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Intracranial meningiomas associated with cervical syringohydromyelia in a cat

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Summary

A 13-year-old, female neutered, domestic shorthair indoor cat was referred to our hospital for treatment of multiple meningiomas. A slight generalized ataxia was seen, proprioception was severely decreased on all four limbs, and menace reaction was bilaterally reduced. Pre- and postoperatively MRI examination were performed. Three supratentorial extra-axial lesions were imaged. The fourth mass was localized infratentorial extra-axial overlying the left cerebellar hemisphere. The caudoventral cerebellum had herniated caudally, approximately one cm through the foramen magnum. Cervical syringohydromyelia was found as coincidental finding. Multiple craniotomies, centered over the meningiomas were performed. Postoperative outcome two years after the surgery is excellent. The authors also reviewed the veterinary and human literature about intracranial tumors associated syringohydromyelia. Generally, the treatment of syringohydromyelia should be targeted at the pathological process, which causes the obstruction of the cerebrospinal fluid flow, and leads to syringohydromyelia formation.

Keywords: syringohydromyelia, meningioma, cat, spinal cord

Meningiom-assoziierte Syringohydromyelie bei einer Katze

Zusammenfassung

Eine dreizehnjährige, weibliche, nicht kastrierte Hauskatze wurde zur chirurgischen Therapie eines multiplen Meningeoms in der Kleintierklinik des Tierspital Bern vorgestellt. Eine leichtgradige generalisierte Ataxie wurde beobachtet, die Propriozeption war generalisiert herabgesetzt und der Drohreflex war beidseitig reduziert. Es wurden eine prä- und eine postoperative Magnetrezonanztomographie durchgeführt. Drei supratentoriale extra-axiale Raumforderungen wurden diagnostiziert. Eine vierte Masse wurde infratentorial extra-axial über der linken zerebellären Hemisphere lokalisiert. Eine Herniation des kaudoventralen Kleinhirn (Vermis) von ungefähr einem Zentimeter Länge durch das Foramen magnum wurde beobachtet. Eine zervikale Syringohydromyelie wurde als Zufallsbefund diagnostiziert. Die Meningeome wurden durch 3 Kraniotomiestellen entfernt. Zwei Jahre nach der Operation ist die Katze normal. Anhand der vorhandenen Literatur wird die tumor-assoziierte Syringohydromyelie besprochen. Die Therapie der Syringohydromyelie sollte gegen den kausalen pathologischen Prozess (z.B. intrakranieller Tumor) für die Liquorzirkulationsstörung gerichtet sein.

Schüsselwörter: Syringohydromyelie, Meningeom, Katze, Rückenmark

Introduction

Syringohydromyelia has been defined as a cystic cavitation of the spinal cord containing fluid that is identical or similar to cerebrospinal (CSF) and extracellular fluid (ECF). The cavity may be formed by a dilatation of the central canal or lie within the parenchymal substance. It may be lined by ependymal cells (hydromyelia) or by gliotic tissue (syringomyelia) (Klekamp, 2002). Syringohydromyelia can be classified into three types: (1) dilatation of the central canal that communicates directly with the fourth ventricles, also known as hydromyelia, (2) non-communicating dilatations of the central canal that arise below a syrinx-free segment of the spinal cord, and (3) extracanalicular syrinxes that originate within the parenchyma of the spinal cord and do not communicate with the central canal (Schijman, 2004). The term syringohydromyelia should not be used unless histopathological examination is provided (Klekamp, 2002). However the term syringomyelia now is generally acceptable for all clinical conditions characterized by spinal cord cavitation containing fluid identical with or closely resembling cerebrospinal fluid (Batzdorf, 2001; Rusbridge et al., 2006).
Syringohydromyelia can be associated with developmental abnormalities (Chiari malformations, Dandy-Walker syndrome, spinal dysraphism, vascular anomalies) or can be acquired due to hydrocephalus, neoplasia, arachnoiditis or trauma (Rusbridge et al., 2000). Primary syringomyelia has been reported in cats by Bone and Wilson (1982). Hydrocephalus associated with syringomyelia in one cat has also been described (Tani et al., 2001). An experimental syringomyelia in cats had been induced by kaolin (Klekamp et al., 2001) or by simulating adhesive arachnoiditis (Chang and Nagakawa, 2004). To the authors' knowledge, this is the first case report of meningioma-associated syringohydromyelia in a cat. Multiple meningiomas in cats have already been discussed (Forterre et al., 2006). Aim of this report was to describe clinical signs, features of magnetic resonance imaging and outcome in a cat with tumor-associated syringomyelia.

