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CASE REPORT
Late onset cerebellar degeneration in a middle-aged cat

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Cerebellar degeneration (abiotrophy) (CD) is a spontaneous and accelerated degeneration of one or several mature cerebellar neuronal cell populations and has been described in many domestic animals, especially in dogs, with numerous breed-related cases. In cats, CD is mentioned as a rare sporadic entity. Late onset CDs are exceptionally uncommon and only two cases are reported in young adults, both aged 18 months. This report describes clinical and pathological findings of a late onset feline CD in a 9-year-old male Persian cat. The cat was presented with a history of progressive ataxia lasting 2 years. Neurological examination revealed severe neurological deficits such as generalised and severe ataxia, hypermetria in all four limbs, and bilateral absence of menace response. The lesion was diffusely localised in cerebellum. On gross pathology, the cerebellum appeared of normal size and shape and kidneys were characterised by mild hyperaemia. Histologically, lesions were limited to the cerebellum and kidneys. In the cerebellum, all cerebellar folia of both hemispheres and the vermis were affected. Changes were characterised by severe and diffuse loss of Purkinje cells, loss of cellularity in the granular layer, mild astrogliosis associated with moderate hypertrophy of Bergmann's glia. Immunohistochemistry for feline parvovirus antigen revealed a negative result. Renal lesions consisted of chronic fibrosis associated with chronic interstitial nephritis. CD is a rare disease and occurs commonly in puppies or young animals, who are clinically normal at birth and usually develop neurological signs within a few weeks or months after birth. This report represents the first case of CD in a middle-aged cat.

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Cerebellar cortical degeneration (abiotrophy) (CD) is commonly considered a neurological condition characterised by premature degeneration and death of one or several cerebellar neuronal cell populations (Jubb and Huxtable 1993, Summers et al 1995). The term CD, used in its original definition, refers to a neurological condition that occurs in cerebellar neurons that have reached complete maturity and are functionally normal. CD has to be differentiated from cerebellar hypoplasia, a term indicating a failure of complete neuronal development (Jubb and Huxtable 1993, Summers et al 1995).

In human neuropathology, the term cerebellar 'abiotrophy' is rarely used; some degenerative conditions, such as Parkinson's and Alzheimer's disease (Summers et al 1995), are considered examples of abiotrophic diseases. A particular form of cerebellar degeneration, called subacute cerebellar degeneration (SCD) seems to be associated with two different conditions: paraneoplastic cerebellar degeneration, which sometimes precedes the diagnosis of neoplasia, and alcoholic or nutritional cerebellar degeneration, caused, eg, by vitamin B1 deficiency (Gray et al 2004).

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CD has been described in many domestic animals, including dogs, cattle, sheep, horses, and pigs. In several cases of CD, a hereditary mechanism has been assumed and numerous canine breed-related abiotrophies are described (De Lahunta 1990, Jubb and Huxtable 1993). CD commonly occurs in puppies or young animals, who are clinically normal at birth and usually develop neurological signs within the first few weeks or months after birth; cases of late onset or geriatric CD have been described in some canine breeds comprising Brittany Spaniels and American Staffordshire Terriers (LeCouter et al 1988, Jubb and Huxtable 1993, Higgins et al 1998, Olby et al 2004).

In cats, CD is considered a rare neurological condition. In Japan, several cases of early onset cerebellar cortical degeneration has been observed in different kitten populations; a feline parvovirus infection was ruled out in these kittens. For some of them, a hereditary condition has been suggested (Inada et al 1996, Aye et al 1998, Barone et al 2002). Only two cases, involving young adults have been reported to date (Shamir et al 1999, Barone et al 2002). Other cases of feline CD have been referred as anecdotal (De Lahunta 1980; Summers et al 1995) without complete breed, age, clinical and pathological descriptions. In the present report, the clinical and pathological findings in a feline late onset cerebellar abiotrophy are described, and to the authors' knowledge, this is the first CD report in a middle-aged cat.

A 9-year-old male Persian indoor cat was presented with a history of ataxia lasting for 2 years. The cat was adopted by the owner at 5 years of age and was found to be clinically normal at that time. At 7 years of age, the cat showed a depressed mental status, associated with an episode of vomiting and diarrhoea. Two weeks later a mild ataxia in the hind limbs was observed, but no diagnostic investigations were performed at this stage to investigate these neurological signs. Vomiting and diarrhoea were interpreted as signs of small intestinal bacterial overgrowth and a symptomatic treatment was instituted. The cat's overall performance temporally improved but neurological signs persisted. The ataxia progressively involved the fore limbs and the neurological signs slowly progressed over 2 years, when the cat was presented to his referring veterinarian for persisting episodes of vomiting. Cell blood count and biochemical parameters (aspartate aminotransferase, alanine aminotransferase, creatine kinase (CK), creatinine, urea, glucose, total bilirubin, triglycerides, total cholesterol, γ-glutamyltransferase, total proteins, albumin, albumin/globulin, calcium, phosphorus, sodium, potassium, chloride) were within normal limits except for an increase of urea (79.9 mg/dl, reference value: 20–65 mg/dl), creatinine (2.58 mg/dl, reference value: 0.8–1.8 mg/dl), total cholesterol (258 mg/dl, reference value: 95–130 mg/dl) and CK (518 UI/l, reference value: 18–230 UI/l), suggesting a possible renal disease. Serologically, titres for feline infectious peritonitis virus, feline leukaemia virus and feline immunodeficiency virus as well as *Toxoplasma gondii* were not detected. No history of past trauma or toxic ingestion was reported. No treatments were instituted and then, the cat was referred to our Institution for neurological evaluation.

Physical examination revealed a poorly groomed coat and moderate dehydration. At neurological examination, generalised and severe ataxia, intention tremor of the head, severe hypermetria in all four limbs, wide based stance, and loss of balance with frequent events of falling and tumbling were recorded. In addition, the cat exhibited a bilateral absence of the menace response with normal pupillary light reflex. Clinically, the lesion was diffusely localised in the cerebellum. Differential diagnoses included degenerative diseases, including late onset lysosomal storage diseases, such as gangliosidosis, and feline cerebellar abiotrophy. Neuroaxonal dystrophy was included in degenerative differential diagnoses even if, with the late onset of neurological signs, it was considered unlikely. Moreover, a metabolic condition or a chronic toxicity was not included, despite the possible underlying renal dysfunction previously detected by blood tests, as cerebellar signs as unique neurological manifestations of a systemic disorder are not well defined in veterinary medicine. Based on the slow progression and protracted clinical signs, an inflammatory and neoplastic process was considered unlikely. Due to the poor prognosis, the owner requested euthanasia and a complete necropsy was performed.

Gross lesions were observed only in the kidneys, which presented diffuse hyperaemia associated to mild and diffuse atrophy. On gross examination, the cerebellum appeared to be of expected size and shape. Tissues of various organs, such as brain, spinal cord, digestive tract, trachea, lungs, liver, spleen, bladder, kidneys, adrenals, pancreas, popliteal lymph nodes, mesenteric lymph nodes, bronchial lymph nodes,