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# Palliative ventriculoperitoneal shunting in dogs with obstructive hydrocephalus caused by tumors affecting the third ventricle

Rocio Orlandi[1] | Cornel G. Vasilache[1] | Isidro Mateo[1,2]

[1]Servicio de Neurología, Hospital Veterinario VETSIA, Madrid, Spain

[2]Servicio de Neurología, Hospital Clínico Veterinario, Universidad Alfonso X el Sabio, Madrid, Spain

**Correspondence**
Isidro Mateo, Hospital Clínico Veterinario, Universidad Alfonso X el Sabio, Avda. de la Universidad s/n, Villanueva de la Cañada, 28691 Madrid, Spain.
Email: isidro_mateo@yahoo.es

## Abstract

**Background:** Hypertensive or obstructive hydrocephalus is a common complication in dogs with tumors affecting the third ventricle for which few therapeutic options are available.

**Objectives:** To describe signalment, neurological status, and pre- and postsurgical findings, complications and survival time in 4 dogs with obstructive hydrocephalus caused by third ventricle tumors that were palliatively treated using ventriculoperitoneal shunting (VPS).

**Animals:** Four client-owned dogs with obstructive hydrocephalus caused by tumors affecting the third ventricle.

**Methods:** Medical records were reviewed for dogs diagnosed with third ventricular tumors. Inclusion criteria were complete medical record, advanced diagnostic imaging for review, and VPS as sole surgical treatment.

**Results:** At the time of diagnosis, all patients displayed acute onset and rapidly progressive diffuse intracranial clinical signs. On advanced imaging, all dogs had a homogeneously enhancing mass occupying or collapsing the third ventricle as well as obstructive hydrocephalus. All of the dogs underwent VPS of the most dilated lateral ventricle. In 2 of the patients, intracranial hypertension followed by normotension after VPS placement was confirmed intraoperatively by means of direct intracranial pressure monitoring. Excellent clinical improvement was observed in all dogs immediately after surgery. Three patients required a second VPS in the contralateral lateral ventricle 3, 7 and 11 months after the first surgery, all of them with renewed improvement in clinical signs.

**Conclusion and Clinical Importance:** Ventriculoperitoneal shunting is a rapid and effective treatment for patients with obstructive (hypertensive) hydrocephalus caused by tumors located within the third ventricle.

KEYWORDS

canine, cerebrospinal fluid, choroid plexus tumor, ependymoma, surgical

**Abbreviations:** CSF, cerebrospinal fluid; CT, computed tomography; FLAIR, fluid-attenuated inversion recovery; ICP, intracranial pressure; MRI, magnetic resonance imaging; VPS, ventriculoperitoneal shunting.

## 1 | INTRODUCTION

The third ventricle occasionally can be affected by neuroepithelial tumors arising from the lining epithelium of the ventricle (ependymomas) or choroid plexus (choroid plexus tumors).[1] Ependymomas rarely are reported in domestic animals, with cats more frequently affected than dogs.[2,3] Choroid plexus tumors are more common, comprising approximately 7% of all primary tumors of the central nervous system in dogs.[4] According to previous reports, the third ventricle is affected in 22% to 36% of dogs with choroid plexus tumors.[5] More rarely, the third ventricle may be collapsed, obstructed, or invaded by other tumors derived from the surrounding brain parenchyma.[6] In any case, obstruction of the interventricular foramen is a cause of obstructive and hypertensive hydrocephalus, and dilatation often affects both lateral ventricles.[7] As in humans, it is suspected that obstructive hydrocephalus (and not compression of the parenchyma itself) is often responsible for the development of acute clinical signs.[8] Therefore, the mainstay of treatment for these cases in which clinical signs are related to increased intracranial pressure (ICP) targets restoring normotension intracranially, which can be achieved by means of ventriculoperitoneal shunting (VPS).[9,10] Although VPS is described elsewhere in the veterinary literature as treatment for congenital internal hydrocephalus,[7,11,12] its utility in patients with acquired hydrocephalus is not well described.[13] Our aims were: (1) to describe the clinical presentation, neurological abnormalities, and histopathological findings in 4 dogs with tumors within the third ventricle or surrounding neural parenchyma causing obstructive hydrocephalus; (2) to evaluate computed tomography (CT) or magnetic resonance imaging (MRI) findings pre- and postsurgical intervention; and (3) to describe complications and outcomes after VPS.

