CASE REPORT



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Transcerebellar ventriculoperitoneal shunt for management of presumed arachnoid diverticulum in the fourth ventricle of a dog

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Abstract

Objective: To describe the approach for placement of a transcerebellar fourth ventriculoperitoneal shunt for management of presumed fourth ventricle arachnoid diverticulum and secondary obstructive hydrocephalus of a dog. To describe the outcome of this procedure.

Study design: Case report.

Animals: Male entire English springer spaniel, 3 years 9 months of age.

Methods: The dog was initially presented for management of acute, progressive, and multifocal brainstem and forebrain dysfunction. Magnetic resonance imaging revealed internal obstructive hypertensive hydrocephalus. The dog was managed via ventriculoperitoneal shunting from the left lateral ventricle and made an excellent recovery. The dog acutely deteriorated 18 months after initial discharge and follow-up magnetic resonance imaging confirmed the ventricular shunt remained in situ with normal-sized lateral ventricles but revealed a cystlike lesion within the fourth ventricle, presumed to be a fourth ventricle arachnoid diverticulum. The diverticulum was causing mass effect and resultant compression of adjacent neuroparenchyma. A second ventriculoperitoneal shunt was subsequently placed into the fourth ventricle via the caudal cranial fossa and cerebellum. This was attached to a three-way connector, to which the existing shunt (within the left lateral ventricle) was also attached, and then secured to the existing medium-pressure valve.

Results: Postoperatively, the dog immediately developed mild vestibular-cerebellar ataxia, with a marked improvement after 3 months. There were no shunt-associated complications. Long-term follow up at 40 months after the second surgical procedure revealed a normal neurological examination.

Abbreviations: CSF, cerebrospinal fluid; CT, computed tomography; FLAIR, fluid-attenuated inversion recovery; IAD, intracranial arachnoid diverticula; MRI, magnetic resonance imaging; RI, reference interval; T1W, T1-weighted; T2W, T2-weighted.

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Conclusion: Transcerebellar ventriculoperitoneal shunt placement for treatment of a presumed fourth ventricle arachnoid diverticulum was performed and was associated with a favorable long-term outcome.

1 | INTRODUCTION

Intracranial arachnoid diverticula (IAD) refers to cerebrospinal fluid (CSF)-filled cystlike lesions, typically located within the quadrigeminal cistern (supracollicular region), and variably associated with the third or fourth ventricle. Less commonly, IAD may arise within the lumen of the fourth ventricle leading to secondary complications such as internal obstructive hydrocephalus and syringohydromyelia. IAD are often congenital and it is postulated their development is linked to abnormal splitting or duplication of the arachnoid membrane during embryonic development. When occurring within the lumen of the fourth ventricle, it is suggested they arise from arachnoid tissue that is displaced secondary to ingrowing vascular mesenchyme associated with the choroid plexus.

A substantial proportion of IAD may be incidental but clinical signs, when noted, are related to their anatomic location, the impact on CSF flow, and their size, with resultant compression of adjacent neuroparenchyma. In cases with clinically significant IAD, surgical intervention is recommended. Craniotomy with cystoperitoneal shunting has been performed successfully in a number of dogs with IAD located within the quadrigeminal cistern, thus achieving sustained improvement or resolution of clinical signs. IAD that arise within the fourth ventricle pose a surgical challenge, however, and the optimum surgical technique for veterinary patients is currently unknown. Previous veterinary reports describing surgical management of such cases failed to result in a good long-term outcome. Second

This case report describes an approach to the management of presumed IAD arising in the fourth ventricle of a dog, with a successful long-term outcome. The surgical technique involved transcerebellar ventriculoperitoneal shunt placement. Although described in humans, ^{9–11} as far as the authors are aware, this has not previously been documented in the veterinary literature.

