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Case Report
Wildlife Medicine

Segmental lumbar spinal cord aplasia in a free-ranging southern tiger cat (*Leopardus guttulus*)[1]

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ABSTRACT.- Tondo L.A.S., Pereira A.H.B., Fortes P.M., Negrão Watanabe T.T., Saranholi B.H., Freitas P.D., D'arc M., Santos A.F., Balthazar D.A. & Ubiali D.G. 2023. **Segmental lumbar spinal cord aplasia in a free-ranging southern tiger cat (*Leopardus guttulus*).** *Pesquisa Veterinária Brasileira 43:e07323, 2023.* Setor de Anatomia Patológica, Instituto de Veterinária, Universidade Federal Rural do Rio de Janeiro, Rodovia BR-465 Km 7, Seropédica, RJ 23890-000, Brazil. E-mail: danielubiali@ufrrj.br

We report a case of a free-ranging five-month wildcat with bilateral hind limbs paralysis since birth due to a segmental lumbar spinal cord aplasia. The species confirmation of the southern tiger cat (*Leopardus guttulus*) was determined by genetic sequencing. This southern tiger cat native to Brazil had autophagy in both pelvic limbs during the initial phase of hospitalization, followed by a right tibial fracture with bone exposition. Euthanasia was chosen due to animal welfare and submitted for *postmortem* examination. Grossly, there was an 8.5cm in-length segmental interruption of the spinal cord between the third and fifth lumbar vertebrae, with a lack of spinal cord tissue and collapsed associated dura mater. Microscopically, the representative sections of the L3 to L5 spinal cord had only .an irregular trace of gray matter adhered to the meninges (lumbar spinal cord aplasia) In the region of L6, a focally extensive, cystic, and well-defined tubular cavitation was noted dorsally to the central canal, replacing and compressing the adjacent nervous tissue (syringomyelia). Metagenomics examination did not detect any virus responsible for the presented spinal cord malformations. This seems to be the first description of segmental spinal cord aplasia reported in a wild feline.

INDEX TERMS: Biodiversity conservation, feline medicine, veterinary pathology, central nervous system, spinal cord, *Leopardus guttulus*.

RESUMO.- [**Aplasia segmentar da medula espinhal lombar em um gato-do-mato-pequeno (*Leopardus guttulus*).**] O objetivo do presente relato foi descrever um caso de aplasia segmentar da medula espinhal lombar em um felino silvestre, com aproximadamente cinco meses, resgatado de seu ambiente natural, apresentando paralisia bilateral dos membros posteriores. A espécie gato-do-mato-pequeno (*Leopardus guttulus*) foi determinada por sequenciamento genético. Após curto período de hospitalização, iniciou autofagia de ambos os membros pélvicos, seguido de fratura com exposição óssea. Optou-se pela eutanásia e o cadáver foi encaminhado ao Setor de Anatomia Patológica da Universidade Federal Rural do Rio de Janeiro para necropsia. Macroscopicamente, havia uma interrupção segmentar grave da medula espinhal entre

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a terceira e a quinta vértebras lombares, medindo 8,5cm de comprimento, com resquícios de tecido nervoso e com meninges colapsadas. Ao exame histológico, em seções da medula espinhal na região de L3 a L5, .havia apenas vestígio de tecido nervoso aderido às meninges, morfologicamente compatível com substância cinzenta (aplasia de medula espinhal lombar). Na região de L6 notou-se áreas multifocais com cavitações tubulares, císticas e bem delimitadas, dorsalmente ao canal central substituindo e comprimindo o tecido nervoso adjacente (siringomielia). O exame de metagenômica não detectou qualquer vírus responsável pelas malformações da medula espinhal. Com base no histórico, sinais clínicos, necropsia e achados histológicos, o diagnóstico de aplasia segmentar grave com siringomielia foi estabelecido em um *L. guttulus*.

TERMOS DE INDEXAÇÃO: Conservação da biodiversidade, medicina felina, patologia veterinária, sistema nervoso central, medula espinhal, *Leopardus guttulus*.

