Tyzzer's Disease in Conventionally reared Rabbits in Sweden

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Three conventionally reared female, New Zealand White rabbits (2.37 kg, 1.89 kg and 2.01 kg B.W.) with the history of food poisoning were submitted for routine necropsy at the National Veterinary Institute, Uppsala, Sweden.

The animals, bred for slaughter, were found dead 20 hours after being given oats. The breeder, suspecting food poisoning, together with the carcasses, also submitted a 200 g sample of the feed.

Necropsy revealed major changes in the intestines and liver. The intestines were markedly distended and filled with a muco-haemorrhagic fluid. The intestinal wall was very thin, very haemorrhagic and had large necrotic areas. These lesions were most pronounced in caecum and colon. The mesenteric lymphnodes showed marked oedema and large haemorrhagic zones.

The liver presented numerous white foci of a diameter from barely visible to up to 5 mm, randomly distributed in the whole organ, and some few white firm areas characteristic of hepatic coccidiosis. The histo-pathological investigation of the intestine revealed lesions characterized by severe oedema and an extensive destruction of the mucosa and a marked infiltration of inflammatory cells. Most villi were totally denuded of the epithelial lining. Necrosis also affected the epithelium of the crypts but to a much lesser extent and the epithelial lining was mainly intact. Necrotic changes were very severe in Peyer's patches. The intestinal changes were most prominent in caecum and colon. Giemsa stained sections revealed numerous extracellular bacteria of variable shapes both in necrotic areas and in the intestinal content but also long slender intracellular bacilli typical of *Bacillus piliformis* in the epithelium of and around necrotic areas (Plate 1).

The liver presented numerous necrotic foci with a moderate neutrophilic and monocytic infiltration (Plate 2). Hepatocytes surrounding necrotic foci showed varying degrees of degenerative changes. Giemsa stain revealed numerous intracellular bacilli, morphologically similar to those observed in the intestines (Plate 3 and 4). In addition a limited number of lesions typical of chronic portal hepatitis due to infection with *Eimeria stideae* was present.

Parasitological examination yielded negative results for *Toxoplasma gondii* and *Encephalitozoon cuniculi* but mild intestinal coccidiosis with low numbers of Eimeria spp. Two rabbits had moderate infestation of both *Passalurus ambiguus* and *Trichostrongylus retortaeformis* and the third rabbit harboured a large number of *T. retorteformis*. *Eimeria stideae* was found in liver lesion of all three rabbits. Routine bacteriology for anaerob and aerob bacteria revealed intestinal infection with *Clostridium perfringens* and a generalized infection with *Escherichia coli*.

Analysis of the food sample evidenced a rich growth of *C. perfringens* and moulds. It is apparent that the flora found in the food sample made the food inappropiate for animal consumption. The lesions observed at necropsy were however characteristic of Tyzzer's disease (Allen et al. 1965, Cuttlip et al. 1971) while at the same time the bacteriological investigation revealed an intestinal infection of *C. perfringens* and a generalized infection of *E. coli*, both organisms often associated



Plate 1. Ileum. Necrotic epithelium with intracellular bacilli (Bacillus piliformis) (Arrow). Giemsa stain. \times 1 000.



Plate 2. Liver. Necrotic focus containing cell debris and few inflammatory cells. Note the scarcity of inflammatory cell infiltration around the lesion. Giemsa stain. \times 100.



Plate 3. Liver. Intracellular Bacillus piliformis in necrotic hepatocytes located at the periphery of the focus depicted in plate 2 (Arrows). Giemsa stain. × 1 000.



Plate 4. Liver. Bacillus piliformis in hepatocytes of best preserved areas (Arrows). Degenerative and necrotic features, though present, are not prominent as in necrotic foci. Inflammatory infiltration around affected cells is absent. Giemsa stain. $\times 1000$.

with enteritis and sudden death in rabbits (Nekkels et al. 1976, Patton et al. 1978). It thus seems appropriate to conclude that the present episode had a multifactorial genesis. Most probably a subclinical infection with Bacillus piliformis was already present when the animals were fed the bacteriologically inappropriate food. The unsuitable food produced changes in the intestinal flora and necessary levels of stress that triggered both the multiplication of Bacillus piliformis, producing lesions characteristic of Tyzzer's disease, and also the infection with C. perfringens and E. coli (Matthes 1969). The present episode shows that in outbreaks having a multifactorial genesis it is necessary to identify the different contributing factors since each one of them could be responsible for health problems in the colony, acting individually or adding to other causes of disease. Health monitoring based on complete post-mortem examination is a necessary step not only for diagnostic purposes but also for improving and maintaining the appropriate health condition required for sucessful animal breeding.

References

- Allen, A. M., J. R. Ganaway, T. D. Moore and R. F. Kinard (1965): Tyzzer's disease syndrome in laboratory rabbits. Amer. J. Pathol. 46, 859-882.
- Cuttlip, R. C., W. G. Amtower, G. W. Beall and P. J. Matthews (1971): An epizootic of Tyzzer's disease in rabbits. Lab. Anim. Sci. 21, 356-361.

- Matthes, S. (1969): Die Darmflora gesunder und dysenteriekranker Jungkaninchen. Zbl, Veterinär Med., Reihe B 16, 563-570.
- Nekkels, R. J., M. W. M. A. Mullink and J. C. J. van Vliet (1976): An outbreak of rabbit enteritis: Pathological and microbiological findings and possible therapeutic regime. Lab. Anim. 10, 195-198.
- Patton, N. M., H. T. Holmes, R. J. Riggs and P. R. Cheeke (1978): Enterotoxemia in rabbits. Lab. Anim. Sci. 28, 536-540.

Abstrakti / Kaj Pelkonen

Yhteenveto

Kolme konventionaalisesti kasvatettua New Zealand white — kania toimitettiin tutkittavaksi, kuolinsyyksi ilmoitettiin ruokamyrkytys. Eläinten suolistosta ja maksasta löytyi akuutteja tulehdusmuutoksia. Histopatologisessa löydökset olivat nekroottinen enteriitti ja fokaalinen nekroottinen maksatulehdus, ja suolistosta ja maksasta löytyi pitkiä ja kapeita solunsisäisiä basilleja, joiden muoto oli tyypillinen Bacillus piliformikselle. Tavanomaisessa aerobisten ja anaerobisten bakteerien tutkimuksessa löydettiin suolistosta Clostridium perfringens-tulehdus ja Escherichia coli-yleisinfektio. Tutkitussa rehuerässä kasvoi runsaana C. perfringens ja homeita.

Parasitologinen tutkimuksessa kaikista kaneista löytyi suoli (Eimeria spp.) ja maksakokkidioosi (Eimeria stideae). Kahdesta kainsta tavattiin jonkinverran Passalurus ambiguus ja Trichostrongylus retortaeformis-loisia.

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