## 2 | MATERIALS AND METHODS

Medical records were searched for dogs with a diagnosis of a tumor affecting the third ventricle. Inclusion criteria consisted of complete medical records until the time of death, advanced imaging of the brain (MRI or CT) with diagnosis of tumor affecting the third ventricle causing obstructive hydrocephalus, and surgical treatment that included VPS. For this study, MRI or CT features that allowed the diagnosis included presence of a single mass within or in close association with the third ventricle causing obstructive hydrocephalus. Intracranial hypertension was considered if ≥1 of the following radiographic characteristics were observed: distension of lateral ventricles, periventricular edema, transtentorial or foramen magnum herniation, flattening of the cerebral sulci, obliteration of the subarachnoid space around the dorsal convexity of the cerebral hemispheres, or any combination of these findings.[14] Imaging was performed using either a 0.2 Tesla MRI unit (Airis Mate, Hitachi, Tokio, Japan) or multislice CT scanner (Astelion 16, Toshiba, Tokio, Japan). Images were acquired before and after IV administration of 0.1 mM/kg paramagnetic (Omniscan, 287 mg gadodiamide/mL, GE healthcare, Madrid, Spain) or 600 mg/kg iodinated contrast medium (Omnipaque, 240 mg iohexol/mL, GE healthcare). Variables recorded were lesion localization (intraventricular or adjacent brain parenchyma), signal intensity or attenuation relative to cortical gray matter, signal or attenuation uniformity, mass effect (brain herniation or midline shift) and whether or not dilatation of mesencephalic aqueduct or fourth ventricle or distension of the spinal cord central canal was present. Ventriculoperitoneal shunting was performed using a commercially available shunt system (Chhabra "Slit N Spring" hydrocephalus shunt system-VP without flushing reservoir; Surgiwear, Shahjananpur, India) as described elsewhere.[7,12,13] The site of insertion of the ventricular catheter was the most dilated lateral ventricle. The shunt system included a medium pressure valve (opening pressure of 85-105 mm $H_2O$, approximately 6.2-7.7 mm Hg) in 3 dogs and a low pressure valve (opening pressure of 30-45 mm $H_2O$, approximately 2.2-3.3 mm Hg) in 1 dog. Intracranial pressure was measured intraoperatively using an invasive arterial blood pressure transducer (DTX Plus; Becton Dickinson Infusion Therapy Systems) connected to an invasive blood pressure channel in a multiparameter monitor (Carescape B650, GE healthcare, Milwaukee, Wisconsin). A 20 or 22 gauge IV catheter was inserted into the lateral ventricle through a small burr hole next to the larger ventricular shunt system catheter hole. Before insertion of the catheter, the dura and cortex were coagulated using bipolar cautery. A length of 300 mm of low-compliance tubing filled with saline was connected to the catheter as well as the pressure transducer. The pressure transducer was placed at the level of the head, connected to the multiparameter monitor and calibrated electronically, following the manufacturer's recommendations. The ICP was measured before the ventricular catheter was inserted. Head and abdominal postoperative radiographs and head CT scans were obtained to document adequate shunt placement in all patients. In cases with relapse of clinical signs, a new CT or MRI scan was obtained. When contralateral ventricular dilatation was observed and the shunted ventricle was collapsed, an obstruction of contralateral interventricular foramen was considered and new VPS was performed on the contralateral ventricle using the same technique. If the shunted ventricle appeared dilated, then obstruction or valve malfunction was considered and the system was surgically re-evaluated and changed as needed. Cephalexin (20 mg/kg PO q12h), metronidazole (10 mg/kg PO q12h), and prednisone (0.5 mg/kg PO q12h) were prescribed postoperatively for 2 weeks. Neurological examination results and response to treatment (improvement, deterioration, or static) were recorded in the medical records until death of the patients.

## 3 | RESULTS

### 3.1 | Case histories

Four dogs met the inclusion criteria. Included breeds were Brittany spaniel, French bulldog, Spanish mastiff, and Bernese mountain dog. Mean age at admission was 7 years (range, 3-12 years). Three dogs were male and 1 was female. The most common clinical signs on admission were obtundation (4 dogs), decreased menace response (3 dogs), and nonlocalized pain (2 dogs). All dogs were presented for

evaluation of acute onset (<3 days) progressive neurological signs despite symptomatic treatment with PO meloxicam (0.1 mg/kg q24h) in 2 dogs or prednisone (0.5 mg/kg q12h) in the other 2 administered by the referring clinician before admission. None of the dogs had previous history of neurological dysfunction. Neurolocalization was considered diffuse intracranial in all dogs. None of the dogs had focal neurological signs or seizures. Physical examination was considered normal in all dogs. Complete blood count, serum biochemical profile, and abdominal ultrasound examination were unremarkable in all dogs.

