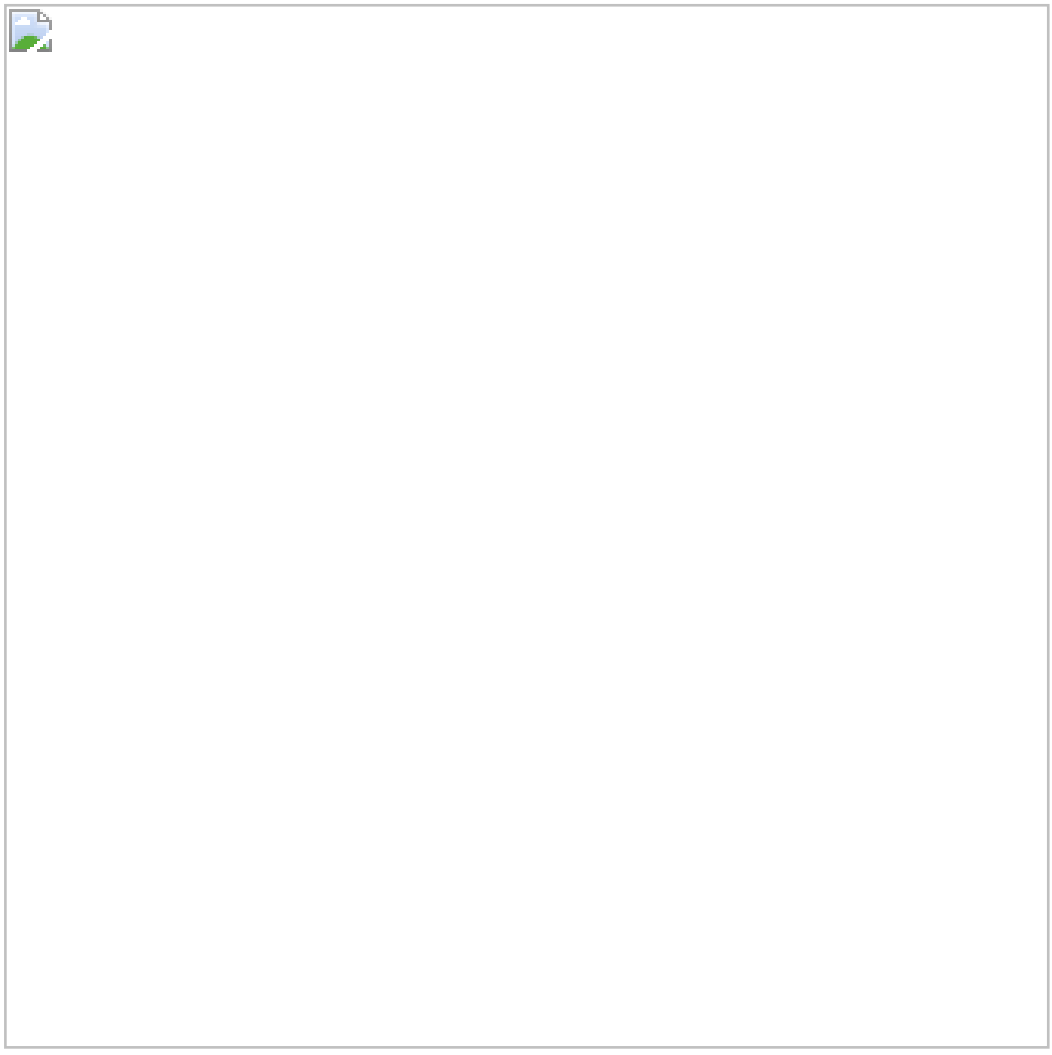

Limited dorsal myeloschisis in three cats: a distinctive form of neural tube defect

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Limited dorsal myeloschisis in three cats: a distinctive form of neural tube defect

Sarah Butterfield[1], Beatriz Garcia-Gonzalez[2], Colin J Driver[1]* and Clare Rusbridge[1,3]

Abstract

Case series summary The aim of this case series was to describe the clinical presentation, imaging findings and histopathology of three cats with limited dorsal myeloschisis (LDM). The history, examination and MRI sequences were reviewed in three cases presented to a single referral hospital. The surgery report and histopathology were described in two cases. All cats were young (10 weeks old, 5 months old, 4 years old), presenting with varying degrees of progressive paraparesis. All had a midline skin defect overlying the spinal column that was either sunken or saccular, containing fluid thought to be cerebrospinal fluid. MRI sequences demonstrated tissue extending from the dura through an overlying bifid spinous process and attached to the dermis, with associated spinal cord tethering, atrophy and syringomyelia. Lesions were located at L2–L3, T8–T9 and L4. Histopathology described a fibroneural stalk with a glio-ependymal lining, surrounded by glial nests and nerve fibres. The youngest and most severely affected was euthanased, while the other two underwent surgery. Both regained independent ambulation with persistent paraparesis; however, one required ongoing management of urinary incontinence.