History and diagnostic procedure

A thirteen-year-old neutered female domestic shorthair indoor cat was referred to our hospital with history of uncoordinated gait for eleven months, especially in the pelvic limbs. An MRI examination of the brain had been performed by the referring veterinarian prior to admission. MRI revealed three intracranial masses suspected to be meningomas located on the surface of the temporal lobe and cerebellum. Treatment was initiated with dexamethason (0.25 mg/kg BW p.o. every 24h; Dexacortim, Streuli&Co AG, Switzerland) to control the vasogenic tumor-induced edema. Because of progressive lethargy and inappetence the cat was referred to our hospital for further examination and possible surgical treatment.

Clinical examination

The presented cat was irregularly vaccinated, dewormed, and FeLV negative. Moderate obesity was the only abnormality found during the general physical examination. The cat was uncooperative on examination and the evaluation remained difficult. When isolated in a quiet place, apathy could be recognized. Each manipulation led to excitation and slight aggression making the neurological evaluation difficult. A slight generalized ataxia was seen, proprioception was severely decreased on all four limbs, and the menace response was bilaterally reduced. Other cranial nerves and spinal reflexes were normal. The neuro-anatomical localisation was consistent with a multifocal intracranial lesion. Differential diagnoses included neoplasia (multiple meningiomas, lymphoma, metastasis) and/or an inflammatory process (viral, bacterial, protozoal or fungal). A complete blood count, serum biochemistry panel and thoracic radiographs were performed, which all were normal.

MRI findings

Since the first MRI scan was performed a few weeks ago, we decided to repeat the MRI scan shortly before surgery to evaluate the progression of the tumors during the elapsed time. The anesthesia was induced with a combination of medetomidin (Domitor®, Orion, Finland, 5 µg/kg BW i.v.), ketamin (Ketasol®, Graeub AG, Switzerland, 2 mg/kg BW i.v.) and propofol (Diprivan®, Astra Zeneca, USA, 4 mg/kg BW i.v.), followed by inhalation anesthesia with isoflurane (Isoflurane®, Abbott, Wiesbaden, Germany) and oxygen. MRI was performed with a 0.3 Tesla permanent magnet MRI unit (Hitachi AIRIS II; Hitachi Medical Systems; Düsseldorf, Germany). Preoperative sequences included a transverse FSE T2 and a dorsal FE 3D MPR T1 (high resolution gradient echo, plain and contrast enhanced) weighted sequence. Gadodiamide (Omniscan®; GE Health Care, München, Germany) was administered intravenously in a dosage of 0.15 mmol/kg BW.
Three supratentorial extra-axial lesions instead of the two previously reported were depicted. The most rostral lesion (2 cm, oval to triangular) was observed at the frontoparietal transition, directly dorsocaudal to the orbital fissure on the left side. The second (rounded, 1.5 cm in diameter) lay immediately caudal (parieto-temporal) to the first. A third lesion of 5 mm in diameter was detected on the right parieto-temporal side of the cerebral cortex. The latter could not be delineated in the previous MRI-examination. The fourth mass (1.5 cm in diameter) was localized infratentorial overlying the left cerebellar hemisphere. All lesions were extraaxial. The first (retroorbital) lesion was slightly hyperintense to gray matter in T2-weighted images. It was surrounded by a thin hyperintense rim in continuation with the external CSF-spaces. It had a slightly irregular surface and severely deformed the rostral left hemisphere including the lateral ventricle. The

caudally adjacent mass and the cerebellar lesion were both isointense to grey matter and surrounded by a similar rim of fluid. Both were deforming the adjacent brain tissue. They all were minimally hypointense to the adjacent brain in plain T1-weighted images. The smallest lesion – on the right side – could not be seen on any of the plain sequences. All lesions showed severe contrast uptake, the large ones (left side) more peripherically and the small right sided mass appeared more homogenous. The left thalamus showed – directly adjacent to the masses – diffusely increased signal intensity in T2-weighted images. This area did not enhance with contrast and was consistent with brain edema. The localisation can be best seen on Figure 2. The caudoventral cerebellum was herniated caudally approximately 1 cm through the foramen magnum. Beginning at the level of C2, the dorsal parts of the spinal cord showed irregularly delineated decreased signal intensity in T1 weighted sequences. In T2 weighted sequences an area of high signal intensity could be found dorsally in the region of the central canal (Fig. 1a, b). These findings were consistent with a syringohydromyelia.

