Encephalitozoon cuniculi

Host species

- Rabbit (principal host), guinea pig, hamster, rat, mouse
- Dog, some wild and zoo animals

Organotropism

- Brain/Spinal cord
- Kidney
- Liver

Clinical disease

- Usually inapparent
- Occasionally (most often seen in rabbits) neurological disturbances such as torticollis

Morbidity and mortality

- Because of the subclinical nature and multiple routes of transmission, undetected infection can persist in a colony with up to 75% infected animals.
- Morbidity and mortality depend on host strain, generally very low with only single cases of clinical disease in immunocompetent animals.
- Different susceptibility to E. cuniculi in different inbred strains of mice (Niederkorn et al., 1981).

Zoonotic relevance

- Spores are excreted via urine, infection of humans is possible. However, only rare cases of human disease have been reported, and susceptibility of man to E. cuniculi is not well known.

Interference with research

Pathology

- Nervous system: multifocal parenchymal and perivascular cell infiltrations, granulomas with pseudocysts, occasional necrotic foci, occasional meningeal lymphocytic infiltrates.
- Kidneys: multifocal interstitial nephritis, occasional granulomas with pseudocysts.
- Liver: occasional granulomas.
- In immunocompromised hosts possibly aggregates of pseudocysts with minimal inflammatory reaction in various organs.
- 2-3 weeks after intraperitoneal inoculation in mice, animals develop ascites.

Immunology

- Uptake of E. cuniculi by host macrophages (Cox et al. 1979; Weidner 1975).
- Murine peritoneal macrophages can be activated with LPS + IFN-gamma to kill E. cuniculi in vitro (Didier and Shadduck,1994).
- T-cells may act by releasing lymphokines to activate macrophages which can then kill the parasite (Schmidt and Shadduck,1984).
- he lpr (lymphoproliferation) gene does not influence susceptibility to murine encephalitozoonosis (Liu and Shadduck,1988).
- Spontaneous hypergammaglobulinemia in MRL/MPJ mice remains unchanged by E. cuniculi (Liu and Shadduck, 1988).
- During early stages of E. cuniculi infection, murine spleen cells express significantly lower blastogenic responses to T-cell mitogens than uninfected mice (Didier and Shadduck, 1988).
- In neonatal dogs, a depressed T-lymphocyte response to blastogenic stimuli, together with hypergammaglobulinemia (IgG, IgM) was found (Szabo and Shadduck, 1987). In rodents, transient suppression of cell mediated immune responses and no evidence of hypergammaglobulinemia, thus indicating species specificity of immune effects.
- Rabbits infected with E. cuniculi show inconsistent response to neural device biomaterial and are thus inadequate test systems for tissue compatibility testing of such materials (Ansbacher et al.,1988).
- In rabbits naturally infected with E. cuniculi, the immune response to the immunogen Brucella aboffus is altered (elevated IgM, depressed IgG) (Cox and Gallichio, 1978).

Infectiology

- Mice infected with E. cuniculi are more resistant to intracerebral inoculation with Chlamydia trachomatis than non-infected mice (Lepine and Sautter, 1949).

Oncology

- Infected rats which were injected with sarcoma cells had a 50% longer survival than controls (Petri, 1965).
- In infected mice, the growth of several transplantable tumors was reduced and the life-span of the host was prolonged (Arison et al., 1966).

References

Ansbacher, L., M. F Nichols and A. W. Hahn. 1988. The influence of Encephalitozoon cuniculi on neural tissue responses to implanted biomaterials in the rabbit. Lab. Anim. Sci. 38:689-695.

Arison, R. N., J. A. Cassaro and M. P. Pruss. 1966. Studies on a murine ascites producing agent and its effect on tumor development. Cancer Res. 26:1915-1920.

Boot, R., F. Van Knapen, B. C. Kruijt and H. C. Walvoort. 1988. Serological evidence for Encephalitozoon cuniculi infection (nosemiasis) in gnotobiotic guineapigs. Lab. Anim. 22:337-342.

Canning, E. U., J. Lom and I. Dykova. 1986. The microsporidia of birds and mammals. In: Canning EU, Lom J (eds.) The microsporidia of vertebrates. Academic, Orlando, pp. 189-238.

Cox, J. C. 1977. Altered immune responsiveness associated with Encephalitozoon cuniculi infection in rabbits. Infect. Immun. 15:392-395.