Relevance and novel information LDM is a primary neural tube defect that may result in neurological deficits, including bladder dysfunction, and is characterised by a fibroneural stalk between the dermis and the spinal cord. Distinct MRI features, such as a visible intrathecal tract, dorsally tethered cord and syringomyelia, help distinguish this condition from the clinically similar dermoid sinus. The presence of progressive neurological signs, with a palpable midline defect overlying the affected spinal cord segment, may raise suspicion for this clinical entity in veterinary patients.

Keywords: Limited dorsal myeloschisis; neural tube defect; spinal dysraphism; dermoid sinus; spinal malformation; midline defect; tethered cord

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Introduction

Neural tube defects (NTDs) are rarely reported in companion animals and refer to a collection of congenital malformations that are the result of abnormal development during embryogenesis.[1] Limited dorsal myeloschisis (LDM) is a distinctive form of NTD, described in humans, characterised by a focal 'closed' midline skin defect and a fibroneural stalk linking the skin lesion to the underlying cord.[2] It is a result of failure of the neural tube to close, with skin ectoderm remaining attached to the borders of the neural plate.[3] This prevents the vertebral arches forming, meaning this condition is always seen concurrently with a bifid

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vertebra. Histopathology provides a definitive diagnosis, describing glial tissue within the fibroneural stalk either with scattered neurons within mesenchymal tissue or arrangement in glial nests.[2]

A dermoid sinus, another form of NTD, forms as a result of the failed separation of the neural tube from the skin ectoderm.[1] It appears clinically similar to LDM and is most commonly documented in the Rhodesian Ridgeback,[4,5] where an autosomal dominant gene mutation that creates the dorsal hair ridge predisposes them to the condition.[6,7] A visible midline skin defect is continuous with a ventral tubular sac that may be classed as type I–V, depending on tissue depth penetrated.[4,8] In comparison with LDM, histologically the lumen consists of sebum, keratin debris and hair follicles. Hair may be seen to protrude from the sinus orifice, which, if pulled, may lead to acute development of neurological signs in previously normal animals.[9] Neurological signs may occur if the dermoid sinus connects to the dura mater, as in type IV, and can be critical if infection tracks to these structures. For this reason, dermoid sinuses that discharge, become infected or cause neurological deficits, are often treated surgically.

Spina bifida, the most common form of NTD in humans, refers to a congenital failure of one or more vertebral arches to close over the spinal cord.[1] Three types have been described: occulta (asymptomatic with no neural tissue involvement); associated with a meningocoele (protrusion of the meninges through the bifid vertebra); or associated with a meningomyelocoele (protrusion of meninges and spinal cord tissue).[10]

The intention of this case series was to describe three cats, two of which were juveniles, that presented with varying degrees of paraparesis and neurological deficits, as well as a visible skin lesion in the localised spinal cord segment. MRI findings in all cases were consistent with a diagnosis of LDM, confirmed by histopathology in the two cases that underwent surgical management. This has never previously been described in veterinary patients.

Case series description

Case 1

A 4-year and 8-month-old male neutered domestic shorthair (DSH) cat presented with a 4-month history of acute-onset, slowly progressive paraparesis following a minor traumatic episode. A similar episode was noted 1 year previously, which resolved with 2 weeks of conservative treatment. Urination was normal. On examination, the cat was strongly ambulatory, and displayed symmetric spastic paraparesis and pelvic limb ataxia. Proprioceptive paw positioning and hopping reactions were slightly delayed on the pelvic limbs with normal segmental spinal reflexes. No pain response was elicited on direct spinal palpation. The cat's neurological deficits were localised to a T3–L3 myelopathy. There was a palpable depression in the vertebral column in the cranial lumbar region that had been present since birth. MRI of the thoracolumbar vertebral column revealed an L2–L3 malformation (Figure 1a,b). There was incomplete closure of the dorsal vertebral column at L2–L3 with extension of the meninges through the defect to create a tissue stalk attaching to the overlying skin. There was marked atrophy of the spinal cord at this level with a tethered cord effect and syringomyelia. There was also dilation of the subarachnoid space with cerebrospinal fluid (CSF).

It was elected to treat the cat surgically owing to the progressive nature of the paraparesis. The surgical approach involved dissection of the fibroneural stalk from the surrounding subcutaneous tissues, a dorsal laminectomy of the L3 vertebra and removal of the stalk by durotomy. The dura was left open and the laminectomy defect covered by collagen sponge. Immediate postoperative analgesia included opioid and non-steroidal anti-inflammatory drugs (NSAIDs). Following surgery, the cat deteriorated neurologically to become non-ambulatory with minimal voluntary pelvic limb movement and absent postural reactions. Following 1 week of hospitalisation and physiotherapy it became ambulatory, similar to the preoperative status, with reflex urination. One month after surgery, it had continued to improve in activity. Spastic paraparesis and pelvic limb ataxia remained with voluntary urination and defaecation, although occasionally in inappropriate locations.