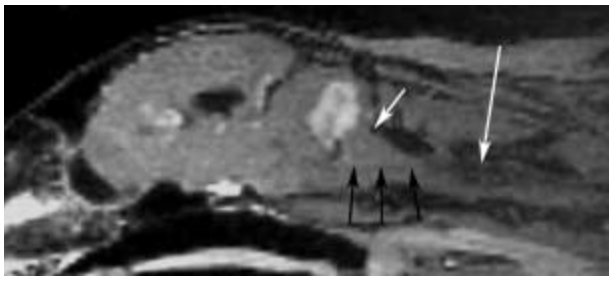

Figure 1a: Post contrast FE 3D MPR T1-weighting through the brain and cranial cervical spine. Sagittal reformation. The two rounded, inhomogenous structures in the forebrain and the cerebellum represent two of the meningiomas. The cerebellum is herniated through the foramen magnum. The black arrows indicate the herniated part with the last arrow marking the caudal tip. The small white arrow indicates the level of the foramen magnum. Caudal of the cerebellum the spinal cord is swollen and shows dorsally decreased signal intensity which is due to the syringohydromyelia (white arrow). Because of the localization of the lesion at the caudal end of the field of view (FOV) and due to the reformation process, contrast and signal to noise ratio at the lesion are low (compare to the brain, which is in the center of the FOV).

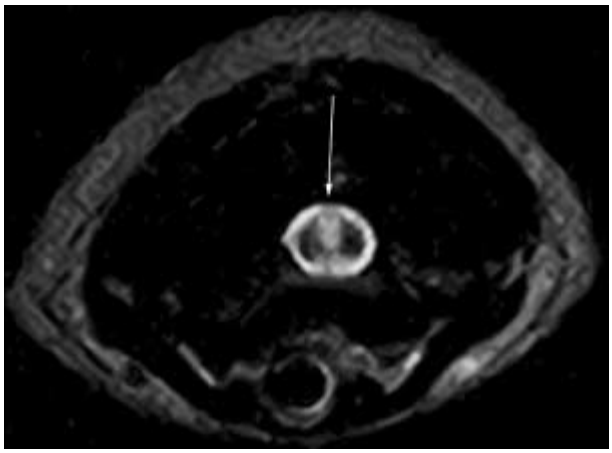

Figure 1b: Transverse T2 weighted MR-image at the level of C2. The centrodorsal portion of the spinal cord shows sharply delineated high signal intensity (white arrow), which is indicative for a widened central canal.

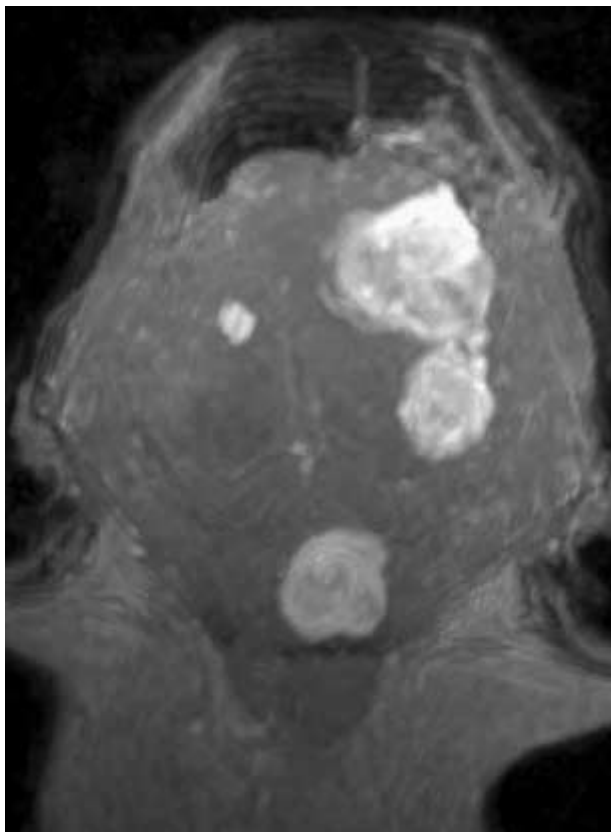

Figure 2: Thick slab (10 mm) maximum intensity projection (MIP), dorsal plane of FE 3D MPR post contrast. The image demonstrates the localisation of the 4 mass lesions, well visible through their contrast uptake (white lesions).