2 | MATERIALS AND METHODS

A 3 year 9 month male entire English springer spaniel was referred for neurological assessment following a 24 h history of acute onset, progressive, left-sided central vestibular syndrome. On presentation, the dog had a

markedly obtunded mental status and was nonambulatory tetraparetic with episodes of decerebrate rigidity, absent postural reactions in all limbs, absent menace response bilaterally, with pathological spontaneous nystagmus of which the direction varied depending on head position, and with reduced gag reflex. Neurological examination was consistent with a multifocal neuroanatomical localization including the brainstem, with involvement of the central vestibular system, and forebrain diffusely. Routine blood tests and urinalysis were unremarkable. Neospora caninum serology titer was negative. The dog received mannitol 0.5 g/kg IV prior to undergoing further investigation under general anesthesia. It was premedicated using butorphanol 0.1 mg/kg IV and anesthetized with lidocaine 2 mg/kg IV and propofol IV to effect. The dog was subsequently intubated and maintained with sevoflurane (initial vaporizer setting 2.5%) delivered in >95% oxygen. Perianesthetic monitoring included measurement of end tidal CO2, noninvasive blood pressure, and pulse oximetry. Magnetic resonance imaging of the brain using a 1.5 Tesla unit (Intera, Philips Medical Systems, Eindhoven, Netherlands) including postgadolinium sequences (gadoterate meglumine 0.1 mmol/kg IV, Dotarem, Guerbet, Milton Keynes, UK) subsequently revealed marked dilation of all ventricles consistent with an acquired internal obstructive hypertensive hydrocephalus, and signal void (on T2-weighted (T2W) sequences) in the region of the mesencephalic aqueduct suggestive of flow void artifact. The cerebellum was markedly compressed and displaced dorsally (Figure 1). While an obvious space-occupying lesion as a cause of the obstructive hydrocephalus was not immediately obvious obstruction at the level of the lateral apertures was considered most likely given the hydrocephalus involved all ventricles.

Due to rapid neurological deterioration, despite supportive care, it was elected to proceed with surgical intervention and the placement of a ventriculoperitoneal shunt as previously described. The dog received methadone 0.1 mg/kg IV premedication and was anesthetized with a combination of lidocaine 2 mg/kg IV, midazolam 0.25 mg/kg IV and propofol IV to effect. The dog received 20 mg/kg IV cefuroxime at the time of induction and every 90 min intraoperatively. An incision was made through skin and subcutaneous tissues from the dorsal aspect of the skull caudally along the midline to the cervical region. The exposed left temporalis muscle was elevated to expose the calvarium. A burr hole was made

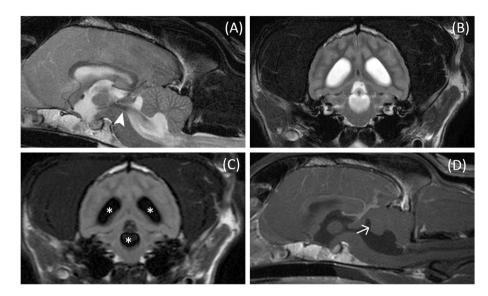


FIGURE 1 Magnetic resonance images of the head of a dog:
(A) T2-weighted (T2W) sagittal, (B) T2W transverse, (C) T2W fluid-attenuated inversion recovery (FLAIR) transverse, and (D) T1-weighted (T1W) postgadolinium sagittal images. There is marked dilation of all ventricles (white asterisk), a flow void artifact affecting the mesencephalic aqueduct (white arrowhead), and the cerebellum is markedly compressed and displaced dorsally (white arrow).

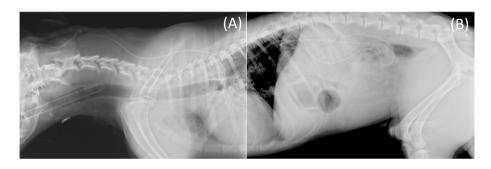


FIGURE 2 Left lateral radiographs of the thorax (A) and abdomen (B) of a dog demonstrating correct placement of a (left) lateral ventriculoperitoneal shunt.

