts, crypt epithelium degeneration and crypt abscesses are found in some animals.
At the moment there is no way to diagnose FIV infection by post mortem investigation unequivocally. None of the alterations mentioned above is specific for a FIV-associated disease in cats. Combinations of these findings, however, allow for the interpretation that FIV infection may have been the primary cause of disease in the animal.

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OVINE LENTIVIRUS INFECTION: ARTHRITIS IN SHEEP RELATED TO VIRUS ISOLATION BY SYNOVIAL MEMBRANE EXPLANTS

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Maedi/Visna is a disease of mature sheep caused by a lentivirus closely related to caprine arthritis-encephalitis (CAE) virus, even though differences between the genome sequences of the two viruses are reported.
Arthritis is a typical lesion in CAE, but it is not a common finding in lentivirus infection of sheep. Oliver R. E. et al. (1981) and Cutlip R. et al. (1985) described arthritis in sheep with ovine progressive pneumonia, the equivalent of Maedi described in USA.
In the present report we describe carpal joint lesions in sheep positive to Maedi/Visna by the agar-gel immunodiffusion (AGID) test for Maedi/Visna. Our observations were made during an attempt to eradicate Maedi infection in two flocks with low prevalence of reactors.

Material and methods

Three ewes, between 3 to 4 years of age without symptoms of disease, were subjected to necropsy. The first was a Garfagnina ewe (A) from the Experimental Farm of Orecchiella park (Lucca, Italy), the second and third (B) were from a private farm in Siena (Italy).
Samples of lung, mammary gland, choroid plexus and synovial membrane were collected and processed for virological and histopathological examinations to verify the specificity of serological reaction. Explants of collected tissues were cultured in 25 cm^2 flasks containing MEM supplemented by 15% bovine fetal serum. When the cellular layer was confluent, cells were passaged at weekly intervals and sowed in chamber slides (Miles-Illinois). Slides were stained with Giemsa after one week from the passage.
Cell cultures in 75 cm^2 flasks were scraped and centrifugated at 1000 g for 18 minutes. Pellets were fixed in 3% glutaraldehyde, post-fixed in O_sO_4 and embedded in Epon-Araldite. Semi-thin sections were stained with methylene blue and ultra-thin selections with uranyl acetate and lead citrate for TEM observation.
Tissues for light microscopical examination were fixed in buffered formalin (pH 7.3), embedded in paraffin and sectioned at 5 μm. Sections were stained with hematoxylin and eosin, Van Gieson and phosphotungstic acid hematoxylin (PTAH).