The cat presented 9 months after the initial surgery with a 2-month progressive non-painful deterioration in pelvic limb ataxia. On presentation, it was non-ambulatory with marked spastic paraparesis, absent proprioceptive paw positioning in the pelvic limbs and normal segmental spinal reflexes. Repeat MRI revealed suspected adhesion of the dura to the overlying laminal defect with persistent subarachnoid dilation (Figure 2). A revision dorsal laminectomy and durotomy confirmed this and larger surgical margins were created. A porcine intestinal submucosal graft was placed over the dural defect and bovine collagen sponge over the laminectomy site. One month after surgery the cat had shown slowly progressive improvement to regain independent ambulation and urination.

Histology of the resected tissue was consistent with a diagnosis of LDM (Figure 3). The stalk extended from the superficial dermis into the subcutis and consisted of glial tissue supported by thick bundles of collagen. The glial tissue presented as streams, sometimes containing few neurons, as well as multifocal nests that were embedded within an abundant eosinophilic matrix. This tissue surrounded a cavitated space lined by a glio-ependymal lining. Also noted within the stalk were some nerve fibres. Bands of condensed fibrous tissue, adipose tissue

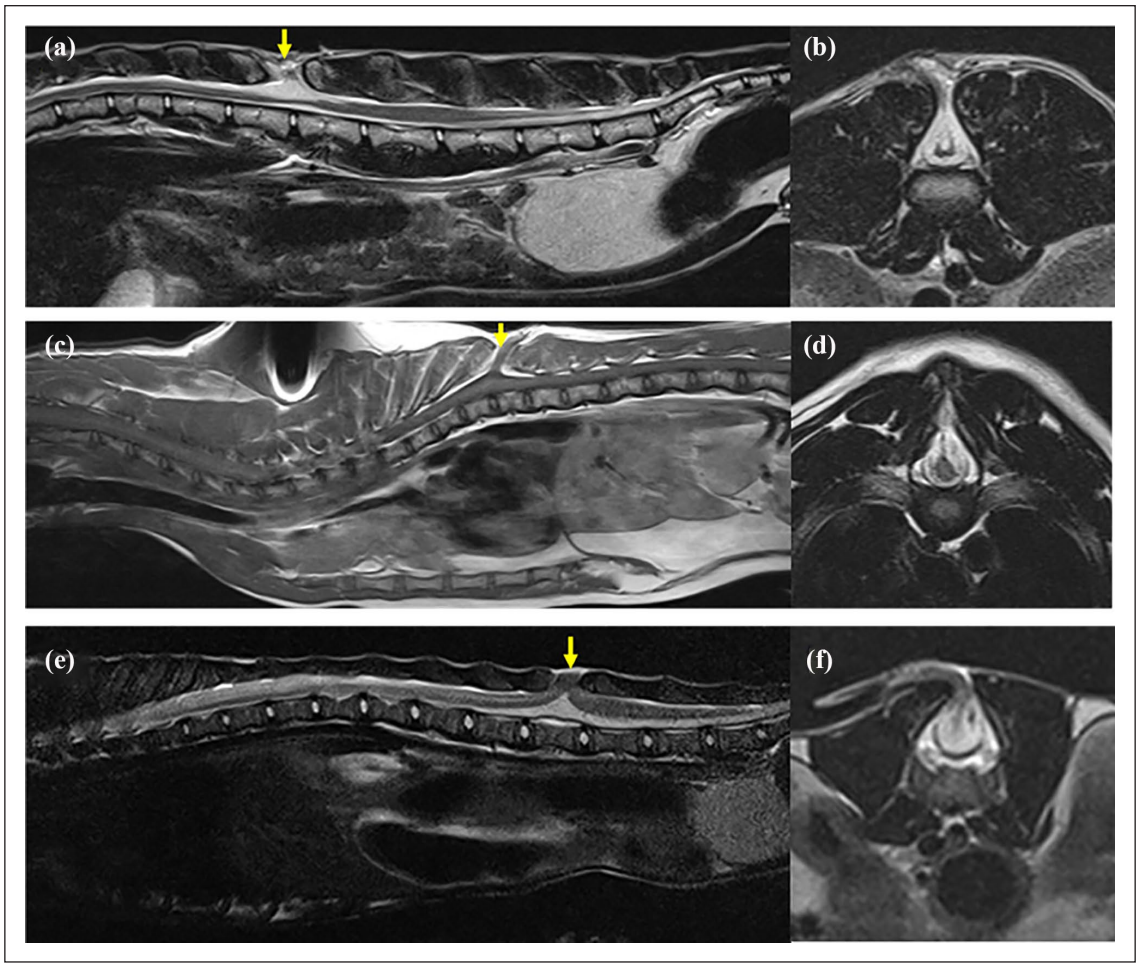

Figure 1 MRI sequences of all three cases. All lesions were predominantly hyperintense on T2-weighted (T2W) images and isointense on T1-weighted (T1W) images, with no clear contrast enhancement following gadolinium administration. Spinal cord dorsal tenting and atrophy was a common feature. (a) T2W midsagittal sequence of the spinal column from T11 to S3 in case 1 showed incomplete closure of the dorsal vertebral column. Tissue extending from the dura is seen attaching to the overlying dermis (arrow), with spinal cord tenting, atrophy and syringomyelia. (b) T2W transverse sequence at the level of L2–L3 in case 1 demonstrating the stalk attaching to the dermis. (c) T1W midsagittal sequence of the spinal column from C2 to L2 in case 2 showed tissue extending from the dura to the dermis at the level of T8–T9 (arrow). (d) T2W transverse sequence at the level of T8 in case 2. (e) T2W midsagittal sequence of the T5–S1 spinal column in case 3 displaying the midline defect at the level of L4 (arrow). (f) T2W transverse sequence at the level of L4 in case 3