with a pneumatic drill through the calvarium over the proposed point of insertion for the ventricular shunt. The meninges were incised and cauterized, and the shunt (Medtronic inc., Minneapolis, MN, USA) was introduced into the caudal horn of the left lateral ventricle using a stylette to a depth of \sim 2 cm. The shunt stylet was then removed, and a free flow of CSF was established before securing the shunt in place with Histoacryl tissue glue (B. Braun Medical Ltd, Sheffield, UK). CSF analysis (sampled at the time of surgery via the ventricular shunt) revealed protein(micro) 0.57 mg/dL (reference interval [RI] < 0.25), total nucleated cell count 10/microliter (RI < 5), and erythrocytes 13 200/microlitre. Cytological evaluation revealed mild macrophagic activation but the apparent pleocytosis was considered related to blood contamination. The peritoneal shunt (Medtronic Inc.) was inserted into the abdominal cavity via an incision 20-30 mm caudal to the last rib on the left side. Surrounding muscle was closed using polydioxanone (PDS Plus, Ethicon, Diegem, Belgium). The peritoneal shunt was then tunneled subcutaneously via blunt dissection using Doyen intestinal forceps cranially along the left lateral flank to enable attachment to a mediumpressure valve (Medtronic Inc.), which was then connected to the ventricular shunt. The valve was sutured to underlying soft tissues in the left lateral cervical region using polydioxanone (PDS Plus, Ethicon). Subcutaneous tissues and skin were closed using poliglecaprone 25 (Moncryl Plus, Ethicon). Correct shunt placement was confirmed with postoperative radiographs (Figure 2). The dog also received buprenorphine 0.02 mg/kg IV every 6 h alongside acetaminophen and supportive care while hospitalized in the intensive care unit. Following the surgery, the dog made a gradual but consistent recovery and was discharged 6 days postoperatively. At the time of discharge, the dog was bright, alert, and responsive with a mild right-sided head tilt, vestibular ataxia, and mildly delayed hopping and paw placement in the right thoracic and pelvic limbs. Reexamination 3 weeks later revealed a mild delay in paw placement in the right thoracic and pelvic limbs but was otherwise unremarkable.

The dog was presented again to the referral hospital 18 months later for evaluation of acute onset right-sided vestibular dysfunction and was perceived by his owner to be quieter than normal. On examination, there was a right-sided head tilt and vestibular ataxia, consistent with a lesion affecting the right vestibular system. There was also hyperesthesia on cervical palpation but, the remainder of the physical and neurological examination was unremarkable. Repeat blood work was within normal limits. The dog was subsequently anesthetized for repeat magnetic resonance imaging (MRI) of the brain (using the same anesthetic protocol and 1.5T machine as for the initial MRI). This revealed marked

FIGURE 3 Magnetic resonance images of the head of a dog: (A) T2-weighted (T2W) sagittal, (B) T2W transverse, (C) T1-weighted (T1W) postgadolinium sagittal, and (D) T2W fluid-attenuated inversion recovery (FLAIR) transverse images. There is marked ventriculomegaly affecting the fourth ventricle (white asterisk) within which there was a focal, well demarcated, regularly marginated lesion, homogeneously isointense to cerebrospinal fluid on T2W sequences (white arrowhead). Within the cranial cervical spinal cord, there is a poorly demarcated T2W intramedullary hyperintensity, which is hypointense on T1W images (relative to normal spinal cord parenchyma), consistent with syringohydromyelia (white arrow).

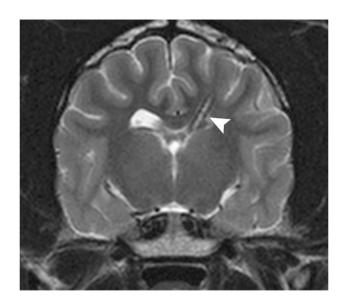


FIGURE 4 T2-weighted (T2W) transverse magnetic resonance image of the head of a dog at the level of the interventricular foramen. The tip of a ventricular catheter can be seen within the lumen of the left lateral ventricle (white arrowhead). The lateral ventricles and third ventricle are within normal limits.

ventriculomegaly affecting the fourth ventricle within which there was a focal, well demarcated, regularly marginated lesion, homogeneously isointense to CSF on T2W sequences, with complete suppression on T2W fluid-attenuated inversion recovery (FLAIR) sequences (Figure 3). There was no abnormal contrast enhancement on postgadolinium T1-weighted (T1W) sequences.

