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NEUROLOGICAL DISEASES IN RABBITS

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INTRODUCTION

Neurological complaints are relatively common in rabbits, accounting for approximately 11% of clinical presentations. Veterinary professionals must be aware that these cases often require urgent attention, especially when impressive symptoms for owners such as head tilt are present. Neurological examinations in rabbits are challenging due to their prey species behavior and the subtlety or severity of clinical signs (1). Performing a thorough neurological examination in rabbits is not always straightforward. Rabbits may be stressed by handling, and their clinical signs can be both subtle and alarming to owners. Despite these challenges, a systematic neurological assessment remains essential for diagnosis and management.

Head Tilt and Vestibular Syndrome

Head tilt is the most frequent neurological complaint in rabbits. This sign is considered a relative emergency and is particularly distressing for owners. Vestibular syndrome in rabbits can have either central or peripheral origins but distinguishing between these is unfortunately not possible clinically in rabbits and diagnostics are required (2). The differential diagnosis for vestibular syndrome in rabbits includes infectious, vascular, traumatic, neoplastic, and toxic causes. Infectious causes are common and include Encephalitozoon cuniculi, otitis interna, cerebral abscesses, meningoencephalitis, toxoplasmosis, and herpes simplex virus. Vascular causes include ischemic lesions of the central nervous system. Traumatic injuries and neoplastic conditions must also be considered. Toxic causes such as lead or fipronil poisoning can also present with vestibular signs.

Encephalitozoon cuniculi Infection

Encephalitozoon cuniculi is an obligate intracellular microsporidian parasite with a tropism for the brain, kidneys, and lens. There is ongoing debate regarding its classification as a fungus. Transmission occurs primarily through urine and in utero exposure. The parasite can be excreted in urine for up to three months after infection and can persist in the environment for up to six weeks. The seroprevalence rates are high, ranging from 20–80% in meat rabbits and 52–75% in domestic rabbits. Encephalitozoon cuniculi is a zoonotic pathogen, although clinical disease in humans is rare.

Clinical signs of E. cuniculi infection include neurological symptoms such as head tilt, vertigo, nystagmus, and ataxia. Urinary signs may include polyuria, polydipsia, and urine scald. Ocular signs, such as phacoclastic uveitis, may also be present.

Diagnosis of E. cuniculi infection is based on a combination of serology (IgG and IgM titers). PCR testing of urine or aqueous humor, could be performed but the shading of spores is intermittent. Protein electrophoresis or C-reactive protein measurement have been described in the literature but these diagnostics a not specific and should be performed in addition to other diagnostics. Postmortem histological examination may reveal meningoencephalitis with perivascular infiltration and disseminated granulomatous encephalitis. Renal parameters such as urea and creatinine may also be evaluated as kidney lesions could occur and induce.

Treatment of E. cuniculi infection typically involves oral administration of fenbendazole at 20 mg/kg once daily for 28 days. If the treatment is not working, albendazole at 7.5–20 mg/kg once daily for 14 days could be used, but liver enzymes should be checked as albendazole could be hepatotoxic. Corticosteroids, such as dexamethasone, have not demonstrated efficacy (3). Supportive care may include environmental adaptation, and physiotherapy such as massage or osteopathy. The prognosis is guarded, as neurological deficits are not always reversible. The degree of head tilt and duration of immobilization are negative prognostic indicators.

Otitis Interna and Media

Bacterial pathogens such as Pasteurella multocida, Staphylococcus aureus, Pseudomonas aeruginosa, and Bordetella bronchiseptica are common causes of otitis interna and media in rabbits. Rhinitis can progress to otitis media and then to otitis interna, or otitis externa can extend inward.
Otitis interna typically presents as a peripheral vestibular syndrome, with vertigo, facial paralysis, and nystagmus.
Diagnosis of otitis interna and media is made by CT sca. These imaging modalities help assess the extent of infection and bony involvement.
Medical treatment includes antibiotics, ideally selected based on culture and sensitivity results. Azithromycin can be administered at 30 mg/kg orally every 12 hours. Medical management alone often yields poor results, and surgical intervention is necessary. Surgical options include lateral ear canal resection and bullectomy (4). In some minor conditions a myringotomy with eh flush of the tympanic bulla could be efficient. Myringotomy is indicated for otitis media or interna without tympanic bulla lysis.

