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

Schlüsselwörter: 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 meningomas 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; Dexacortin, 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 excitement 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 (Isolurane®, 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 frontotemporal 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 (retro-orbital) 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 peripherally 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.

**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 CO₂: 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...
Post-operatively 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 was clinically noticed. 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.

Postoperative follow up

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; Sgarrella and Ferria, 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. 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 syringomyelomyelia. 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 menigioma 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 syringomyelomyelia developing secondary to intracranial tumors have been 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 syringomyelomyelic 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 hyporeflexia 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 syringomyelomyelic 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 syringomyelomyelia in our cat had resolved. Only one human patient with symptomatic syringomyelomyelia of our references necessitated a syringomyelomyelic targeted surgery (Koziarski and Zielinski, 2001). Asymptomatic syringomyelomyelia associated with intracranial tumors predominates in human literature. Surgical excision of the tumors leads to the resorption of normal circulation of CSF flow (Da Costa et al., 2004), and radiographical regression of the cervical syringomyelomyelia 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


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