Affecting the cervical spinal cord was a focally extensive, poorly demarcated intramedullary T2W/FLAIR hyperintensity (relative to normal spinal cord parenchyma), which was most consistent with syringohydromyelia. The lateral and third ventricles were considered within normal limits and the previous ventriculoperitoneal shunt remained in situ within the left lateral ventricle (Figure 4). Based on these findings, the dog was diagnosed with presumed fourth ventricle arachnoid diverticulum and cervical syringohydromyelia.

It was elected to place a second ventriculoperitoneal shunt into the fourth ventricle via the cerebellum and caudal cranial fossa. Computed tomography (CT) imaging of the head using a 320 slice multi detector row unit (Aquilion One Genesis Edition (320 slice), Canon Medical Systems, Otawara, Japan) was performed preoperatively to enable surgical planning and accurate determination of bony anatomical landmarks. The dog subsequently received methadone 0.2 mg/kg IV premedication and was anesthetized with a combination of lidocaine 1 mg/kg IV, midazolam 0.3 mg/kg IV, and propofol IV to effect. It was intubated and anesthesia maintained with sevoflurane (initial vaporizer setting 2.5%) delivered in >95% oxygen. The dog received 20 mg/kg IV cefuroxime at the time of induction and every 90 minutes intraoperatively. An incision was made through skin and subcutaneous tissues from the dorsal aspect of the skull caudally along the midline to the cervical region. The exposed left temporalis muscle was elevated to expose the occipital bone and C1 vertebra. A burr hole was made with a pneumatic drill through the

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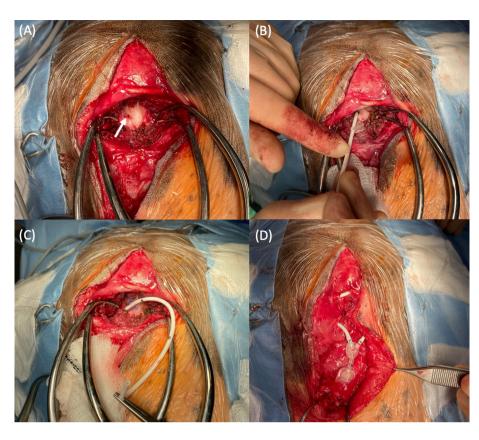
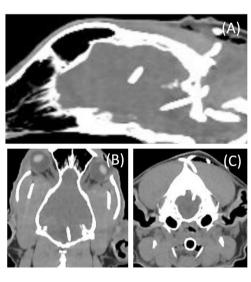


FIGURE 5 Intraoperative images. A burr hole was made through the calvarium over the point of insertion of the ventricular shunt (2 cm ventral to the nuchal crest and 1 cm lateral to the left of the midline) (A). The shunt was introduced into the fourth ventricle using a stylette (B), and was secured to the calvarium (C), before attaching to a three-way connector and medium-pressure valve (D).



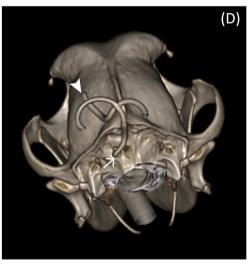


FIGURE 6 Computed tomography (CT) images of the head of a dog in (A) sagittal, (B) dorsal, and (C) transverse orientation in soft tissue image reconstruction algorithm documenting correct placement of a transcerebellar catheter within the fourth ventricle. Caudal view of the skull in 3D CT image reconstruction (D) reveals entry points of both ventricular shunts within the left lateral (white arrowhead) and fourth ventricle (white arrow).

