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Case Report: Lafora bodies in a black-tailed prairie dog *(Cynomys ludovicianus)*

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Summary

Lafora bodies (LBs) are polyglucosan inclusions predominantly found in the central nervous system (CNS), although they are also reported elsewhere. They typically accumulate in neurons, their processes and the cytoplasm of glial cells. Lafora disease (LD) is reported in humans, companion and wild animals alike and results from an accumulation of insoluble LBs. In dogs and humans, LD is inherited as an autosomal recessive trait. It shows a progressive course and leads to myoclonic seizures and a wide range of other neurological symptoms. LBs are also found in asymptomatic cases and are considered as age-related changes. We present a case of a 5-year-old, captive-held black-tailed prairie dog *(Cynomys ludovicianus)* that was found dead without reported clinical signs. LBs were found in the cerebellum, cerebrum and extracranially in the liver and heart. Purkinje cells were predominantly affected, with approximately 80 % of these cells involved. The LBs were positive in PAS stain and were PAS-diastase resistant. An ultrastructural investigation revealed two types of LBs in the Purkinje cells. This is the first report of the spontaneous occurrence of LBs in a black-tailed prairie dog.

Abbreviations: CNS = central nervous system; HE = haematoxylin and eosin; LB/LBs = Lafora body/bodies; LD = Lafora disease; PAS = Periodic Acid-Schiff; PAS-D = Periodic Acid-Schiff diastase; TEM = transmission electron microscopy

Zusammenfassung

Fallbericht: Lafora Körperchen bei einem Schwarzschwanz-Präriehund *(Cynomys ludovicianus)*

Lafora-Körperchen stellen Polyglucosaneinschlüsse dar, die vor allem im Zentralnervensystem (ZNS) beobachtet werden, aber auch außerhalb des ZNS vorkommen können. Typischerweise treten sie in Neuronen und deren Fortsätzen, im Zytoplasma von Gliazellen und extrazellulär auf. Die Lafora-Krankheit wird sowohl bei Menschen als auch bei Haus- und Wildtieren beobachtet und ist auf eine Akkumulation von unlöslichen Einschlüssen zurückzuführen. Bei Hunden und Menschen wird die Lafora-Krankheit autosomal-rezessiv vererbt. Sie zeigt einen progressiven Verlauf und führt zu myoklonischen Anfällen und zahlreichen anderen neurologischen Symptomen. Dennoch treten Lafora-Körperchen auch in asymptomatischen Fällen auf und werden als altersbedingte Veränderungen angesehen. Dieser Bericht beschreibt den Fall eines 5 Jahre alten Schwarzschwanz-Präriehundes *(Cynomys ludovicianus)*, der in einem Zoo lebte und plötzlich tot aufgefunden wurde. Lafora-Körperchen wurden im Kleinhirn, in den zerebralen Perikarya, in den Gliazellen der Großhirnrinde, in der Granularzellschicht des Hippocampus und extrakraniell in der Leber und im Herzen gefunden. Überwiegend waren Purkinje-Zellen (ca. 80 % aller Zellen) betroffen. Die Lafora-Körperchen stellten sich PAS-positiv und PAS-Diastase-resistent dar. Die ultrastrukturelle Untersuchung ergab zwei verschiedene Arten von Lafora-Körperchen in den Purkinje-Zellen. Dies ist die erste Beschreibung des spontanen Auftretens von Lafora-Körperchen bei einem Schwarzschwanz-Präriehund.

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Introduction

Lafora disease (LD) is characterized by myoclonic seizures and an accumulation of intracytoplasmic material known as Lafora bodies (LBs) (Chambers et al. 2018; Demeny et al. 2020). LBs are composed of polyglucosan filaments and are strongly positive in periodic acid-Schiff (PAS) staining while resistant to PAS-diastase stain (PAS-D) (Simmons 1994; Turnbull et al. 2016). They have either dense central cores and pale, radiating, peripheral rings or an entirely homogenous appearance (Holland et al. 1970). LBs were first described in 1911 by Gonzalo Rodríguez Lafora, the last of Cajal's great Spanish disciples and exponent of the Cajal School or the Spanish Neurological School (Nanduri et al. 2008).