Paresis and Paralysis
Vertebral fractures or luxations are usually traumatic in origin, often resulting from falls. The lumbar vertebrae L4–L7 are most commonly affected. Vertebral fractures account for 23.2% of all non-neoplastic fractures in rabbits and 47.7% of fractures in rabbits under three years of age. Clinical signs include paresis or paralysis, fecal and urinary incontinence, and loss of the panniculus reflex.
Diagnosis of vertebral fractures or luxations is achieved through radiography, CT scan, cone beam CT, micro CT.
Surgical treatment is complex, with no established gold standard, and requires advanced surgical skills and intensive postoperative nursing but could restore entirely the functional mobility. Medical management is possible if deep pain perception is preserved. Conservative treatment may extend for more than three months and requires intensive supportive care, including fluid therapy, analgesia, nutritional support, and nursing care such as daily bathing, cleaning, and manual bladder expression. Rehabilitation and the use of wheelchairs may also be necessary.
Other causes of paresis and paralysis in rabbits include spondylosis, spondylitis, spinal abscesses, synovial cysts, spinal neoplasia (such as lymphoma or osteosarcoma), osteoarthritis, vertebral disk herniation, degenerative myelopathy, infectious diseases (E. cuniculi, toxoplasmosis, sarcocystis), heavy metal intoxication, hypovitaminosis A, and Floppy Rabbit Syndrome.

Convulsive Disorders
Convulsive disorders are less common in rabbits than in gerbils or ferrets. Seizures can be classified by etiology as primary (idiopathic) epilepsy, symptomatic (secondary or acquired) epilepsy, or reactive (metabolic or toxic) convulsions. Types of seizures include generalized (tonic-clonic, tonic, clonic, myoclonic) and focal (simple or complex) seizures. Focal seizures may progress to secondary generalized seizures.
Infectious causes of seizures in rabbits include bacterial encephalitis, viral encephalitis (such as herpesvirus simplex), cerebral abscesses (often due to Pasteurella multocida), encephalitozoonosis, cerebral larva migrans, sarcocystis, and rabies.
Non-infectious causes include intoxication (lead, fipronil, permethrin), gestational toxemia, metabolic disturbances (hypoxemia, azotemia, hypocalcemia, electrolyte imbalances), environmental or nutritional factors (heat stroke, hypovitaminosis A), terminal systemic disease (such as septicemia or rabbit hemorrhagic disease virus), and idiopathic epilepsy, which is particularly noted in Blue-Eyed White rabbits.
Lead intoxication can result from ingestion of paint, metallic objects, or linoleum. Clinical signs include chronic weight loss, weakness, anorexia, ileus, anemia, posterior ataxia, and convulsions. Diagnosis is based on radiography (to try to find metallic objects), blood lead levels greater than 1.1 µmol/L, and characteristic hematological findings. Treatment involves administration of Ca-EDTA at 27.5 mg/kg subcutaneously every 12 hours for five days, with a five-day break before repeating the course.
Symptomatic treatment of seizures includes administration of midazolam at 0.1–1 mg/kg intravenously or intramuscularly, levetiracetam at 10–20 mg/kg orally three times daily, and phenobarbital at 1–2 mg/kg orally twice daily. Etiological treatment targets the underlying cause and may involve antibiotics, mannitol at 0.5–1 g/kg intravenously over 20 minutes, activated charcoal, Ca-EDTA, and other specific therapies.

CONCLUSION

Neurological disorders in rabbits are multifactorial and require a systematic diagnostic approach. Infectious causes, particularly Encephalitozoon cuniculi and bacterial otitis, are prevalent and should be prioritized in the differential diagnosis. Early intervention and aggressive supportive care can improve outcomes, but the prognosis could be guarded, especially in cases with severe or chronic neurological deficits. Owner education and intensive nursing care are essential components of management. The zoonotic potential of E. cuniculi should be discussed with owners, particularly those who are immunocompromised.

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