calvarium over the planned point of insertion of the ventricular shunt (2.6 cm ventral to the nuchal crest and 1 cm to the left of the midline, based on the CT measurements) (Figure 5A). The dura was incised and cauterized as necessary, and the shunt (Medtronic Inc.) was introduced into the fourth ventricle through the cerebellum using a stylette to a depth of 2.2 cm and at an angle of 50° in a rostroventral direction (Figure 5B). The shunt stylet was removed, and a free flow of CSF was established before securing the shunt in place with Histoacryl tissue glue (B. Braun Medical Ltd) (Figure 5C). The CSF was not

analyzed, based on the low index of suspicion for inflammatory or neoplastic disease, and financial constraints of the owner. Similarly, no attempt was made to biopsy the lesion in the fourth ventricle (presumed IAD). The shunt was then attached to a three-way connector (Medtronic Inc.) to which the existing shunt (within the left lateral ventricle) was also attached (Figure 5D). This was then secured to the existing medium-pressure valve. Muscles were closed with polydioxanone (PDS Plus, Ethicon), and subcutaneous tissue and skin were closed with polyglecaprone 25 (Moncryl Plus, Ethicon). Postoperative CT

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imaging (Aquilion One Genesis Edition) confirmed correct placement of the shunt (Figure 6).

3 **RESULTS**

The dog recovered without anesthetic complications and received buprenorphine 0.02 mg/kg IV every 6 h, gabapentin 10 mg/kg orally every 8 h, and prednisolone 0.5 mg/kg orally every 24 h. Once the dog regained ambulation, the dog was noted to exhibit a vestibularcerebellar ataxia with mild right-sided head tilt. Despite this, the dog remained otherwise neurologically stable, and was discharged 4 days postoperatively, appearing comfortable and clinically well.

Short-term follow-up evaluation at our hospital 4 weeks postoperatively revealed persistence subjective improvement of the head tilt and ataxia. Fourmonths later, a telephone update with the owner confirmed resolution of the right head tilt. The dog's gait was reportedly normal aside from a transient ataxia when excited or turning sharply.

Long-term follow up was by re-examination at our hospital 40 months after transcerebellar fourth ventriculoperitoneal shunt placement. Neurological examination was unremarkable, with complete resolution of the previous head tilt and ataxia. The owners reported that the dog continued to experience sporadic momentary uncoordinated gait when turning or moving quickly while focusing on an object such as chasing a ball; however, the owners felt the dog had an excellent quality of life and was able to engage in all activities that it did prior to the initial onset of clinical signs associated with the presumed fourth ventricle IAD.

DISCUSSION

IAD are an uncommon occurrence and those arising within the fourth ventricle have rarely been reported.^{3,5} A previous case series reported clinical signs, MRI findings, and outcome of five dogs associated with IAD arising within the fourth ventricle lumen resulting in secondary (acquired) obstructive hydrocephalus.³ Surgical management with suboccipital craniectomy, durotdiverticulectomy and marsupialization was undertaken in four of those patients. All had a poor outcome and were euthanized. The authors concluded that, despite a good initial response in three dogs, failure of the surgery was linked to ongoing pathology. In contrast, the dog of the current report had a sustained good longterm outcome. Transcerebellar fourth ventriculoperitoneal shunting has not been reported previously in

veterinary medicine, and it is possible this technique may offer a viable solution to managing fourth ventricle IAD in dogs. Although the technique must be evaluated in a larger number of dogs before making any wider conclusions, this report describes a favorable outcome in at least one.

In the human literature, several methods have been reported for management of IAD.¹³ For those located within the fourth ventricle, endoscopically guided transtentorial shunt catheters have been associated with successful long-term outcomes. 10 Transcerebellar fourth ventriculoperitoneal shunting has been reported as an alternative technique in human medicine. 9,11 Shunts of this nature are often placed with stereotactic guidance.9 Although in depth discussion regarding different techniques for shunt placement within the caudal cranial fossa is beyond the scope of this report, the authors emphasize the possible importance of accurate surgical planning for insertion of the ventricular shunt as part of the success in this case. Shunt placement was guided by preoperative CT, which allowed precise identification of external landmarks of the cranium to guide the insertion point for the shunt and calculation of insertion depth and angle. In dogs, the nuchal crest demarcates the attachment of the tentorium osseum, within which lie the transverse sinuses. This formed the dorsal boundary of a safe shunt insertion window, thus avoiding the transverse sinus and the associated risk of substantial intraoperative complications. 14 Medially, the area overlying the region of the cerebellar vermis was also avoided as medial insertion of fourth ventricle catheters has previously been associated with increased complications. 15 The occipital condyles formed the ventrolateral boundary, allowing avoidance of condylar veins, which drain the sigmoid sinuses and may be associated with substantial hemorrhage if penetrated.¹⁶ Finally, the foramen magnum formed the ventral boundary of the ventricular shunt insertion window.

