

NEUROLOGICAL DISEASES OF RABBITS AND RODENTS

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Abstract

Clinical signs of neurological disease, such as head tilt, hind limb paresis or paralysis, seizures, and muscle weakness, are commonly encountered in pet rabbits, and in the authors' experience, less often in rodent species. Moreover, localisation of neurological lesions and establishment of a definitive diagnosis can be challenging for any of the exotic small mammal species. In many rabbit and rodent cases, distinguishing neurological disease from musculoskeletal disease is difficult. The parasitic disease encephalitozoonosis is commonly diagnosed in pet rabbits; in both rabbits and rodents, bacterial infections are also a common underlying cause of neurological disease. Other causes of neurological diseases that adversely affect pet rabbits and rodents include toxins, trauma, metabolic and degenerative disorders, viral infections, neoplasia, and hereditary abnormalities. Copyright 2014 Elsevier Inc. All rights reserved.

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RABBITS

Neurological signs such as head tilt (Fig. 1), circling, ataxia, paresis or paralysis, nystagmus, and seizures are frequently diagnosed in rabbit patients.¹⁻³ Differential disease diagnoses, in rabbits that present with neurological problems, are broadly based on the clinical signs listed in Table 1.

Parasitic Infections

Encephalitozoonosis. *Encephalitozoon cuniculi* is an obligate intracellular protozoal parasite, belonging to the phylum Microspora. This parasite appears to be widespread in the domestic rabbit population (e.g., seroprevalence in clinically healthy pet rabbits has been reported as 52% in the United Kingdom,⁴ 49% in the United States,⁵ 18% in Germany,⁶ and between 27.9% and 75.2% in Japan).⁷ *E. cuniculi* infections occur primarily in rabbits, but have also been diagnosed in many other mammals including sheep, goats, pigs, dogs, cats, foxes, rodents, monkeys, and birds (e.g., gyrfalcons).⁸ It is a zoonosis and can cause a range of clinical signs in

humans from self-limiting diarrhoea to severe life-threatening infection. Immunocompromised humans are at greatest risk of developing clinical signs. Infections in humans have been shown to be caused by the same strain that infects rabbits,⁹ and in some cases previous contact with rabbits has been reported.¹⁰ Transfer of human strains to rabbits is possible.¹¹ Infective spores are shed in the urine and transmission usually occurs by ingestion of contaminated food and water, although transplacental infection and infection via inhalation have also been reported. The *E. cuniculi* spores can remain viable in the environment in dry conditions at 22°C for approximately 4 weeks. Spores of *E. cuniculi* are easily destroyed by most routine disinfectants (including 0.1% solution of bleach and 70% ethanol), as well as by boiling and autoclaving.

Once ingested the parasite infects the intestinal epithelium where it replicates, and infective spores are carried in circulating macrophages to the liver, kidney, central nervous system (CNS), lungs, and heart. Cell rupture eventually occurs, releasing infective spores and causing inflammation and granuloma formation in these target organs. In the

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FIGURE 1. Neurological signs, such as torticollis, are common clinical conditions affecting rabbits.

CNS, the *E. cuniculi* organisms cause focal nonsuppurative granulomatous meningoencephalitis. Infected rabbits may develop a subclinical carrier status. Overt clinical disease signs associated with encephalitozoonosis in rabbits are listed as follows:

- Head tilt
- Torticollis
- Hind limb paresis
- Paralysis
- Retarded growth
- Collapse
- Tremors
- Convulsions
- Urinary incontinence
- Renal failure
- Cataracts and phacoclastic uveitis
- Death

Diagnosis of *E. cuniculi* in the live rabbit is difficult as the organism is difficult to isolate. Indirect enzyme-linked immunosorbent assay tests are used to measure rabbit serum antibody levels (IgM and IgG) to the parasite. A positive serological result indicates exposure to the parasite but is not conclusive that it is the cause of the clinical signs presented. A presumptive diagnosis

TABLE 1. Differential diagnoses for clinical signs of neurological disease in rabbits

Clinical Sign	Differential Diagnoses
Head tilt/ torticollis	Central vestibular disease (cerebellum and medulla oblongata): Bacterial infection Encephalitozoonosis Toxoplasmosis Herpes virus (herpes simplex virus 1) encephalitis Cerebrovascular accident Cerebral larva migrans Trauma Toxins (e.g. lead) Neoplasia Rabies Peripheral vestibular disease (vestibular portion of CN VIII and inner ear): Bacterial otitis media/interna Toxins (e.g., aminoglycosides) Neoplasia
Paresis/ paralysis	Vertebral fracture/luxation Spondylosis/spondylitis Spinal abscess Spinal neoplasia Osteoarthritis Intervertebral disc protrusion Encephalitozoonosis Toxoplasmosis Splay leg Hypovitaminosis A Neoplasia
Seizures	Bacterial encephalitis Encephalitozoonosis Herpes virus encephalitis Lead toxicity Fipronil toxicity Pyrethrin/permethrin toxicity Heat stroke Pregnancy toxemia Hypocalcaemia Hypoxia Azotaemia Electrolyte imbalance Terminal systemic disease (e.g., septicemia, toxemia and rabbit haemorrhagic disease) Neoplasia Brain abscess Cerebral larva migrans Hypovitaminosis A Hereditary ataxia Idiopathic epilepsy (blue-eyed white rabbits) Rabies

CN, cranial nerve.

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