INTRODUCTION

Congenital spinal cord malformations occur in several animal species, including syringomyelia, absence or duplication of the central medullary canal, hydromyelia, aplasia, and segmental hypoplasia (Lahunta & Glass 2009). The pathogenesis of these processes results from defects in neural tube closure (Deforest & Basrur 1979). Spinal cord segmental aplasia is a myelodysplasia rarely reported in humans and animals, especially wild animals. It is characterized by an incomplete formation of a spinal cord segment during embryogenesis (Negrin et al. 2009). Neurological signs can be observed in animals at birth and become progressive. The clinical signs include ataxia, paraplegia, and lack of sensitivity to deep pain (De Lahunta 1983). Syringomyelia consists of the appearance of cavitary formations with an accumulation of cerebrospinal fluid liquid in the spinal cord without connection with the ependymal canal (Tudury et al. 2000). The diagnosis of myelodysplasia is based on the history, clinical signs, and imaging tests such as myelography and magnetic resonance imaging. It has lately been confirmed by postmortem examination and histopathology (Albernaz et al. 2014). The prognosis is poor, as the complications of the malformation make sensory and motor recovery impossible, in addition to the inability to regenerate the nervous tissue (De Lahunta 1983).

In veterinary medicine, morphologic alterations in the spinal cord, such as *Spina bifida*, neural tube closure defects, spinal duplication, segmental hypoplasia (SHSC), aplasia (SASC) of the spinal cord, and syringomyelia are classified as myelodysplasias (Tudury et al. 2000, Imai & Moritomo 2009, Binanti et al. 2013, Souza et al. 2020). Segmental spinal cord hypoplasia is the incomplete formation of a segment of the spinal cord (De Lahunta 1983, De Lahunta & Glass 2009). Myelodysplasias are reported in various species, including domestic felines (Albernaz et al. 2014), dogs (Dodd et al. 2020) and bovines (Souza et al. 2020). It can be associated with viral infections, toxic and drug expositions (exogenous causes), and inheritance or congenital origins (endogenous causes) (Deforest & Basrur 1979, Rusbridge & Knowler 2003, Minato & Baroni 2018, Souza et al. 2020). In domestic cats, congenital cerebellum hypoplasia can be related to intrauterine Feline Panleukopenia Virus (FPLV) infection (Sharp et al. 1999, Poncelet et al. 2013). However, to this date, few studies have associated viral agents with spinal cord segmental hypoplasia in domestic, wild, and zoo animals (Souza et al. 2020). Herein, we describe the clinical and pathological findings of a wild *Leopardus guttulus* kitten with paralysis due to a spinal cord lumbar segmental aplasia.

CASE REPORT

A wild five-month *Leopardus guttulus* kitten was rescued in the metropolitan region of Rio de Janeiro State, showing bilateral hind limb paralysis. The feline was referred to the teaching veterinary hospital for clinical evaluation. The central nervous testing related to the brain functions testing was normal. Motor and sensory testing of the central nervous revealed severe dysfunction related to spinal cord functions. The kitten had autophagic behavior, biting the left pelvic limb, and a limb amputation surgery was performed. A few hours after surgery, the cat had autophagic behavior, biting the right pelvic limb, and euthanasia was elected. The haired skin, skeletal muscles, and bones from the right pelvic limb had a marked laceration, hemorrhage and tibial fracture related to autophagy and dragging (Fig.1).