Anaesthesia and surgical treatment

Preoperatively intravenous premedication consisted of methylprednisolone sodium succinat (Medrate solubile®, Pfizer, Karlsruhe, Germany, 30 mg/kg BW i.v.) and mannitol (Braun-melsungen®, Melsungen, Germany, 0.5 g/kg BW i.v. over 20 minutes). Prophylactic antimicrobial therapy with cefazolin (Kefzol®, Medica, Aesch, Switzerland, 25 mg/kg BW i.v.) was also administered. Therapy with Phenobarbital (Aphenylbarbit®, Streuli, Switzerland, 5 mg/kg BW i.v.) initially and 6 hours later was started to reduced risk of postoperative epileptic seizures, and continue with 2 mg/kg BW i.v./p.o. every 12h. The anaesthesia was maintained with 1.5–2% isoflurane (Abbott®, Wiesbaden, Germany) and oxygen. Mild mechanical hyperventilation (end-tidal $C0_2$ 3–3.5%) was applied. Intraoperative analgesia was provided with lidocain (Xylesin®, Amino AG, Switzerland, 0.03 mg/kg/min constant rate infusion (CRI)) and fentanyl (Janssen®, Neuss, Germany, 0.05 mg/kg CRI). Lactated Ringer's solution (3 ml/kg/h

i.v.) was administered throughout surgery. Monitoring included electrocardiography, blood pressure (BP) and end-tidal $C0_2$ concentrations.
The cat was positioned in sternal recumbency with the head flexed at 60°, slightly elevated, taking care to maintain airway patency. The head was shaved from the eyebrows to the level of C3, scrubbed with chlorhexidine and disinfected with alcohol and povidone-iodine.
A straight skin incision extending from the frontal bone to C2 was performed. Two milliliters of cerebrospinal fluid (CSF) were released from the cisterna magna. The CSF pressure was not monitored during this procedure. Two rostrotentorial lateral craniotomies and one suboccipital craniotomy centered over the meningiomas were performed. Small bleeding vessels were cauterized with bipolar microforceps. Larger venous structures were obliterated with methylcellulose. Two forebrain superficial tumors were removed through en-bloc resection. The two other meningiomas were first internally decompressed through a single-window technique: the visible surface of the tumour was fenestrated and the centre of the tumor enucleated using tumour forceps through this window. The tumor margins could then be carefully dissected away from the brain tissue favoured by a good cleavage plane. After removal of all blood clots and copious lavage of the craniotomy site with saline solution, the operation site was closed by suture of the temporal muscle to its attachment, and closure of the subcutaneous tissue (Vicryl 3-0, Ethicon, Norderstedt, Germany) and the skin (Prolene 4-0, Ethicon, Norderstedt, Germany). Postoperative MRI revealed complete gross tumor removal. No signs of peritumoral edema as a consequence of iatrogenic parenchyma manipulation and removal of the space-occupying lesions were seen on T2 sequences. The cerebellar herniation appeared slightly reduced and there were no significant changes in the appearance of the syrinx (Fig 3).

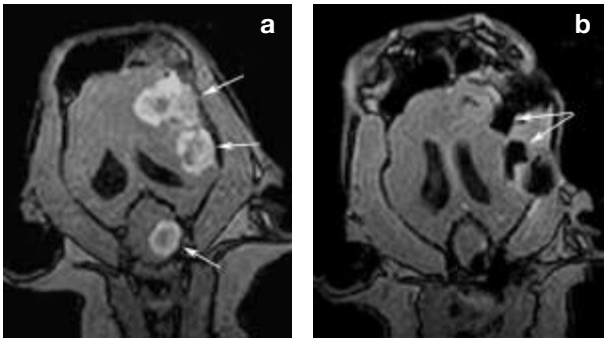

Figure 3: Prae- (a) and post- (b) surgery dorsal post contrast FE 3D MPR T1 at the level of the tentorium cerebelli. In Figure 3a the white arrows demonstrate the three meningiomas visible in this plane. The post surgery image shows slightly different orientation, so the cerebellar lesion is not clearly demonstrated. The white arrows refer to the hypointense defects containing gas and metallic debris from the drill burr. No residual neoplastic tissue can be seen.