The neurological signs probably result from the accumulation of insoluble glycogen molecules (polyglucosan bodies) in the brain (Swain et al. 2017). LD in dogs can manifest as myoclonus, generalized or focal seizures, blindness, deafness, faecal and urinary incontinence and behavioural changes (Swain et al. 2017; Demeny et al. 2020). Although LBs are also observed extracranially, non-neurological symptoms are rare (Minassian 2001). The disease is progressive and eventually leads to death (Chambers et al. 2018). In asymptomatic cases, LBs may represent age-related changes (Kamiya & Suzuki 1989; Simmons 1994). In cattle, age-related LBs have been found intracellularly in the liver and the CNS, predominantly in the thalamus and rostral midbrain (Simmons 1994). The hepatocytes have a “ground glass” appearance in asymptomatic age-related cases (Simmons 1994).

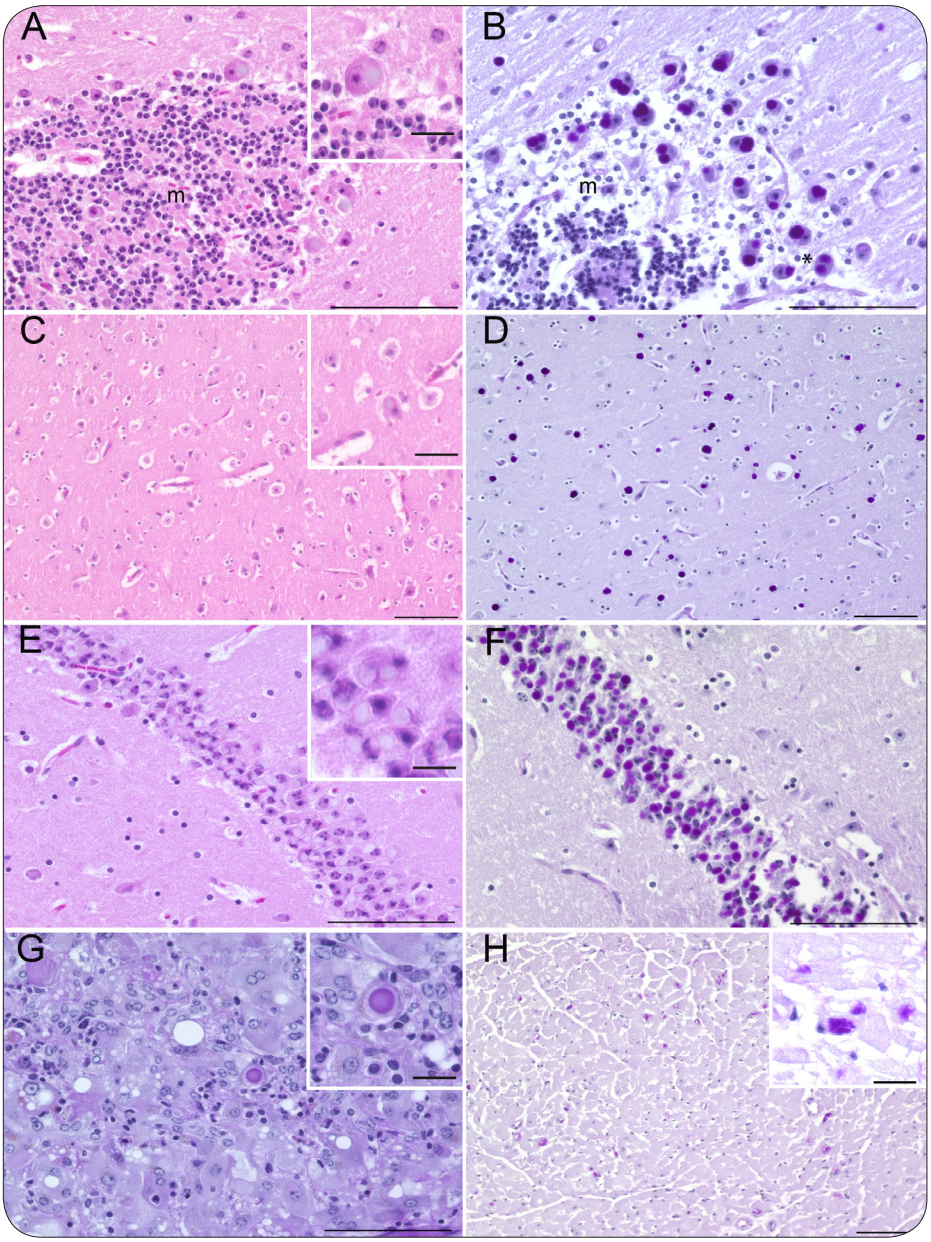
LD is an autosomal recessive genetic disorder in humans, dogs, grey-headed flying foxes, fennec foxes, Alexandrine parakeets, moose, rats, cats and cattle (Holland et al. 1970; Suzuki et al. 1979; Jayaraj 1980; Simmons 1994; Hall et al. 1998; Gabor & Srivastava 2010; Honnold et al. 2010; Stent et al. 2015; Ravi et al. 2022). LD in dogs has an autosomal trait and is frequently observed in predisposed breeds such as miniature wirehaired dachshunds, basset hounds, beagles, Chihuahua, French Bulldogs and Griffon Bruxellois (Jian et al. 1990; Gredal et al. 2003; Swain et al. 2017; von Klopmann et al. 2021). There have been no reported cases of spontaneous LD in rodents. We now provide the first report of LBs in a black-tailed prairie dog *(Cynomys ludovicianus)*.