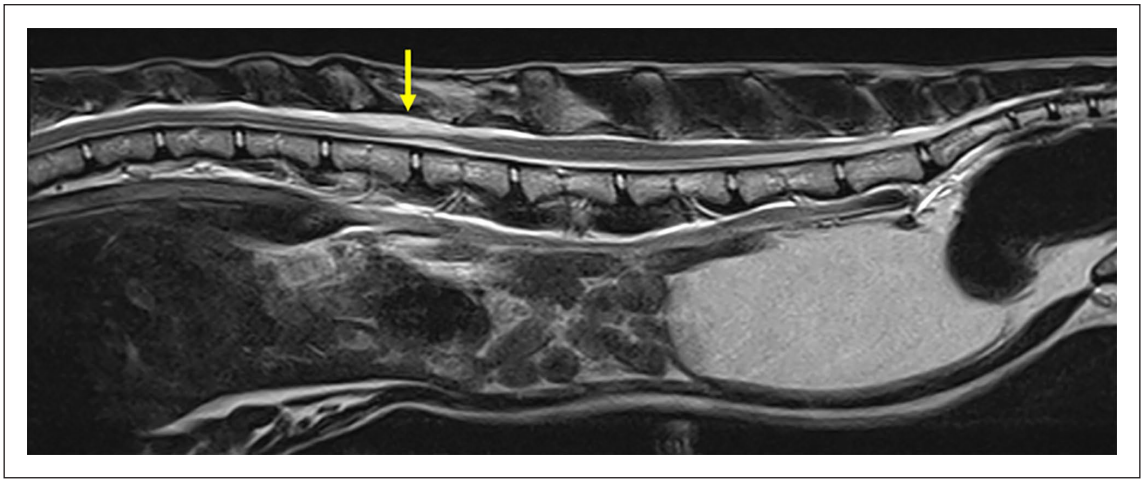

Figure 2 T2-weighted midsagittal MRI from T11 to S3 in case 1 showed adhesion of the dura to the previous dorsal laminectomy site with persistent dilation of the subarachnoid space with cerebrospinal fluid and associated cord compression (arrow)