Postoperative follow up

Postoperatively the cat was kept in an oxygen box for 12 hours. Trias, capillary refill time (CRT), blood pressure (BP), auscultation, control of neurological status and micturition were monitored. Postoperative analgesia was provided with fentanyl (Janssen®, Neuss, Germany, 0.02 mg/kg BW CRI) for the first twelve hours, and later with buprenorphine (Temgesic®, Essex, München, Germany, 0.01 mg/kg BW s.c. every 8h). Lactated Ringer's solution (2 ml/kg/h CRI) was administered for 24–36 hours. The cat received cefalexin (Cefaseptin mite®, Chassot, Ravensburg, Germany, 25 mg/kg BW p.o. every 12h) for the first post operative week , and phenobarbital (Aphenylbarbit®, Streuli, Switzerland, 2 mg/kg BW p.o. every 12h) was progressively discontinued over four weeks postoperatively. The histopathological and immunohistochemical result of all removed masses was a transitional meningioma.
Neurological deterioration was exacerbated in the early post-operative period. An involvement of the cerebellum and the brainstem was noticed, most probably consistent with the intraoperative manipulation. Moderate tetraparesis and generalised ataxia, anisocoria with dilated left pupil, decreased menace reaction on both side, decreased palpebral reflex on the right side and intension tremor were observed. Postural reactions were severely decreased. On the second day after the operation the cat was discharged home in order to reduce stress and to improve recovery. The follow-up examinations were performed by the private veterinarian. He reported a quick improvement of the neurological state within the first two weeks after discharge. Eight weeks after the operation the cat was neurologically inconspicuous. Two years after surgery no clinical signs of recurrence have yet been observed. The owners declined a follow-up MRI due to the great distance to our hospital, and because the cat appeared clinically normal.

Discussion

In humans several cases of brain tumors, most commonly meningiomas, associated with syringomyelia have been described (Fukui et al., 1993; Klekamp et al., 1995; Tachibana et al., 1995; Anegawa et al., 1997; Sheehan and Jane, 2000; Koziarski and Zielinski, 2001; Karttunen et al., 2002). In addition, also astrocytoma, glioma, medulloblastoma and epidermoid tumors have been reported with syringomyelia formation in human patients (Tachibana et al., 1995; Williams and Timperley, 1977; Agarwal et al., 1994; Klekamp et al., 1995; Tachibana et al., 1995; Sgaramella and Perria, 1996; D'Osvaldo et al., 2002). A search of the veterinary literature yielded only one report describing syringomyelia associated with a brain tumor in a dog (Da Costa et al., 2004). To the author's knowledge, no case of meningioma associated with syringohydromyelia has been published in cats.
Cerebellar tonsilar herniation and foramen magnum obstruction due to a growing intracranial mass, as reported

in human cases, has been considered as a cause for the development of cervical syringomyelia. Yamazaki et al. (1995) produced CSF flow obstruction at the foramen magnum in rats by injection of tumor cells into the occipital bone, which led to progressive epidural compression and cerebellar herniation in some animals. Cerebellar herniation was seen on MRI images also in our cat.

The literature about Arnold-Chiari malformation associated syringomyelia describes several pathogenetical theories of compression at the cranio-cervical angle (Rusbridge et al., 2000; Klekamp et al., 2001; Klekamp, 2002; Dewey et al., 2004; Schijman, 2004). Rusbridge et al. (2006) and Klekamp (2002) reviewed in details pathophysiology of syringomyelia. Intramedullary pulse pressure theory is one of the first general theories to explain the pathophysiology of syringomyelia in patients with etiologies leading to this spinal cord abnormality (Chiari malformation, posttraumatic syringomyelia, arachnoiditis, tumors in the caudal fossa or in the vertebral canal) (Rusbridge et al., 2006). This theory is based on experimental work (Greitz et al., 1999; Josephson et al., 2001; Greitz and Flodmark, 2004). The main principles of this theory are that syringomyelia is caused by repeated mechanical distension of the spinal cord and the ensuing cavitation arises from extracellular fluid originating from the high-pressure system in the microcirculation of the spinal cord and not the cerebrospinal fluid space from the low-pressure system in the subarachnoid space. Based upon the literature it seems that there is no correlation between type or location of the intracranial tumor and accompanying syringohydromyelia. Supratentorial as well as infratentorial tumors are reported to be associated with syringomyelia in humans. Also in supratentorial tumors the same pathomechanism (occluded obex due to cerebellar herniation) is expected to cause cervical syringomyelia. We believe that the infratentorial meningioma was the cause of cervical syringomyelia in our cat.