Case description

A 5-year-old female black-tailed prairie dog *(Cynomys ludovicianus)* kept in a zoo was presented for necropsy. The female had given birth to five cubs four weeks before her death. At the time of death, 14 adult animals and 7 cubs were kept in an outdoor enclosure of 136 sqm. The enclosure was divided into two parts connected by a tunnel and contained several shelters of altogether 6 sqm. The female was seen mostly in the den with her cubs, only occasionally coming outside to feed, drink, defecate and urinate. No clinical signs were reported, although three other mature animals were found dead without clinical symptoms. The prairie dog had a good condition score. The necropsy was performed in the Department of Pathology, University of Veterinary Medicine Hannover. Macroscopically, multifocal white spots measuring 0.1 x 0.1 x 0.1 cm were seen in the liver and kidney. The following organs were fixed and examined histologically: tongue, brain, trachea, oesophagus, heart, lung, mesenteric and tracheobronchial lymph nodes, spleen, stomach, uterus, intestine, pancreas, pituitary gland, diaphragm, liver, kidney, urinary bladder, adrenal glands, thyroid glands, skeletal muscles, *nervus ischiadicus* and *plexus brachialis*. Tissue samples were fixed in 10 % neutral buffered formalin for 24 h, embedded in paraffin wax, sectioned and routinely stained with haematoxylin and eosin (HE), Periodic Acid-Schiff (PAS) and Periodic Acid-Schiff diastase (PAS-D).

Microscopically, intracytoplasmic, up to 15 μm in diameter, roundish material was found in the cerebellum, the cerebral perikarya, the glial cells of the cerebral cortex and in the hippocampal granular layer. It was characterized by basophilic cores surrounded by amphophilic outer layers, consistent with LBs (Fig. 1). The most affected cell population was Purkinje cells and the neuropil, with approximately 80 % of the Purkinje cells involved. Some of the LBs in the Purkinje cells displaced cell nuclei and we observed duplication or fusion of the LBs. LBs were also seen extracranially in the liver and the heart. The hepatocytes showed single LBs with characteristic dense cores and peripheral paler margins (Fig. 1G). Multifocal Lafora bodies in the muscle fibres of the left myocardium were homogenously basophilic and lacked a central core (Fig. 1H). All LBs in the brain, liver and heart were strongly PAS-positive and PAS-D-resistant. We saw focal mild fibrosis in the left ventricular myocardium. The hepatocytes were diffuse and mildly vacuolated and some of them had kidney-shaped inclusions and a “ground-glass” appearance. The liver had mild, periportal and parenchymal lymphoplasmahistiocytic infiltrations and showed a mildly increased number of bile ducts. The hepatocytes showed slight anisokaryosis and an increased ratio of nucleus:cytoplasm. Some multinucleated hepatocytes were seen and a chronic, mild, cortical multifocal, lymphoplasmacytic infiltration was present in the kidney. Single tubular epithelial cells were necrotic and the kidney tubuli were mildly to moderately dilated.