Ventriculoperitoneal shunting of the lateral ventricles has been associated with a number of complications including shunt obstruction, pain, infection, disconnection, excessive shunting, and shunt kinking, among others. 12,17 The literature suggests shunt complication rates in dogs and cats of 22-29%. 12,18 In human medicine, fourth ventricle shunt placement appears to be associated with a higher rate of postoperative complications, which developed in 42% cases.¹⁵ Cranial nerve dysfunction due to direct injury to the floor of the fourth ventricle, intracystic hemorrhage, shunt malfunction, and insertion into the brainstem have all been cited as potential sequalae. 11,15 The dog in the current report did not suffer any substantial shunt complications although the development of vestibular-cerebellar ataxia postoperatively was

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likely due to iatrogenic trauma to the cerebellum. Evaluation of long-term outcome in a larger number of dogs must be undertaken to document reliably the long-term efficacy, safety, and complication rates.

Given that sampling for histopathological evaluation was not performed in our patient, definitive diagnosis was not made and instead, the diagnosis of fourth ventricle IAD was largely based on patient signalment, clinical progression, imaging, and response to treatment. Other lesions that may arise within the fourth ventricle include neoplastic lesions such as choroid plexus tumor, 19 or ependymoma.²⁰ However these would typically present with characteristic imaging findings of a mass lesion and almost certainly result in progressive clinical signs despite ventriculoperitoneal shunt placement. True cystic lesions such as epidermoid and dermoid cysts can also be considered and, less commonly, intracranial neuroenteric cysts, choroid plexus cysts, or ependymal cysts.^{2,21-23} Magnetic resonance sequences such as FLAIR, typically indicative of incomplete suppression, and diffusion weighted imaging consistent with restricted diffusion, can be useful to differentiate dermoid and epidermoid cysts from IAD. 24,25 Finally, although not yet documented in the veterinary literature, so-called isolated fourth ventricle is another possibility. This arises due to occlusion of the lateral apertures or the mesencephalic aqueduct and develops following inflammation, bacterial infection, or subsequent to intraventricular hemorrhage in patients managed with long-term interventricular shunting.^{26,27} In the present case, there is no evidence of inciting features to support development of isolated fourth ventricle. Consideration of the list of possible differential diagnoses for fourth ventricle lesions is important as treatment strategies would vary between IAD and the others.

At initial presentation, the dog's clinical signs were attributed to secondary effects of internal obstructive hypertensive hydrocephalus. Although a cause was not immediately apparent based on MRI, hydrocephalus secondary to obstruction of the lateral apertures was presumed.²⁸ In retrospect however, it is possible that the presumed fourth ventricle IAD was present but not identified on initial MRI examination. Effective drainage of CSF and resolution of the interventricular hypertension following the first surgery therefore led to dramatic clinical improvement initially,²⁹ but did not address the true underlying cause. Later neurological deterioration was likely the result of mass effect associated with the presumed fourth ventricle IAD enlarging and then compressing the adjacent cerebellum and brainstem structures. This case report therefore highlights the importance of carefully evaluating imaging studies for the possibility of fourth ventricle IAD as a cause of acquired obstructive hydrocephalus in dogs. In cases such as ours, traditional methods of treatment using ventriculoperitoneal shunt placement within the distended lateral ventricles may not lead to a sustained long-term improvement.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest related to this report.

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