As observed in the present case, clinical signs of syringomyelia secondary to intracranial neoplasia are not overt or masked by the primary lesion. Clinical signs associated with cervical syringohydromyelia developing secondary to intracranial tumors have benn seldomly reported in veterinary and human literature (Williams and Timperley, 1977; Koziarski and Zielinski, 2001; D'Osvaldo et al., 2002). During experimental studies about kaolin-induced syringomyelia in cats (Klekamp et al., 2001), no animal developed clinical or neurophysiological evidence of neurological symptoms at any time. This is in contrast to the clinical signs seen in Cavalier King Charles Spaniels with congenital hypoplasia of the occipital bone where clinical signs of syringomyelia dominate the clinical picture. Cervical pain and involvement of the lower motor neurons in the ventral horns of the cervical spinal cord have been in this animal very well described (Rusbridge et al., 2000). If the distension of the syringohydromyelic cavity involves white matter tracks lying in spinal cord more superficially, pelvic limb ataxia and proprioception deficits are seen in the animal. We believe that the preoperative deficits presented in our cat - proprioception deficits combined with bilateral decreased menace response and apathy - are more consistent with the forebrain lesions. The cause of the generalized ataxia was regarded to be due to an involvement of sensory system rather than cerebellar deficits, because other symptoms such as hypermetria and intention tremor were not observed during the preoperative period.

Postoperative deterioration of the neurological status was compatible with intraoperative manipulation in the caudal fossa leading to temporary cerebellar and brainstem dysfunction. However, on post-operative MRI cerebellar herniation was reduced, and no other abnormalities could be detected. The clinical symptoms, if only present during peri- and post-operative period, could also be explained by the administration of opioids and Phenobarbital. The syringohydromyelic changes in our cat seem to be asymptomatic. Unfortunately, we could not perform a control MRI scan after a longer postoperative period to evaluate, if the syringohydromyelia in our cat had resolved.

Only one human patient with symptomatic syringohydromyelia of our references necessitated a syringohydromyelia targeted surgery (Koziarski and Zielinski, 2001). Asymptomatic syringohydromyelia associated with intracranial tumors predominates in human literature. Surgical excision of the tumors leads to the resortation of normal circulation of CSF flow (Da Costa et al., 2004), and radiographical regression of the cervical syringohydromyelia in the patients documented (Fukui et al., 1993; Agarwal et al., 1994; Klekamp et al., 1995; Tachibana et al., 1995; Klekamp et al., 1995; Tachibana et al., 1995; Sgaramella and Perria, 1996; Anegawa et al., 1997; Sheehan and Jane, 2000; Karttunen et al., 2002). Treatment should be targeted at the pathological process, which causes CSF flow obstruction and cord tethering and to inhibit the pathophysiological cascade before a decisive point is reached.

References

Agarwal V. K., Behari M., Ahuja G. K.: Cervical syringomyelia in a case of brainstem tumour. J. Assoc. Physic. India 1994, 42: 839.

Anegawa S., Hayashi T., Torigoe R., Iwaisako K., Higashioka H.: Cerebellopontine angle meningioma causing asymptomatic syringomyelia-case report. Neurol. Medic. Chir. 1997, 37: 624–626.

Batzdorf U.: A brief history of syringomyelia. In: Syringomyelia: current concepts in pathogenesis and management. Eds. N. Tamaki, U. Batzdorf, T. Nagashima, Springer-Verlag, Tokyo, 2001, 3–9.

Bone D. L. and Wilson R. B.: Primary syringomyelia in a kitten. J. Am. Vet. Med. Assoc. 1982, 181: 928–929.

Chang H. S. and Nakagawa H.: Theoretical analysis of the pathophysiology of syringomyelia associated with adhesive arachnoiditis. J. Neurol. Neurosurg. Psych. 2004, 75: 754–757.

Da Costa R. C., Parent J. M., Poma R., Duque M. C.: Cervical syringohydromyelia secondary to a brainstem tumor in a dog. J. Am. Vet. Med. Assoc. 2004, 225: 1061–1064.