### 3.2 | Imaging findings

Magnetic resonance images were available for review in 2 cases and CT images in the other 2 cases. Imaging findings for all dogs were similar and consisted of a space-occupying, spherical lesion located inside (3 cases; Figure 1A,B), or immediately caudal to the third ventricle below the mesencephalic aqueduct (1 case; Figure 1C). The size of the masses ranged from 9 × 7 mm to 12 × 13 mm at their maximal diameter. All dogs had marked dilatation of the lateral ventricles; in 3 cases more prominently on the left side and in 1 case more prominently on the right side. The dog with the intra-axial lesion had marked dilatation of the third ventricle (Figure 1D). Two dogs had associated syringomyelia (Figure 1E). All dogs showed signs of periventricular edema (hyperintense signal on fluid-attenuated inversion recovery [FLAIR] MR images or blurring of the normal sharp ventricular margins) that resolved after shunting (Figure 2). All lesions showed strong homogeneous (3 cases) or ring-like (1 case) contrast enhancement (Figure 3A). Table S1 summarizes the clinical and imaging findings.

### 3.3 | Management

Despite initial management with anti-inflammatory doses of prednisolone (0.5 mg/kg q12h) and mannitol (1 g/kg IV bolus), all dogs underwent rapid neurological deterioration suspected to be caused by a progressive increase in ICP. Thus, surgical treatment with VPS was warranted to restore normal ICP. The ventricular catheter was placed in the most dilated ventricle. The 2 dogs that underwent intraoperative ICP monitoring (with initial pressures of 28 mm Hg and 31 mm Hg, respectively) had reestablishment of physiological pressure (5 mm Hg; reference range, 5-12 mm Hg) immediately after insertion of the ventricular catheter. Complete recovery and return to normal neurological status were observed in all dogs within the first 24 hours after surgery.

### 3.4 | Complications

All dogs experienced recurrence of their previous clinical signs after a median of 7 months (range 3-11 months). In all cases, imaging studies at relapse indicated minimal tumor growth (<2 mm), marked decrease