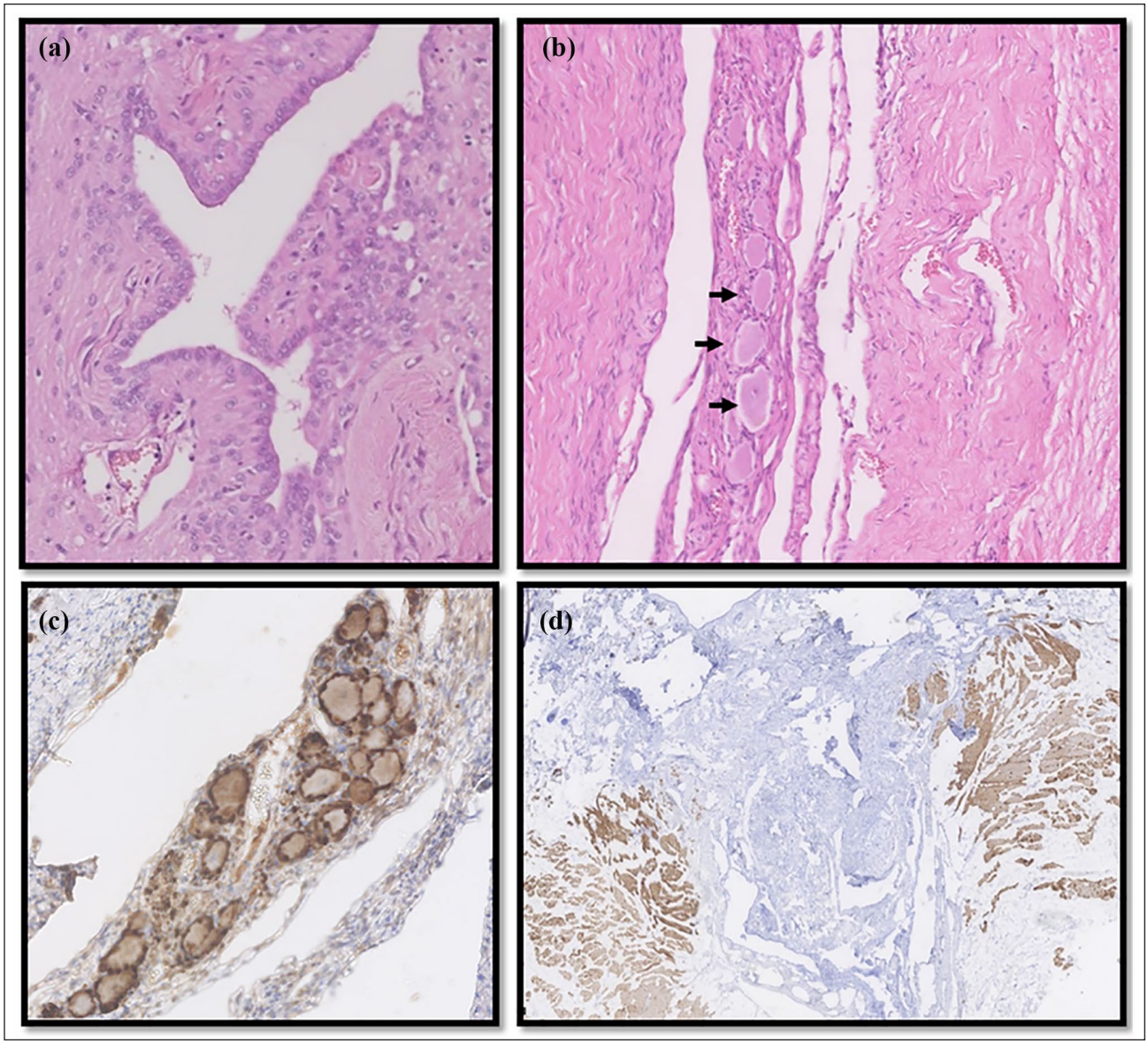

Figure 3 Histopathology with haemotoxylin and eosin stain from case 1 were consistent with a diagnosis of limited dorsal myeloschisis (LDM). Additional immunohistochemistry confirmed cells were of neural origin. (a) A cavitated space within the centre of the fibroneural stalk is seen lined by an epithelium, consistent with glioependymal tissue from case 1 (× 20). (b) Neuronal bodies (arrows) shown from case 1 are characteristic of LDM (× 10). (c) Immunohistochemistry. Positive staining with S100 confirms that these cells are of neural origin (× 2.5). (d) Immunohistochemistry. Clusters of glial fibrillary acidic protein (GFAP)-positive glial cells are seen within the fibroneural stalk. The glioependymal lining also stains positively with GFAP (× 2.5)

and bundles of skeletal muscle were noted around the stalk. Additional immunohistochemistry of case 1 documented S100 and glial fibrillary acidic protein-positive cells within the stalk, confirming they were of neural origin.

Case 2

A 5-month-old male neutered Bengal cat presented with a 3-day history of acute onset paraparesis, which was possibly secondary to an unwitnessed traumatic event. Urination was normal. On presentation, the cat was ambulatory paraparetic with normal proprioceptive paw positioning and segmental spinal reflexes. No pain response was elicited on direct spinal palpation. The cat localised to a T3–L3 myelopathy. It had a distinct area of whorled hair overlying the T8 vertebra dorsally (Figure 4a).

Haematology and biochemistry revealed a mild anaemia (haematocrit 28.8%; reference interval [RI] 30.3–52.3%) and mild elevation in phosphate (2.45 mmol/l; RI 1.00–2.42 mmol/l). Initial investigations included electromyography and nerve conduction velocity testing to assess for chronic polyneuropathy previously reported in a cohort of young Bengal cats.[11]

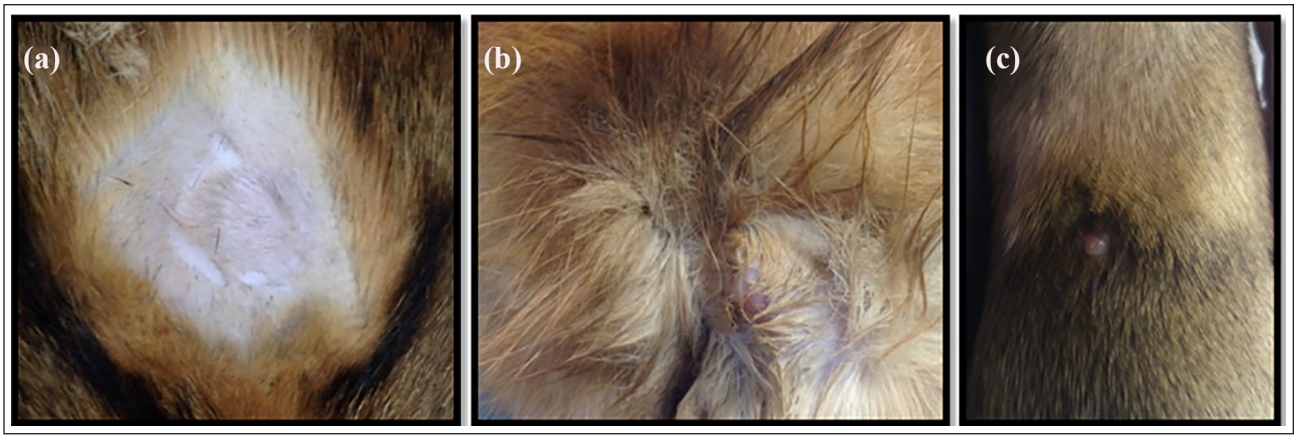

Figure 4 The cutaneous defects varied in appearance. (a) An area of whorled hair with no palpable skin depression was seen at the level of T8–T9 vertebrae in case 2. (b) A saccular cutaneous lesion was evident overlying the mid-lumbar region with fluid discharge, likely cerebrospinal fluid in origin, as a result of rupture of the membranous sac in case 3. (c) A suspected limited dorsal myeloschisis cutaneous saccular lesion was seen in a juvenile pug not included in this study, but included here to show the characteristic appearance