Dewey C. W., Berg J. M., Stefanacci J. D., Barone G., Marino D. J.: Caudal occipital malformation syndrome in dogs. Comp. Cont. Educ. Pract. Vet. 2004, 26: 886–896.

D'Osvaldo D. H., Otero J. M., Mosconi J. B., Oviedo J. D.: Regression of symptomatic syringomyelia after resection of posterior fossa tumour. Acta Neurochir. 2002, 144: 385–388.

Forterre F., Tomek A., Konar M., Vandevelde M., Howard J., Jaggy A.: Multiple meningiomas: clinical, radiological, surgical, and pathological findings with outcome in four cats. J. Fel. Med. Surg. 2007, 9: 36–43.

Fukui K., Kito A., Iguchi I.: Asymptomatric syringomyelia associated with cerebellomedulary angle meningioma-case report. Neurol. Medic. Chir. 1993, 33: 833–835.

Greitz D., Ericson K., Flodmark O.: Pathogenesis and mechanics of spinal cord cysts: A new hypothesis based on magnetic resonance studies of cerebrospinal fluid dynamics. Int. J. Neurorad. 1999, 5: 61–78.

Greitz D. and Flodmark O.: Modern concepts of syringohydromyelia. Rivista di Neurorad. 2004, 17: 360–361.

Josephson A., Greitz D., Klason T., Olson L., Spenger C.: A spinal thecal sac constriction model supports the theory that induced pressure gradients in the cord cause edema and cyst formation. Neurosurg. 2001, 48: 636–646.

Karttunen A., Heikkinen E., Tuominen J., Jartti P.: Secondary syringomyelia disappearing after removal of tentorial meningioma. Acta Neurochir. 2002, 144: 741–742.

Klekamp J., Samii M., Tatagiba M., Sepehrnia A.: Syringomyelia in association with tumours of the posterios fossa. Pathophysiological considerations, based on observations on three related cases. Acta Neurochir. 1995, 137: 38–43.

Klekamp J., Völkel K., Bartels C.J., Samii M.: Disturbances of cerebrospinal fluid flow attributable to arachnoid scaring cause interstitial edema of the cat spinal cord. Neurosurg. 2001, 48: 174–186.

Klekamp J.: The pathophysiology of syringomyelia – historical overview and current concept. Acta Neurochir. 2002, 144: 649–664.

Koziarski A. and Zielinski G.: Syringomyelia associated with intracranial tumors. Case report and literature review. Neurol. Neurochir. Pol. 2001, 35: 139–152.

Rusbridge C., Greitz D., Iskandar B. J.: Syringomyelia: current concepts in pathogenesis, diagnosis, and treatment. J. Vet. Inter. Med. 2006, 20: 469–479.

Rusbridge C., MacSweeny J. E., Davies J. V., Chandler K., Fitzmaurice S. D. N., Dennis R., Capello R., Wheeler S. J.: Syringohydromyelia in Cavalier King Charles Spaniels. J. Am. Anim. Hosp. Assoc. 2000, 36: 34–41.

Sgaramella E. and Perria C.: Regression of syringomyelia after removal of a posterior fossa epidermoid tumour. Brit. J. Neurosurg. 1996, 10: 409–411.

Sheehan J. M. and Jane J. A.: Resolution of tonsillar herniation and syringomyelia after supratentorial tumor resection: case report and review of the literature. Neurosurg. 2000, 47: 233–235.

Schijman E.: History, anatomic forms, and pathogenesis of Chiari I malformations. Child's Nervous System 2004, 20: 323–328.

Tani K., Taga A., Itamoto K., Iwanaga T., Une S., Nakaichi M., Taura Y.: Hydrocephalus and syringomyelia in a cat. J. Vet. Med. Science 2001, 63: 1331–1334.

Tachibana S., Harada K., Abe T., Yamada H., Yokota A.: Syringomyelia secondary to tonsillar herniation caused by posterior fossa tumors. Surg. Neurol. 1995, 43: 471–477.

Yamazaki Y., Tachibana S., Ohta N., Yada K., Ohama E.: Experimental model of chronic tonsillar herniation associated with early stage syringomyelia. Acta Neuropathologica 1995, 90: 425–431.

Williams B., Timperly W. R.: Three cases of communication syringomyelia secondary to midbrain gliomas. J. Neurol. Neurosurg. Psych. 1977, 40: 80–88.

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