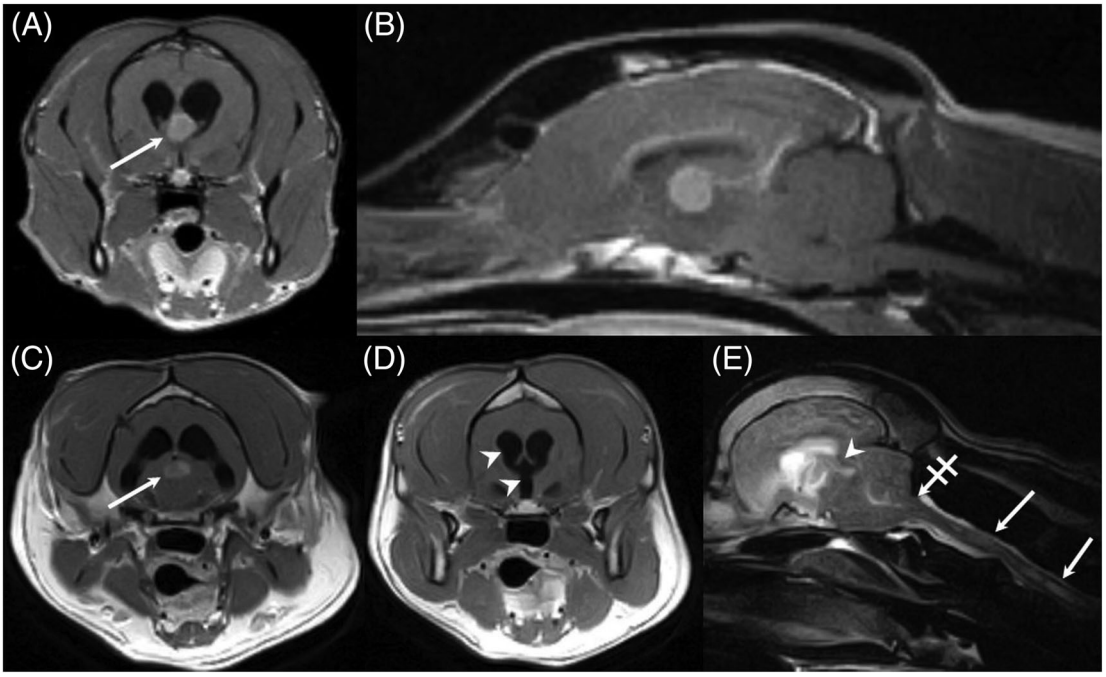



**FIGURE 1** A-D, Transverse T1W + Gd and, E, sagittal T2W magnetic resonance images. In case 1, a uniformly contrast-enhancing round mass (arrow) is seen within the third ventricle at the level of the interventricular foramina (A, B). Note an intra-axial mass immediately caudal to the interventricular foramina below the mesencephalic aqueduct (arrow in C) causing severe dilatation of lateral ventricles (arrowheads) and third ventricle, D, in case 3. Syringohydromyelia (arrows), collapsed supracolicular space (arrowhead), rostrotentorial herniation, and protrusion of the cerebellar vermis through the foramen magnum (crossed arrow) are evidenced, indicating an increased ICP in case 2 (E). ICP, intracranial pressure

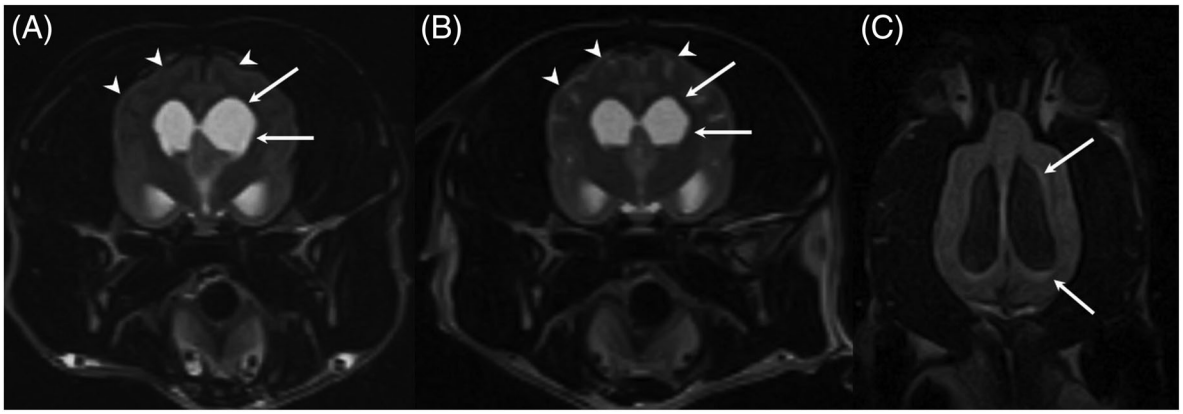



**FIGURE 2** A, Transverse T2W preoperative and, B, immediately postoperative and dorsal FLAIR magnetic resonance images of case 2. Notice how effacement of cerebral sulci (arrowheads) disappears immediately after VPS. Reduction of left lateral ventricle is also observed (arrows). C, Periventricular hyperintensity suggesting vasogenic edema (arrows). FLAIR, fluid-attenuated inversion recovery; VPS, ventriculoperitoneal shunting