On postmortem examination, the cat measured 21cm from the atlantooccipital joint to the sacral bone and 21cm from the scapular cartilage to the distal phalanx of the right arm. The body condition score was 3 (1 to 5 scale) and weighed 1.09kg. The left pelvic limb was absent, with a suture line measuring 2.1cm. The right pelvic limb had an external communitive fracture that extended from the tibia's proximal epiphysis to the diaphysis. There was a marked muscular atrophy from the second lumbar vertebrae to the sacral bone. The *longissimus dorsi*, the *gluteus medius*, and the *quadratus lumborum* muscles are diffusely pallor, soft, and moderately smaller (atrophy). After the exposure of the spinal cord canal, there was an 8.5cm in-length segmental interruption of the spinal cord from the third (L3) to the fifth (L5) lumbar vertebrae. After opening the dura mater layer, there was a complete lack of spinal cord tissue in the sixth lumbar vertebrae. Several

Fig.1. Segmental lumbar spinal cord aplasia in a free-ranging southern tiger cat (*Leopardus guttulus*). The kitten had extensive deep muscle and bone exposition due to self-traumatic injury in the right pelvic limb. Although the extensive and severe hind limb lesion, no apparent pain signs were observed. The central neuronal clinical signs were related to spinal cord functions, and the brain functions were normal.

organs were sampled in 10% buffered formalin solution and routinely processed for histological evaluation.

The microscopic findings of the representative segments of the spinal cord from L3 to L5 revealed that the white matter was severely reduced with the complete effacing of the adjacent gray matter and composed of a few remaining axons (Fig.2 and 3). A focally extensive, cystic, and well-defined tubular cavitation was noted dorsally to the central canal, replacing and compressing the adjacent nervous tissue (syringomyelia). In the sections of L6, the spinal cord architecture was markedly disorganized with loss of the dorsal and ventral gray matter conformation (Fig.4 and 5). Within the gray matter, multifocally, the neurons were necrotic, showing hypereosinophilic cytoplasm, pyknotic and karyorrhectic nuclei (Fig.6). The skeletal muscles had myocytes undergoing degeneration and necrosis (loss of nuclear and cellular details, loss of striations, hypereosinophilic sarcoplasm, and pyknotic and karyorrhectic nucleus) or atrophied (retracted sarcoplasm and a decrease in the cellular size). The skeletal muscle fibers were replaced by mature, well-differentiated adipose tissue.