We used transmission electron microscopy (TEM) to characterize the LBs in the brain. Tissues were fixed in glutaraldehyde, followed by 1 % osmium tetroxide fixation, dehydration in graded alcohols and embedding in epoxy resin. Ultrathin sections were cut and stained with uranyl acetate and lead citrate (Allnoch



et al. 2021). We found LBs in the perikarya of Purkinje cells, where they pushed the nuclei to an eccentric position and partially displaced the subcellular compartments (Fig. 2). The LBs were not bound to cell membranes. The examination revealed two distinct morphological appearances of the LBs. The first type of LB consisted of branching filaments without a core (Fig. 2 A), while the second type had a prominent, electron-dense, homogenous central core and a filamentous, peripheral area (Fig. 2 B). The LBs measured approximately 800 nm in diameter.

Discussion

Black-tailed prairie dogs have a lifespan of about 11 years in captivity (Gorbunova et al. 2008). The cause of the prairie dog´s death in the present case is unknown. Myocardial fibrosis and the degenerative and inflammatory changes in the liver and kidneys represent chronic, unspecific alterations. The intracellular material in the brain, liver and heart is consistent with Lafora bodies. The mature age of the animal

Fig. 1: Histological examination reveals numerous Lafora bodies in the brain, liver and heart. (A) In the *cerebellum*, numerous Lafora bodies are present predominantly within the Purkinje cells, m = molecular layer, HE staining, 40x, scale bar = 100 µm; Insert: scale bar = 20 µm. (B) Single and duplicated (*) PAS-diastase negative Lafora bodies are present in the Purkinje cells. Lafora bodies partially displace nuclei of Purkinje cells, m = molecular layer; PAS-diastase staining, 40x, scale bar = 100 µm. (C) Lafora bodies in the cerebral cortex are visible in the perikaryon, HE staining, 20x, scale bar = 100 µm; insert: scale bar = 20 µm. (D) Multiple Lafora bodies in the cerebral cortex are PAS-diastase negative, 20x, scale bar = 100 µm. (E) Numerous Lafora inclusion bodies in the granular layer of the hippocampus, HE staining, 100x, scale bar = 100 µm; insert: scale bar = 20 µm; (F) Lafora bodies present in high numbers in the granular layer of the hippocampus, PAS-diastase staining, 100x, scale bar = 100 µm; insert: scale bar = 20 µm; (G) Lafora body in the liver presenting a characteristic dense core and peripheral pale margin, PAS-staining, 20x, scale bar = 100 µm; insert: scale bar = 20 µm; (H) Conglomerated Lafora bodies lacking central core in myofibres of the heart, 20x, scale bar = 100 µm, PAS-staining; insert: scale bar = 20 µm / Die histologische Untersuchung ergibt zahlreiche Einschlüsse, die unter anderem im Gehirn lokalisiert sind. (A) Im Kleinhirn befinden sich zahlreiche Lafora-Körperchen in den Purkinjezellen und Gliazellen der Molekularschicht (m), HE-Färbung, 40x, Balken = 100 µm; Insert: Balken = 20 µm. (B) PAS-Diastase-resistente Einschlüsse (einzeln und doppelt = *) in den Purkinje-Zellen und in der Molekularschicht; die Lafora-Körperchen verdrängen teilweise die Kerne der Purkinje-Zellen, PAS-Diastase-Färbung, 40x, Balken = 100 µm. (C) Lafora-Körperchen in der Großhirnrinde, HE-Färbung, 20x, Balken = 100 µm; Insert: Balken = 20 µm; (D) Lafora-Körperchen in der Großhirnrinde, PAS-Diastase-Färbung, 20x, Balken = 100 µm; (E) Lafora-Körperchen in der granularen Schicht des Hippocampus, HE-Färbung, 100x, Balken = 100 µm; Insert: Balken = 20 µm; (F) Lafora-Körperchen in der granulären Schicht des Hippocampus, PAS-Diastase-Färbung, 100x, Balken = 100 µm; Insert: Balken = 20 µm; (G) Lafora-Körperchen in der Leber mit charakteristischem dichten Kern und peripherem blassen Rand, PAS-Färbung, 20x, Balken = 100 µm; Insert: Balken = 20 µm; (H) Konglomerierte Lafora-Körperchen ohne zentralen Kern im Herzen, 20x, Balken = 100 µm, PAS-Färbung; Insert: Balken = 20 µm