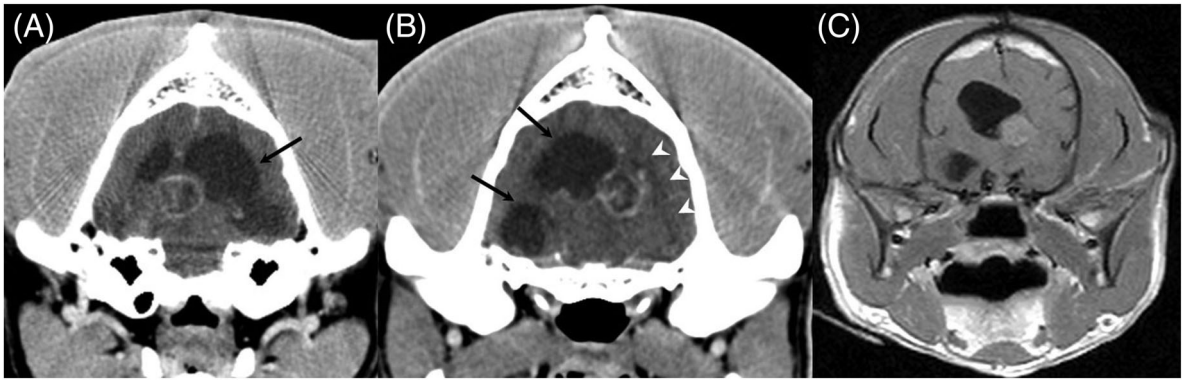
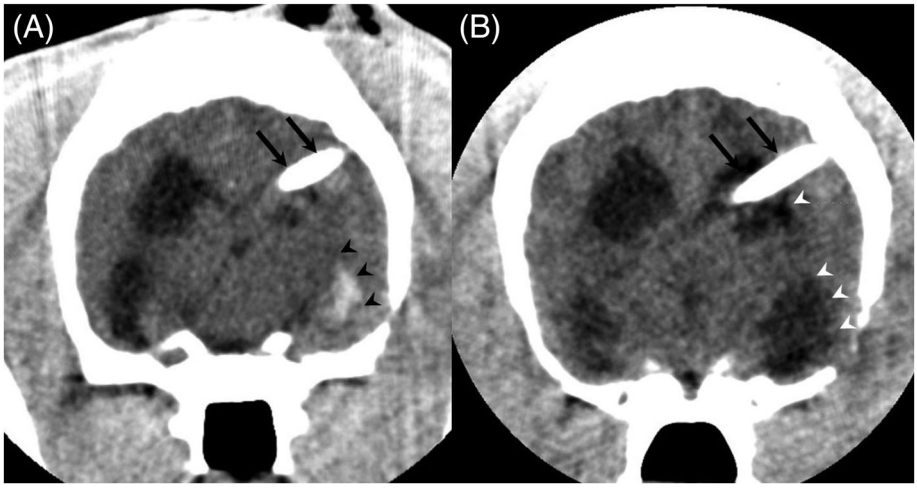



**FIGURE 3** A,B, Transverse computed tomography and, C, magnetic resonance images of case 4, showing a round ring enhancing (A, B) or homogeneously enhancing (C) mass within the third ventricle. Image (A) was obtained before surgical intervention and shows marked dilatation of left lateral ventricle (arrows). Image (B) was obtained from the same dog as in image (A), 7 months after VPS, at the time of relapse. Note similar mass size but severe dilatation of contralateral (right) ventricle (arrows). The shunted ventricle appears collapsed (arrowheads). Similar situation was observed in case 2, C, in which relapse of clinical signs was associated with collapsed shunted (left) lateral ventricle and dilatation of the contralateral ventricle. VPS, ventriculoperitoneal shunting

**FIGURE 4** Transverse postoperative computed tomography images obtained, A, immediately and, B, 2 months after surgery in case 3. Note proper ventricular catheter placement (arrows) in a collapsed left ventricle. Also note hemorrhage within the ventricle (arrowheads in A) resolved at the time of relapse of clinical signs when ventricular dilatation was present again (arrowheads in B)




in the size of the shunted ventricle and severe dilatation of the contralateral ventricle (Figure 3B,C). Patency of the previously placed VPS was considered adequate based on the marked decrease in size of the shunted ventricle. Relapse of clinical signs was considered a consequence of ventricular dilatation. Therefore, 3 of the dogs had VPS repeated in the contralateral ventricle the day after presentation,

and all of them experienced complete recovery shortly after intervention. The dog that did not undergo revision surgery was medically treated long term with prednisone (0.25 mg/kg PO q12h) with clinical signs remaining stable. This dog was suspected of having transient partial obstruction. Two dogs also had revision surgeries because of occlusion of the ventricular catheter suspected on the basis of advanced imaging (dilatation of the shunted ventricle) that was surgically confirmed. In 1 of them, a second revision surgery was needed because of migration of the peritoneal catheter. Revision surgery resulted in sustained clinical improvement in both cases. One dog experienced intraventricular hemorrhage immediately after shunt placement (Figure 4A) that spontaneously resolved (Figure 4B).

### 3.5 | Outcome

Three dogs were euthanized. Median survival time for these dogs was 14 months (range, 6-24 months). Two dogs were euthanized because of persistent collapse and obstruction of the ventricular catheter and valve. The dog with longer survival time (also the older dog) was euthanized because of cardiac failure 24 months postoperatively. The remaining dog was still alive 30 months after surgery and received prednisone treatment (0.25 mg/kg PO q12h).