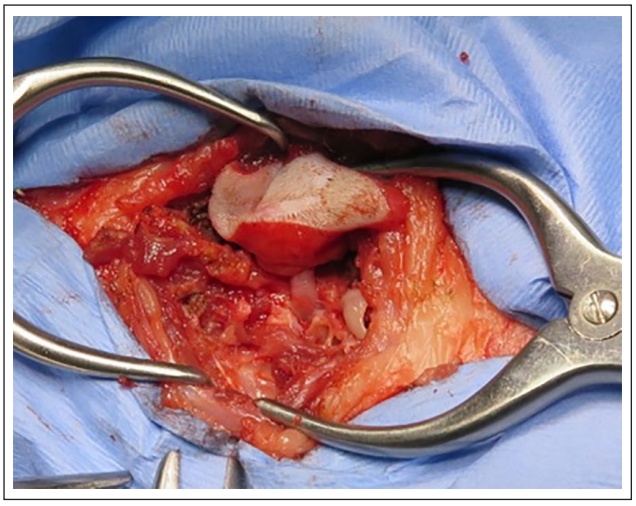

Figure 5 Intraoperative image of case 2 showing the fibroneural stalk extending to attach to the overlying dermis

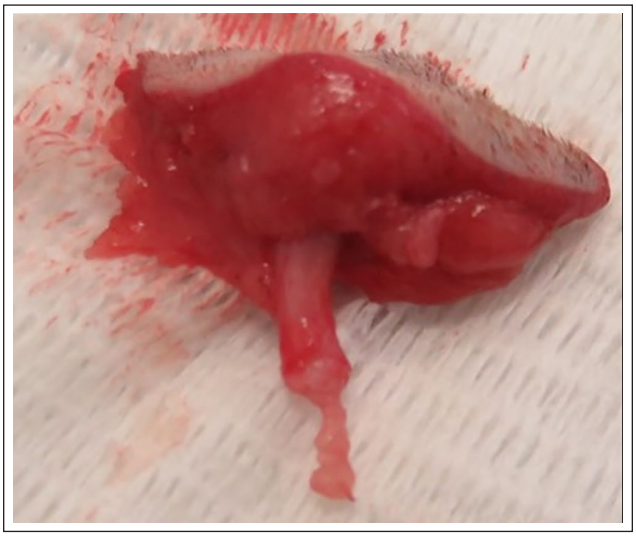

Figure 6 The fibroneural stalk can be seen in its entirety attaching to the overlying dermis after complete surgical excision (case 2)

This was deemed an unlikely cause given that the clinical signs of polyneuropathy would manifest as 'lower motor neuron' weakness rather than upper motor neuron, as in this case. These results were normal. MRI of the thoracolumbar vertebral column (Figure 1c,d) confirmed the presence of a defect in the dorsal lamina at T8–T9, with dorsal elevation of the spinal cord and dorsolateral cord compression.

A dorsal surgical approach to the defect was made (Figure 5). The fibroneural stalk was dissected from surrounding tissues (Figure 6) and a dorsal laminectomy performed. The tissue was seen to attach to the meninges and also to the spinal cord. A circular durotomy was performed around the lesion, and the dura left open. Immediate postoperative analgesia included opioid and NSAIDs. The cat remained ambulatory but ataxic immediately after surgery and was discharged with rehabilitative care after 7 days of hospitalisation. It continued to improve and had mild pelvic limb ataxia at 14 days following surgery. Despite good voluntary movement, the cat remained unable to urinate voluntarily and so was managed by manual expression, diazepam, prazosin and bethanecol. This resolved after 4 weeks of supportive treatment.

Histology of the resected tissue was consistent with a diagnosis of LDM (changes seen similar to that in Figure 3). Again, the lesion showed fibroneural tissue extending from the superficial dermis into the subcutis. The lesion was surrounded by thick bands of fibrous tissue. Although nervous tissue could not be demonstrated on