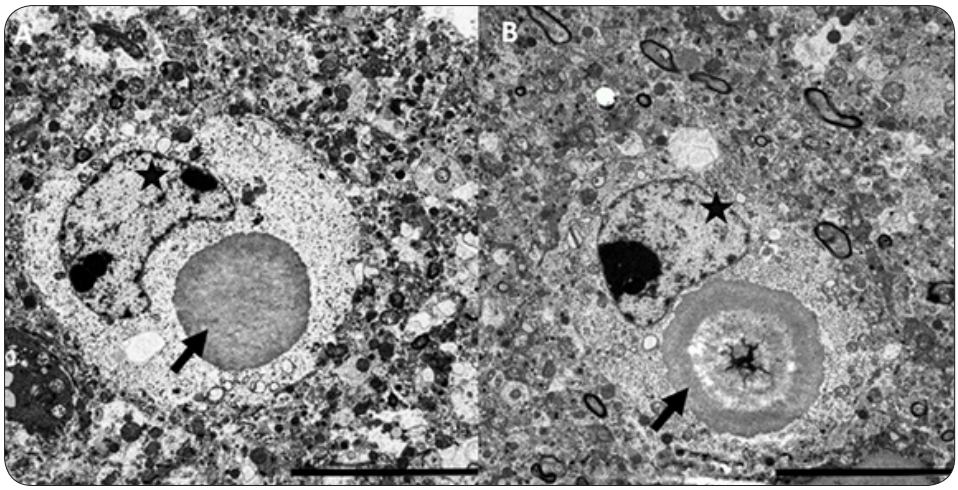


Fig. 2: Ultrastructural findings in the cerebellum show the different morphology of the inclusion bodies. (A) Purkinje cell with an intracytoplasmic inclusion body composed of filamentous, moderately electron-dense elements; Lafora-body (→) displaces the nucleus (⁎) eccentrically within the cell. (B) The Lafora-body (→) shows a prominent central core and peripheral radiation of electron-lucent and moderately electron-dense material; nucleus (⁎), scale bar = 10,000 nm. / Ultrastrukturelle Befunde im Kleinhirn zeigen eine unterschiedliche Morphologie der Einschlüsse. (A) Purkinje-Zelle mit einem intrazytoplasmatischen Einschluss, der aus fadenförmigem, mäßig elektronendichtem Material besteht. Der Einschluss (→) verdrängt exzentrisch den Zellkern (⁎) in der Zelle. (B) Der Einschluss (→) zeigt einen markanten zentralen Kern und eine periphere Ausstrahlung von elektronendurchlässigem und mäßig elektronendichtem Material; Kern (⁎), Balken = 10.000 nm.

and localization and morphology of the Lafora bodies are suggestive of an early diagnosed, pre-symptomatic case of Lafora disease. The clinical observation of very subtle changes such as "jaw smacking", impaired vision or urinary incontinence may be very challenging in groups of wild animals (Swain et al. 2017). A diagnosis of LD is based on clinical observations, histopathological investigations and genotypic examinations (Chambers et al. 2018). Lafora bodies may also represent senile changes.

In the present case, LBs were predominantly found in the CNS, especially in the Purkinje cells, the cerebral cortex and the hippocampus, with some also observed in the liver and heart. In symptomatic LD, LBs may be found in various organs including the CNS, skeletal muscles, skin, heart, urinary bladder, liver, spleen and eye (Gredal et al. 2003; Chambers et al. 2018). Within the CNS, LBs are found in the cerebrum, the cerebellum and the spinal cord in the perikarya and their processes and in glial cells (Turnbull et al. 2016; Chambers et al. 2018). The most severely affected regions in animals are the ganglionic and molecular layer of the cerebellum, the dentate nucleus and thalamic nuclei (Holland et al. 1970; Suzuki et al. 1979; Jian et al. 1990; Flegel et al. 2021; von Klopmann et al. 2021). In humans, LBs are reported to be rather diffusely scattered throughout the brain (Gorbunova et al. 2008). In many cases, the occurrence of LBs in the CNS is not associated with histopathological lesions (Honnold et al. 2010) or corresponds to mild changes such as senile lipofuscinosis (Jian et al. 1990; Minassian 2001), although reactive changes such as astrocytosis or microgliosis may occasionally be present (Chambers et al. 2018). Dogs affected with LD have a reduced thickness of the cerebral cortex and a decreased number of Purkinje cells (Chambers et al. 2018). As LBs are found in asymptomatic cases, the occurrence of LBs is not synonymous with the diagnosis of LD. However, incidental LBs in cats tend to be found in the neuropil rather than in nerve-cell bodies (Hall et al. 1998). In cattle, age-related LBs have been found in the liver, the thalamus and the rostral midbrain and the hepatocytes had a "ground-glass" appearance (Simmons 1994). However, the LBs in the liver may relate to asymptomatic, age-related lesions (Simmons 1994), or to early cases (Baumann et al. 1983) or late onsets of LD (Honnold et al. 2010; Turnbull et al. 2016).

