Intracytoplasmic inclusion bodies associated with vesicular ulcerative and necrotizing lesions of the digestive mucosa in fallow deer (Dama dama L.)

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Abstract: Intracytoplasmic epithelial inclusion bodies in the digestive mucosa of fallow deer (Dama dama L.) were found to most probably be the result of an unspecific degenerative or post mortal change. There are reasons to believe that this is true also for the inclusion bodies found in reindeer, roe deer and moose.

Key words: epithelial inclusion bodies, alimentary mucosa, reindeer digestive organs

Introduction

Vesicular, ulcerative and necrotizing lesions associated with intracytoplasmic inclusion bodies, have been observed in the epithelium of the upper alimentary tract of various wild cervidae such as reindeer (Rangifer tarandus L.), roe deer (Capreolus capreolus L.), moose (Alces alces L.) and fallow deer (Dama dama L.). (Rehbinder et al 1985; Feinstein et al 1987).

The type of lesions as well as the occurance of inclusion bodies were suggestive of a viral etiology. Electron microscopical studies of the inclusion bodies, however, failed to reveal virus particles. The hypothesis was raised that, irrespective of the etiology, the inclusion bodies could constitute a non-specific degenerative cell response of the digestive epithelium. In the present study we examined, by means of light and electron microscopy, alimentary tissues

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from clinically healthy fallow deer and also from fallow deer suffering of a disease resembling BVD/MD. To evaluate whether autolysis could play a role in the genesis of the inclusion bodies, we allowed alimentary tissues from clinically healthy fallow deer to undergo from mild to advanced autolysis by delaying the start of fixation for 24, 48 and 96 hours.

Results

Diseased animals

Erosions, ulcers, and necrotizing lesions were observed in the oral mucosa (tongue, gingiva and pallatum molle) and rumen. These lesions were deep, irregularly rounded, 5 to 15 mm in diameter with elevated margins and red bases. Some of the animals presented linear erosions



Fig. 1. Oesophagus. Fissured necrotic material covering linear erosions.

covered by a layer of fissured necrotic material in the oesophageal mucosa (Fig. 1).

Histopathology of the mucous membranes of the mouth, oesophagus and fore-stomachs revealed numerous intracytoplasmic basophilic inclusion bodies in cells of the stratum basale and spinosum (Fig. 2). Cells generally presented one inclusion body, but in occasional cells two could be seen. Inclusion bodies were round, 2 to 10 microns in diameter and surrounded by a clear halo. They often compressed the nuclei into a crescent shape and were negative for keratine and prekeratin.

Control groups 0 hs. samples:

All the animals were clinically healthy and no microscopical lesions were observed. Inclusion bodies were not observed in tissues fixed immediately after excision.

24 and 48 hs. samples:

In the oral cavity and oesophagus, of the 24 h samples, a few cells in stratum basale and spinosum exhibited intracytoplasmic basophilic inclusion bodies. In the 48 hs. samples a moderate increase in the number of inclusion bodies was observed.



Fig. 2. Mucous membrane of the mouth. Note swollen cells and numerous inclusion bodies. H&E x 450.



Fig. 3. Diseased fallow deer, oral mucosa. Cells with an inclusion body (IC). Note compressed nucleus (N), intercellular oedema (arrows) and desmosomes (D) x 24600.

96 hs. samples:

The autolytic changes were very advanced, the histological details of the organs being hardly recognizable. Rests of inclusion bodies, however, were still discernable in few cells of the buccal and ruminal epithelium.

Electron microscopical studies

Diseased animals

Inclusion bodies appeared round or oval with a

single membrane and with granular protein-like content. Numerous inclusion bodies were only partly filled or almost empty. Inclusions were located close to the nucleus which was compressed into a crescent shape (Fig. 3). A constant feature was clumping of tonofilaments and inter- and intracelluar edema. Viral particles were not found.



Fig. 4. Control fallow deer, 24 hs. sample. Oral mucosa. Cell with intracytoplasmic inclusion body (IC). Note intercellular oedema (O) and swollen mitochondriae (arrows) x 11700.

Control animals

0 hs. samples:

The mucous membranes of the mouth and oesophagus appeared normal exept for mild inter- and intracellular edema. Inclusion bodies were not observed.

24 hs. samples:

Autolytic changes were moderate. Inter- and intracellular edema was evident. Inclusion bodies very similar to those described for the diseased animals were observed in few cells of the stratum basale of the mucous membranes of the mouth and oesophagus (Fig. 4). Some of them contained a more loose granular proteinlike material.

48 hs. samples:

Autolytic changes were more advanced, swelling of mitocondria and clumping of tonofilaments were clearly observed. Inter- and intracellular edema was pronounced and desmosomes were sharply demarcated. Inclusion bodies were very similar to those described above and more numerous than in the 24 hs. samples (Fig. 5).

96 hs. samples:

Electron microscopical studies were not performed.

Conclusion

The present study on the nature of the inclusion bodies in fallow deer have shown that they did not contain any virus, and most probably were the result of unspecific degenerative- or post mortal changes. (Díaz et al 1989). There are reasons to believe that this is true also for the inclusion bodies found in reindeer, roe deer and moose. (Rehbinder et al 1985, Feinstein et al 1985).



Fig. 5. Control fallow deer, 48 hs. sample. Oral mucosa. Cell with an inclusion body (IC). Note marked autolytic changes, clumping of tonofilaments (T), desmosomes (arrows) and compressed nucleus (N) x 11700.

The original an extended version of the present study is to be published in Acta Vet Scand during 1990.

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