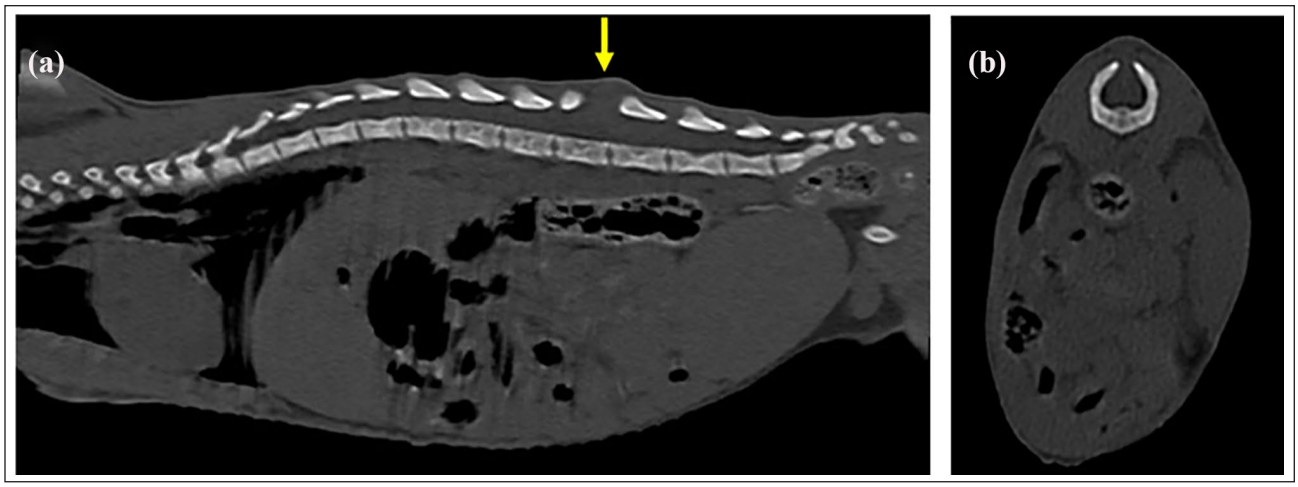

Figure 7 CT images showing the defect in case 3. (a) Midsagittal CT sequence from T1 to S3 showing the defect in the L4 vertebra (arrow). (b) Transverse CT sequence at the level of L4 vertebra

the slides available, immunohistochemistry showed many spindled-to-stellate cells present within the structure that exhibited S100 positivity, indicating a neuronal or glial tissue origin.

Case 3

A 10-week-old male entire DSH cat presented with congenital non-ambulatory paraparesis and pelvic limb proprioceptive deficits. The cat was also noted to have a subcutaneous mass lesion overlying the mid lumbar vertebral column, which had previously shown discharge of a clear fluid suspected to be CSF (Figure 4b). Proprioceptive paw positioning and hopping reactions were absent on the pelvic limbs, with slightly reduced withdrawal reflex and apparently absent pelvic limb and tail nociception. The cat was reportedly urinary and faecally continent with no evidence of spinal pain. Thoracic limb function, cranial nerves and perineal reflexes were unremarkable. The cat localised to a L4–S3 myelopathy.

Serum haematology and biochemistry showed a slightly decreased urea (42 µmol/l; RI 53–141 µmol/l) and mild elevation in chloride (134 mmol/l; RI: 115–126 mmol/l). CT (Figure 7) and MRI (Figure 1e,f) of the thoracolumbar spine were performed, revealing a bifid abnormality of the L4 vertebra. The spinal cord appeared tethered at this level with a communication between that and the cutaneous mass. The images were consistent with a diagnosis of LDM with a saccular skin lesion. The previous fluid discharge noted from the mass was considered highly likely to be CSF in origin. Owing to the poor prognosis for neurological improvement, this cat was euthanased shortly after diagnosis and no histopathology was available to support our imaging findings.

Discussion

LDM is a distinctive form of NTD characterised by an intrathecal extraspinal fibroneural stalk extending from the dura to the overlying dermis with a visible external midline defect closed by a bridging layer of squamous epithelium.[2] It is thought to be a result of incomplete disjunction between cutaneous and neural ectoderms during embryogenesis.[1–3] Development of the surrounding myofascial tissue continues, and so the neural tube is progressively pulled deeper into the body, leaving a dorsal median neural tissue. This results in incomplete dorsal midline fusion of the skin that eventually becomes bridged by an epithelial membrane.[2]

Two types of cutaneous lesion have been described in the human literature:[2] non-saccular (with a flat or sunken squamous epithelial crater or pit); and saccular (a skin-based CSF-filled sac covered by a squamous epithelial dome [see Figure 4c for a suspected example in a juvenile Pug]). Three distinguishable internal sac types have been proposed from imaging findings:[2] a saccular myelocystocoele; saccular-dome stalk; or saccular-basal nodule. Additionally, transitional skin lesions may occur where there is swelling of an otherwise flat skin surface following straining, presumably secondary to CSF being forced through a usually collapsed dural fistula.[2] LDMs in humans have been documented in all regions of the vertebral column cranial to the conus medullaris.[2]

Three characteristic features specific to an LDM have been defined in human medicine to aid in its diagnosis. MRI demonstrates a focal cutaneous lesion (midline crater or saccular swelling), a well-circumscribed internal fibroneural stalk connecting the dermis to the spinal cord and a dural fistula encompassing the stalk.[2] The MRI characteristics of LDM and congenital dermal sinus (CDS) have been compared in humans, in an attempt to distinguish between these two similar entities on imaging alone.[12] Clinically, a greater incidence of potentially fatal infection is seen with CDS cases in humans, where a cutaneous entry point and lumen

provides a pathway for intraspinal pathogens.[13] More immediate surgical intervention is indicated in these cases to prevent potentially catastrophic neurological complications,[2,12] such as meningitis. Significant imaging findings to diagnose LDM included greater visibility of the intrathecal tract, direct attachment to the dorsal spinal cord and dorsal tenting of the cord at the tract–cord union with associated tethering effect (seen in 83% of cases).[12] Additionally, only patients with LDM showed evidence of syringomyelia near the cord–tract union.[12] The images of our three cats were consistent with these imaging findings.