Histopathological analysis was available for 2 dogs. Based on the histological findings, 1 mass was diagnosed as an ependymoma and the other as a choroid plexus carcinoma.

## 4 | DISCUSSION

Intracranial neoplasia is encountered frequently in dogs. However, its incidence in the general population is unknown, with an estimated incidence ranging from 14.5/100 000 to 3.0%.[4] In a study with a large population of dogs examined at necropsy, the prevalence of intracranial neoplasia was 4.5%.[15] Choroid plexus tumors account for approximately 7% of all primary intracranial tumors.[4] They often (75%) are associated with ventriculomegaly and obstructive hydrocephalus.[5] Survival time in dogs with choroid plexus tumors ranged from 2 to 15 months with medical treatment and up to 25 months with cytoreductive surgery.[16] Because of their relative inaccessibility, surgical resection of tumors located within the ventricles, especially the third ventricle, is challenging and carries a high rate of complications or postoperative death.[17,18] Only 1 case (a cat) in the veterinary literature is described that underwent successful surgical resection of a tumor arising from the third ventricle.[19] In this case report, the tumor (an ependymoma) was approached through the ventromedial wall of the lateral ventricle. The cat remained stable 7 months after discharge, which suggests that such tumors are amenable to surgical intervention, even when localized to the third ventricle.[19] In human medicine, primary surgical resection is the first and crucial step of standard treatment with accessible intraventricular solitary tumors.[20,21] However, in the dogs described here, rapid clinical deterioration despite aggressive medical treatment and the high mortality rate of cytoreductive surgery were reasons to search for other therapeutic options. Radiation therapy can be the primary treatment of choice if anatomic considerations and expected postoperative deficits render a central nervous system lesion inoperable or if surgical expertise is not available. Hypofractionated radiation therapy and frameless stereotactic radiosurgery have been reported to increase expected survival times in dogs with ventricular tumors.[22,23] In these studies, survival times were comparable to our results obtained using palliative VPS (1-3 years). However, direct comparison of results is not possible because tumor localization and clinical condition of the patients were not described in previous reports. Moreover, rapid deterioration in a patient with suspected increasing ICP would preclude inclusion in a radiotherapy protocol. Other treatment options such as chemotherapy are not currently considered for managements of choroid plexus tumors in dogs. Few studies have described the clinical progression of dogs with tumors affecting the third ventricle and the effect of hydrocephalus on survival time. Guidelines for the treatment of these tumors are also lacking. In humans, approximately 1% to 5% of patients with cerebral metastasis and 40% of patients with primary brain tumors suffer from hydrocephalus, usually as a result of the obstruction of cerebrospinal fluid (CSF) pathways by the tumor or dissemination of metastatic cells in the subarachnoid space resulting in CSF malabsorption.[9] Hydrocephalus can lead to severe clinical deterioration (eg, headache, nausea, vomiting, cognitive dysfunction) that may preclude systemic cancer treatment.[24] These disabling clinical signs often do not respond adequately to aggressive ICP management and VPS has been used as a palliative treatment, with up to 93% of patients achieving immediate symptomatic improvement.[9,10,24] Similarly, all dogs receiving VPS experienced immediate postoperative resolution of clinical signs. As in humans, the benefit of VPS may affect patients either with obstructive, communicating, hypertensive, or normotensive hydrocephalus.[9] In our cohort of dogs, nonspecific clinical signs (eg, abnormal mental status, unlocalizable pain, decreased menace response, impaired vision) were exclusively caused by increased ICP, as demonstrated by their immediate resolution after shunt placement, which was associated with a decrease in ventricular volume as observed in postoperative images. The decrease in ventricular size has been positively correlated with improvement of clinical signs in dogs with communication hydrocephalus.[25] As previously described, MRI features such as hyperintensity on FLAIR images and blurring of the ventricular margins were strongly associated with the presence of hypertensive hydrocephalus, suggesting the need for surgical management.[14] The ICP measurements in our patients were much higher than those found in anesthetized normal dogs (mean ICP of 7.2 mm Hg) or dogs with congenital communicating hydrocephalus (mean ICP of 8.8 mm Hg).[26,27] Long-term outcome of dogs with congenital communicating hydrocephalus treated by VPS is comparable to medical treatment with prednisone alone.[28] However, a recent study indicated that ICP > 12 mm Hg (the upper limit of the physiological range) was only seen in 5 of 23 dogs (22%) with communicating hydrocephalus. Maximal ICP in this case series was 18 mm Hg, which was observed in only