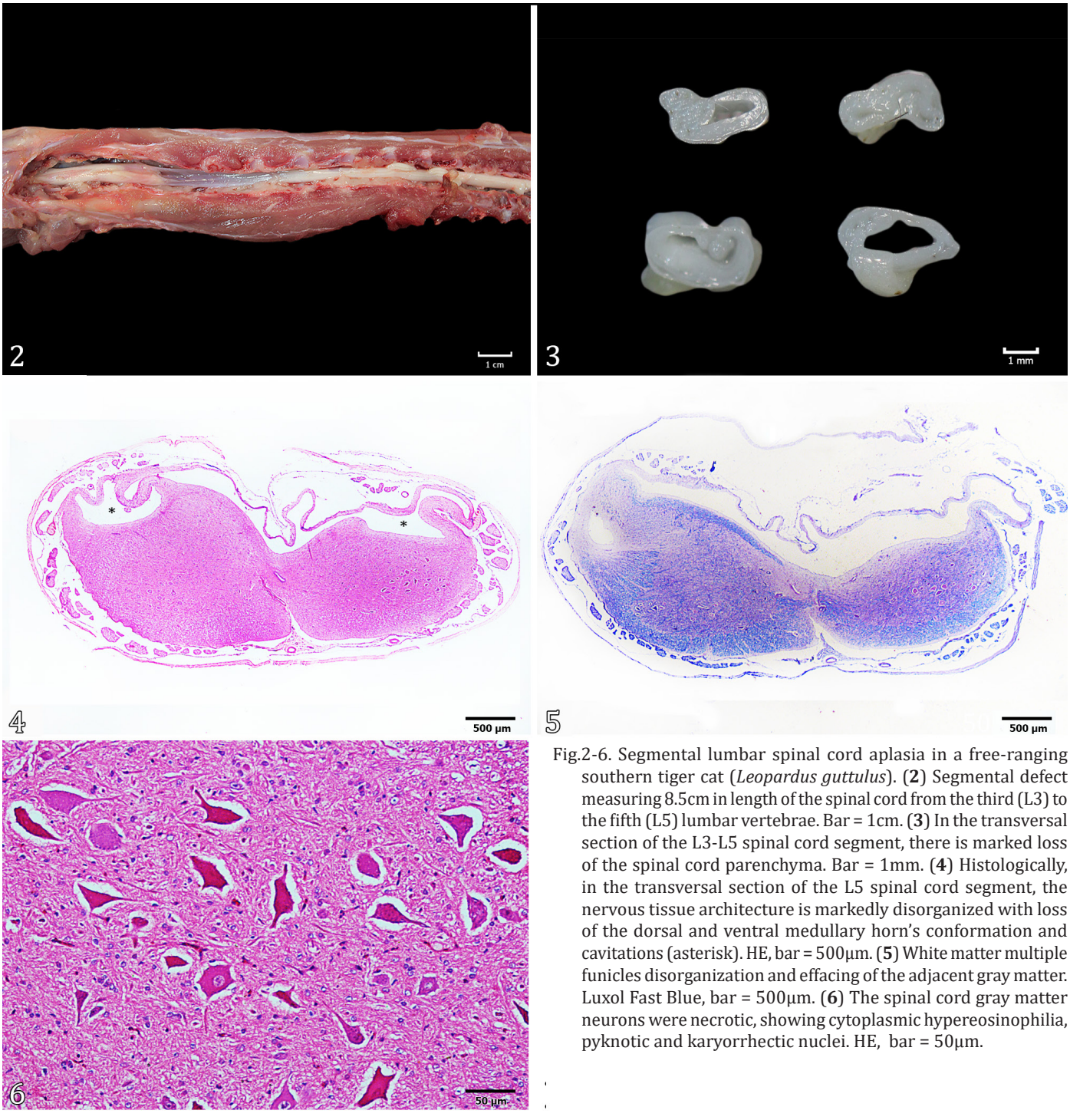

Fig.2-6. Segmental lumbar spinal cord aplasia in a free-ranging southern tiger cat (*Leopardus guttulus*). (**2**) Segmental defect measuring 8.5cm in length of the spinal cord from the third (L3) to the fifth (L5) lumbar vertebrae. Bar = 1cm. (**3**) In the transversal section of the L3-L5 spinal cord segment, there is marked loss of the spinal cord parenchyma. Bar = 1mm. (**4**) Histologically, in the transversal section of the L5 spinal cord segment, the nervous tissue architecture is markedly disorganized with loss of the dorsal and ventral medullary horn's conformation and cavitations (asterisk). HE, bar = 500µm. (**5**) White matter multiple funicles disorganization and effacing of the adjacent gray matter. Luxol Fast Blue, bar = 500µm. (**6**) The spinal cord gray matter neurons were necrotic, showing cytoplasmic hypereosinophilia, pyknotic and karyorrhectic nuclei. HE, bar = 50µm.

Investigation into the genetic sequencing for determining the feline species was performed. For the DNA-based species identification, DNA was isolated from a skeletal muscle sample of the kitten using a salt extraction protocol (Aljanabi & Martinez 1997) and quantified using a biophotometer (Eppendorf). Then, polymerase chain reactions (PCRs) were performed to amplify mitochondrial fragments of cytochrome c oxidase subunit 1 (COI) and 16S rRNA genes using the primer pairs described by Folmer et al. (1994) and Palumbi et al. (1991), respectively. PCRs were performed in a Veriti thermal cycler (Applied Biosystems) following the recommendations proposed by Xavier et al. (2019). Amplified products were first visualized under a UV transilluminator after electrophoresis (agarose gel 1% with GelRed and bromophenol blue) and then purified using ExoSAP-IT (Affymetrix). Sequencing was performed on an ABI3730XL (Applied Biosystems), and the electropherograms were analyzed with Geneious 7.1.7 (Kearse et al. 2012). The sequences obtained were subjected to comparative alignments in GenBank[8] and BoldSystems[9] databases, resulting in a similarity of 99.35% for the southern tiger cat *L. guttulus* (Hensel 1872) (private data, no ID available) and 96.15% for *Leopardus tigrinus* (GTICO061-18) according to BoldSystems. No *L. guttulus* sequence was available in GenBank, and a maximum similarity of 95.80% for *L. tigrinus* (KP202287.1; NC_028317.1) was retrieved from this database search.