Cox, J. C. and H. A. Gallichio. 1978. Serological and histological studies on adult rabbits with recent, naturally-acquired encephalitozoonosis. Res. Vet. Sci. 24:260-261.

Cox, J.C., R. C. Hamilton and H. D. Attwood. 1979. An investigation of the route and progression of Encephalitozoon cuniculi infection in adult rabbits. J. Protozool. 26:260-265.

Didier, E. S. and J. A. Shadduck. 1988. Modulated immune responsiveness associated with experimental Encephalitozoon cuniculi infection in BALB/c mice. Lab. Anim. Sci. 38:680-684.

Didier, E. S. and J. A. Shadduck. 1994. IFN-y and LPS induce murine macrophages to kill Encephalitozoon in vitro. J. Eukaryotic Microbiol. 41:34S.

Gannon, J. 1980. The course of infection of Encephalitozoon cuniculi in immunodeficient and immunocompetent mice. Lab. Anim. 14:189-192.

Greenstein, G., C. K. Drozdowicz F. G. and Garcia. 1991. The incidence of E. cuniculi in a commercial barrier-maintained rabbit breeding colony. Lab. Anim. 25:287-290.

Illanes, O. G., E. Tiffany-Castiglioni, J. F. Edwards and J. A. Shadduck. 1993. Spontaneous encephalitozoonosis in an experimental group of guinea pigs. J. Vet. Diagn. Invest. 5:649-651

Kunstyr, I. and S. Naumann. 1985. Head tilt in rabbits caused by pasteurellosis and encephalitozoonosis. Lab. Anim. 19:208-213.

Lepine, P. and V. Sautter. 1949. Resistance au virus de la Iymphogranulomatose venerienne engendrie chez la souris par Encephalitozoon cuniculi. Ann. Inst. Pasteur Immunol. 77:770-772.

Liu. J. J., E. H. Greeley and J. A. Shadduck. 1988. Murine encephalitozoonosis: the effect of age and mode of transmission on occurrence of infection. Lab. Anim. Sci. 38:675-679

Liu, J. J. and J. A. Shadduck. 1988. Encephalitozoon cuniculi infection in MRL/MPJ-LPR (lymphoproliferation) mice. Lab. Anim. Sci. 38:685-688.

Majeed, S. K. and A. J. Zubaidy. 1982. Histopathological lesions associated with Encephalitozoon cuniculi (nosematosis) infection in a colony of Wistar rats. Lab. Anim. 16:244-247.

Niederkorn, J. Y., J. A. Shadduck and E. C. Schmidt. 1981. Susceptibility of selected inbred stains of mice to Encephalitozoon cuniculi in mice. J. Immunol. 133:2712-2719.

Owen, D. G. and J. Gannon. 1980. Investigation into the transplancental transmission of Encephalitozoon cuniculi in rabbits. Lab. Anim. 14:35-38.

Petri, M. 1965. A cytolytic parasite in the cells of transplantable malignant tumors. Nature 205:302-305.

Shadduck, J. A. and S. P. Pakes. 1971. Encephalitozoonosis (nosematosis) and toxoplasmosis. Am. J. Pathol. 64:657-674.

Schmidt, E. C. and J. A. Shadduck. 1983. Murine encephalitozoonosis. Model for studying the host-parasite relationship of a chronic infection. Infect. Immun. 40:936-942.

Schmidt, E. C. and J. A. Shadduck. 1984. Mechanisms of resistance to the intracellular protozoon Encephalitozoon cuniculi in mice. J. Immunol. 133:2712-2719.

Szabo, J.R. and J. A. Shadduck. 1987. Experimental encephalitozoonosis in neonatal dogs. Vet. Pathol. 24:99-108.

Waller, T., B. Morein and E. Fabiansson. 1978. Humoral immune response to infection with Encephalitozoon cuniculi in rabbits. Lab. Anim. 12:145-148.

Weidner, E. 1975. Interaction between Encephalitozoon cuniculi and macrophages. Parasitophorous vacuole growth and the absence of lysosomal fusion. Z. Parasitenkd. 47:1-9.

Wilson, J. M. 1979. The biology of Encephalitozoon cuniculi. Med. Biol. 57:84-101.

Wilson. J. M. 1986. Can Encephalitozoon cuniculi cross the placenta? Res. Vet. Sci. 40:138.

Author: G. Pohlmeyer / M. Mähler