Neurological signs can vary, with up to 50% of patients documented as neurologically normal in one human study.[2] All three of our cats were significantly affected, from marked ataxia as in case 2 to non-ambulatory paraparesis in case 3. These signs predominantly relate to the spinal cord tethering effect by the neural stalk to the myofascial tissue.[2,14] Early surgery is recommended in humans, often before 9 months of age.[2] This tethering effect on the cord increases the likelihood of further neurological injury with longitudinal growth of the vertebral column in humans, and so it has been suggested that, if left untreated, it will likely worsen neurologically over time.[2] As in case 2, this may explain the young age at onset and progression of clinical signs.

It has also been hypothesised that traumatic events exacerbate clinical signs in humans,[15] causing extra tension or injury to the fibroneural stalk. There was circumstantial evidence of minor trauma in cases 1 and 2. Persistent tension, scarring or acute inflammation of the stalk secondary to trauma may contribute to the spinal cord injury and therefore a short-term course of corticosteroids could be indicated as an alternative to surgical management. However, there is a high likelihood of progressive injury and in humans prompt surgical management of spinal cord tethering is recommended. The main aim of the surgery is to remove this tethering effect, with careful resection of the stalk from the attached dura, frequently requiring a durotomy in humans.

Postoperative 'tethered cord syndrome' has also been documented in humans, secondary to scar tissue formation or the use of grafts, years after the initial surgery.[16] We propose this may have been the cause of clinical deterioration seen in case one and its re-presentation 9 months postoperatively.

There have been several reports of other vertebral malformations in cats, predominantly seen in the Manx breed. The Manx cat has been proposed as an animal model for NTDs where the absence of the tail has been documented in conjunction with sacrum agenesis, coccygeal agenesis, absence of the cauda equina and spina bifida with a meningomyelocele, similar to that described in humans.[17,18] A meningocutaneous tract, tethered spinal cord and intradural lumbosacral lipoma in an 8-month-old male neutered Manx has also been reported.[19]

The Burmese breed has also been highlighted, with several reports of cats presenting with progressive pelvic limb ataxia and skin defects.[20–22] A diagnosis of dermoid sinus was made in these cases, with one kitten being immediately euthanased,[21] and one 2-year-old male being successfully treated by a dorsal laminectomy procedure but remained persistently urinary incontinent.[22] Dermoid sinus and associated spina bifida have also been reported in dogs presenting with depressive skin lesions, abnormal hair growth and mild pelvic limb neurological deficits,[4–7,20] where surgical treatment has been successful with improvement in neurological function.[20] Histopathology of these cases described a fibrous cord with associated cystic structures containing keratinous material, hair follicles, and apocrine and sebaceous glandular tissue.[20]

There has been one notable case report of a 7-month-old male neutered cat presenting with an ambulatory T3–L3 myelopathy and a dermal lesion.[23] A dorsal laminectomy was performed and histopathology of the dermal stalk revealed the presence of neural tissue with glial fibrillary acidic protein, as we have demonstrated in cases 1 and 2. This may suggest this previous case report demonstrates features consistent with a diagnosis of LDM.

Three other dysraphic malformations have been documented in association with LDMs in humans. These are dorsal lipomas,[2] dermal sinus tracts[2] and split cord malformation,[24,25] presumably due to similar disjunction during primary neurulation. Other congenital abnormalities have been documented in animals with other forms of NTDs, including hydrocephalus,[26] syringomyelia,[26] cryptorchidism[27] and cleft palate.[28]

Conclusions

The three cats presented showed clinical signs and MRI findings consistent with a diagnosis of LDM; an NTD characterised by a fibroneural stalk extending from the dura to the overlying dermis with a visible midline skin defect. It can be distinguished from a dermoid sinus by its distinct intrathecal tract, dorsal tenting of the spinal cord at the cord–tract union and the possible presence of syringomyelia. Neurological deficits are presumed to be related to the tethering effect on the spinal cord, which may be exacerbated by additional trauma. Histopathology demonstrates the presence of a fibroneural stalk directly extending from the spinal cord to the overlying dermis. Surgical management may provide some success in terms of preventing disease progression. However, complications may include postoperative worsening of neurological signs; temporary or persistent urinary incontinence; or late recurrence of clinical signs.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval This work involved the use of non-experimental animal(s) only (owned or unowned), and followed established internationally recognised high standards ('best practice') of individual veterinary clinical patient care. Ethical approval from a committee was therefore not necessarily required.

Informed consent Informed consent (either verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work for the procedure(s) undertaken. No animals or humans are identifiable within this publication, and therefore additional informed consent for publication was not required.

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