The literature distinguishes three categories of LB based on their morphology in PAS staining. LB type I are uniformly PAS-positive, 3–10 μm in diameter and observed in the perikarya and neuropil of the cerebral cortex and the glial cells of the cerebellum (Jian et al. 1990). Type II have a strongly PAS-positive, homogenous core and radiating periphery, are 13–30 μm in diameter and can be found in the Purkinje cells of the cerebellum and the neurons of the midbrain (Jian et al. 1990). Type III have a peripheral ring of PAS-positive material and are 5–20 μm in diameter (Jian et al. 1990). The present case showed type I LBs in the cerebral cortex and the glial cells of the cerebellum and type II LBs in the Purkinje cells but type III LBs were absent. Ultrastructurally, two types of LBs can be distinguished. The first type consists almost entirely of faintly osmiophilic, fibrillary material (Holland et al. 1970), while the second type is characterized by dense, homogenous, extensive osmiophilic material (Holland et al. 1970; Suzuki et al. 1978; Jian et al. 1990). The LBs lack limiting membranes and are found in close association with the endoplasmic reticulum (Holland et al. 1970).

In humans, LD is an autosomal recessive, inherited neurodegenerative disorder caused by mutations in one of the three genes *EPM2A* (laforin), *EPM2B* (*NHLRC1*, malin) and *PRDM8* (Turnbull et al. 2012). Disease onset is in childhood or adolescence (Minassian 2001). The average age of onset in dogs is 6.94 years, although it can strongly vary (Jian et al.

1990; Swain et al. 2017). The molecular basis of LD in animals is unknown, except for the case of dogs, in which it has an autosomal recessive trait and is associated with a mutation in the *EPM2B* gene that results in a decreased expression of malin and accumulation of abnormal, insoluble glycogen in the cardiomyocytes, in myofibres in skeletal muscle, in hepatocytes, in epithelial cells of the apocrine sweat glands and in smooth muscle of the urinary bladder (Swain et al. 2017; Chambers et al. 2018). Accumulation of insoluble glycogen may lead to a disruption of axoplasmic flow and to neurological symptoms (Hall et al. 1998; Chambers et al. 2018). Polysaccharide bodies resembling LBs have been seen to accumulate after treatment of rats with D-penicillamine (Jayaraj 1980).

We describe LBs in a prairie dog, compare their neurological localization with that found in other species and draw attention to the spontaneous occurrence of LBs in wild animals. Unfortunately, the aetiology of the changes to the brain remains unclear and further studies will be needed to test whether the condition is inherited in prairie dogs. The discovery of a genetic mutation responsible for the accumulation of LBs and eventual LD in prairie dogs, and identification of the carrier gene, would help in the identification of carrier animals, which could be excluded from breeding programmes.

Conclusion

This is the first report of LB accumulation in a black-tailed prairie dog *(Cynomys ludovicianus)*. In age of onset, distribution of LBs, their morphological appearance and their occurrence, the case shows marked similarities with the changes reported in other species, including dogs. However, future studies will be required to investigate the pathogenesis of Lafora disease. A better characterization of the disease in black-tailed prairie dog colonies could help improve veterinary care and conservation efforts for this and other exotic species.

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Fazit für die Praxis:
Die Lafora(-ähnliche)-Krankheit kann bei Präriehunden auftreten und sollte in spontanen Todesfällen bei dieser Tierart berücksichtigt werden. Der Nachweis von Lafora-Körperchen in der histologischen Untersuchung weist auf eine Lafora-Krankheit hin, jedoch sind solche Veränderungen nur im Zusammenhang mit einem klinisch-anamnestischen Befund zu interpretieren. Spezialfärbungen, wie PAS und PAS-Diastase, sind hilfreich, um die Lafora-Körperchen von anderen Einschlüssen zu unterscheiden. Die Durchführung einer kommerziell verfügbaren, genetischen Untersuchung ist bei Wildtieren derzeit nicht möglich.

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