Metagenomics was performed at the central nervous system (brainstem, cerebellum, forebrain, and spinal cord) and cerebrospinal fluid samples for viral identification, following the protocol developed by Cosentino et al. (2022). Metagenomics examination did not detect any virus associated with the presented spinal cord malformations.

DISCUSSION AND CONCLUSION

The diagnosis of severe segmental lumbar spinal cord aplasia was established based on the history, clinical signs, necropsy, and histological findings. The gross and histological findings observed in the L3 to L5 segment of the spinal cord are compatible with severe segmental aplasia with syringomyelia, subjacent muscular degeneration, necrosis, atrophy, and neuronal necrosis within the spinal cord. All samples were negative for any virus at molecular investigation.

The species confirmation was done by molecular analyses since *Leopardus guttulus* and *Leopardus tigrinus* have very similar morphological features and overlap with species feature differentiation (Nascimento 2010, Trigo et al. 2013, Payan & Oliveira 2016). The genus *Leopardus* is represented by small neotropical felids in Brazil, Bolivia, Paraguay, and northern Argentina (Trigo et al. 2013). Both *L. guttulus* and *L. tigrinus* populations are classified as Vulnerable (VU) by the red list of the International Union for Conservation of Nature (Oliveira et al. 2016)

Segmental spinal cord aplasia has already been described in domestic animals (Tudury et al. 2000, Imai & Moritomo 2009, Binanti et al. 2013, Albernaz et al. 2014, Souza et al. 2020). This condition is usually identified at a young age in domestic cats because of the progressive and prominent neurological clinical signs (Tudury et al. 2000, Negrin et al. 2009, Albernaz et al. 2014) associated with a poor prognosis. Some typical clinical signs include paraparesis or paraplegia and urinary and fecal incontinence (De Lahunta 1983). Although manageable in domestic cats, the clinical signs represent a survival incapacity for wild felines. Herein, the *L. guttulus* would not be able to survive in nature due to the impossibility of hunting, movement, and the possibility of infection development due to urinary and fecal incontinence and self-mutilation behavior. The self-mutilation observed in this case is related to proprioception and profound pain perception loss. In small animals, self-mutilation can be related to obsessive-compulsive disorders of neurological origin, pruritus, chronic pain, and spinal cord lesion (Overall & Dunham 2002, Dodd et al. 2020).

Negative molecular results for retroviruses do not rule out viral infections as etiology of spinal cord segmental hypoplasia in early-phase gestation infections (Souza et al. 2020). In the present case, there is no association between myelodysplasia and exogenous or endogenous causes in wild cats. Feline Panleukopenia Virus infections cause cerebellar hypoplasia in cats and wild animals (Aeffner et al. 2006, Poncelet et al. 2013, Wünschmann et al. 2020). In this case, no viral infection or viral agents were identified in the organs sampled, although this possibility should not be excluded and must be further investigated in future cases. Intrauterine infections could cause congenital lesions with possible negative viral molecular results at the neonatal and kitten age sampling. Transboundary and spillover viral infections occur between domestic and wild animals (Van Moll et al. 1995, Beineke et al. 2015) and can intensify in peri-urban forests. Infections of wild felines with a common virus from domestic cats have already been described (Jessup et al. 1993, Ostrowski et al. 2003, Sacristán et al. 2021, Liu et al. 2022).

To the authors' acknowledgment, this is the first case report of a congenital segmental spinal cord aplasia in a wild feline. Although no cause for this congenital myelodysplasia was confirmed, a viral infection should not be completely ruled out, and further medical investigation is warranted.

Conflict of interest statement.- The authors declare that there are no conflicts of interest.

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