2 dogs (9%). This finding suggests that treatment with prednisone alone would be ineffective in dogs with obstructive and hypertensive hydrocephalus that have not responded to initial medical treatment and that are experiencing rapid clinical deterioration. Therefore, increasd ICP should be considered a medical emergency and VPS should be considered as a suitable treatment option. In humans, a permanent CSF diversion procedure has been recommended in patients suffering from hypertensive hydrocephalus associated with intracranial tumors, before or after surgical resection.[29] However, this approach still is controversial, because the need for shunting was highly dependent on tumor histology.[29] To our knowledge, only 1 report describes the use of VPS in the treatment of dogs with obstructive hydrocephalus caused by intracranial neoplasia.[13] Nonetheless, this article did not describe the location or type of neoplasia producing the obstruction of CSF flow.[13] Shunting should be considered cautiously in patients with intracranial neoplasia and increased ICP because of the inherent risk and possible complications of the procedure, including hemorrhage, occlusion, infection, malfunction, mechanical disconnection, overdrainage, or, more rarely, peritoneal carcinomatosis caused by seeding from the central nervous system tumor.[30] In humans, these complications are not infrequent after VPS surgery, with a complication rate of 23.8% according to 1 study.[31] Similar complication rates have been observed in dogs treated by VPS for idiopathic or acquired hydrocephalus.[13,32] Mechanical shunt failure was reported as the most common complication in 1 study and occurred in 3 of the dogs with acquired hydrocephalus.[13] They speculated that the presence of an acquired cause of hydrocephalus (intraventricular mass and inflammatory disease) might have predisposed these dogs to mechanical shunt failure. These results are similar to those of other studies of idiopathic hydrocephalus in veterinary patients and children where shunt catheter obstruction was by far the most frequent cause for shunt malfunction.[32,33] In these studies, it was hypothesized that obstruction was caused by introduction of brain parenchyma into the catheter while placing it into the ventricle or occlusion caused by blood and proteinaceous fluid from the ventricle itself.[32,33] In our population of 4 dogs, 2 developed catheter obstruction and 1 experienced migration of the peritoneal catheter. In all of the dogs in our study, VPS resulted in an initial decrease in the volume of the cerebral ventricles. However, all of the dogs developed contralateral ventricular dilatation and relapse of clinical signs within a median of 7 months, indicating need for repeated VPS in 3 dogs. The high incidence of this complication in our population of dogs raises the question of whether 2 simultaneous VPS catheters should be placed in each ventricle routinely as palliative treatment for dogs with neoplasia of the third ventricle. Other options to be considered are bilateral ventricular catheters connected to a T-shaped tube which in turn is attached to a valve or a unique ventricular catheter introduced into both lateral ventricles through the septum pellucidum. Two dogs had syringohydromyelia, but it was not considered clinically relevant because clinical signs could not be attributed to this lesion. A similar finding is reported frequently in dogs with choroid plexus tumors or other types of intracranial neoplasia.[5] Tumor mass, blood products, tumor products, metastases, ependymitis, arachnoiditis, and protein deposition all may contribute to decreased absorption and accumulation of CSF, allowing the development of syringohydromyelia. We also describe an easy, rapid, and economical method for measuring intraoperative ICP using a conventional multiparameter monitor, an invasive blood pressure transducer, and a channel. Although this method needs to be validated, it seems to be an easy and reliable way to measure ICP *in situ*. This technique would be available in most veterinary hospitals, where more sophisticated ICP monitoring such as intraparenchymal or intraventricular microsensor transducers is not available.[26,27] Our results suggest that VPS in patients with hypertensive hydrocephalus caused by tumors affecting the third ventricle potentially can achieve survival times similar to those of surgical resection or radiotherapy. All of our patients presented with acute and severe intracranial clinical signs associated with obstructive hydrocephalus that resolved immediately after VPS. Although VPS has been shown to be a life-saving procedure in an emergency setting in dogs with increased ICP caused by obstruction of the third ventricle, it should be considered cautiously because of the high rate of complications that may occur.

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## CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

## OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

## INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was need.

## HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

## ORCID

*Rocio Orlandi* https://orcid.org/0000-0002-8118-4176
*Isidro Mateo* https://orcid.org/0000-0002-2667-9459

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## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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