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ENCEPHALITOOZON CUNICULI IN PET RABBITS

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Encephalitozoon cuniculi is a single-cell, microsporidial organism that is found in a range of animals and is a significant cause of disease in pet rabbits. The spores of *E. cuniculi* are oval in shape and measure approximately 2.5 x 1.5 µm. They are characterised by a strongly Gram-negative capsule. Within the spore, there is a coiled polar filament which can be extruded to inject sporoplasm into a vacuole in a neighbouring cell. Multiplication takes place within the vacuole until mature spores develop. Eventually, the vacuole becomes so distended that the cell ruptures to release the spores. Cell rupture is associated with an inflammatory response and the development of granulomatous lesions.

Incidence of *Encephalitozoon cuniculi*

E. cuniculi has been isolated from a number of species including dogs, foxes, rats, mice and chickens. Although *E. cuniculi* primarily affects rabbits, it can cause disease in other animals. It has been found in dying puppies. In humans, *E. cuniculi* has been linked with diarrhoea, rhinosinusitis, keratoconjunctivitis, nephritis or hepatitis in patients that are immunocompromised by AIDS or by anti-rejection treatment following organ transplants. Three strains of *E. cuniculi* can be identified genetically. Strain I is found predominately in rabbits, strain II in rodents, and strain III in dogs. In the USA, isolates from humans have been shown to be strain III, however, human isolates from Europe have been strain I. Antibodies to *E. cuniculi* are found in many species including dogs, goats and humans.

Infection in rabbits occurs by oral ingestion of food contaminated with infected urine or *in utero* from an infected dam. Infection spreads to other organs including the kidney, central nervous system (CNS) and the heart. Infection of the lens can occur, especially in utero.

Clinical manifestations

There is a range of clinical manifestations of encephalitozoonosis in rabbits. Acute neurological signs such as vestibular disease or seizures may be life-threatening. Chronic myocardial lesions can cause heart failure and death. Vague signs of ill health, such as ataxia, unresponsiveness or weight loss may be due to subclinical neurological or renal disease caused by chronic granulomatous lesions. Other cases are asymptomatic. Vestibular disease a common clinical manifestation of encephalitozoonosis and can range in severity from a minor head tilt to an animal that is unable to right itself and is rolling and hemiparetic. Posterior paresis and ataxia are other neurological manifestations of encephalitozoonosis. Renal disease is a feature of *E. cuniculi* infection and characteristic scarring of the kidneys is a common *post mortem* finding, even in rabbits that have shown no obvious signs of encephalitozoonosis during life. Intraocular disease, such as cataracts, hypopyon or uveitis can be caused by *E. cuniculi*. Cataracts are due to spontaneous rupture of the infected lens at its thinnest point on the anterior surface. Release of the contents of the lens into the anterior chamber causes phacoclastic uveitis.

Diagnosis

Definitive diagnosis of *E. cuniculi* as the cause of disease in the live rabbit is difficult. There are many differential diagnoses and even after death, the diagnosis is often presumptive. Apart the possibility of identifying spores in tissue, urine or lens contents, at present, in the UK, there is no method of detecting antigen, although PCR tests may become available soon. At necropsy, there may be no gross changes apart from minor kidney lesions. On histopathological examination, the presence of spores is diagnostic, but they are not always found. They are usually seen in the kidney and brain. A diagnosis of encephalitozoonosis is often presumptive, especially in chronic cases, because it is based on the presence of characteristic inflammatory lesions rather than the presence of the organism. Serology can be helpful but is far from diagnostic. Laboratory studies have shown that rabbits develop circulating antibody within two to three weeks after infection. Some laboratories report antibody titre levels although antibody titres do not appear to correlate with organism shedding or severity of lesions found at

necropsy. Laboratory studies have not monitored antibody titres over the 10-12 year natural lifespan of a rabbit and it is not clear whether exposed rabbits eventually become seronegative even though they may have residual lesions in their kidneys, CNS or myocardium.

Treatment

Despite extensive literature on the life-cycle and diagnostic tests for *E. cuniculi*, there is little published information on effective treatment protocols for rabbits. Clinical signs are not only related to the presence of the parasite but also to the inflammatory reaction that it evokes. Killing the parasite does not reverse the chronic changes that have already taken place in many organs. Several medications have been used to kill the parasite although it is difficult to prove their efficacy. The subjective opinion of the owner of whether their rabbit 'seems better' may be the only information that is available. Prior to 2001, albendazole was the preparation that was usually recommended.

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Albendazole is an oral anthelmintic that kills the spores of *E. cuniculi* in rabbit kidney cell tissue culture without evidence of cytopathic change. The drug is only parasitostatic. Suggested dose rates for rabbits vary from 10-25 mg/kg/day. Recommendations for the length of treatment vary between sources. Periods of 10-30 days are usually suggested. Infected human AIDS patients may receive lifelong treatment. In UK, fenbandazole is used more than albendazole since the publication of a small study that described the eradication of *E. cuniculi* organisms in rabbits treated with fenbandazole for 28 days at a dose rate of 20mg/kg/day. There are anecdotal reports of bone marrow suppression in a small number of rabbits treated with benzidamazoles.

Other products that have been used to treat *E. cuniculi* in rabbits include oxytetracycline and pyrimethamine. Lufenuron has also been investigated because the cell wall of the parasite contains chitin. However, *in vivo* assays indicate no therapeutic effect.

Corticosteroids are indicated for acute neurological signs associated with *E. cuniculi* to suppress the inflammatory response associated with cell rupture. However, high doses of long term corticosteroid therapy is contraindicated because of the immunosuppressive effect. A single dose of a short acting corticosteroid, e.g., 0.5-1mg/kg dexamethasone, can be given to rabbits that have recently developed acute neurological signs. If further treatment is required, anti-inflammatory, rather than immunosuppressive doses of 0.2mg/kg dexamethasone is suggested. Additional symptomatic treatment may be directed at the clinical signs e.g. midazolam (0.5-1mg/kg) can be used to control fits or prochlorperazine (500µg/kg oral tds) may help control vestibular signs.

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