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 torticolis

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).
- The 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


Author: G. Pohlmeyer / M. Mähler