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CASE REPORT

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Magnetic resonance imaging findings and clinical management of suspected intracranial hypovolemia after transfrontal craniotomy in a dog

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Abstract

Objective: To report the diagnosis and clinical management of a case of suspected intracranial hypovolemia (IH) in a dog after resection of a large fronto-olfactory chordoid meningioma.

Study design: Clinical case report.

Animal: One 8-year-old border collie with forebrain neurological signs caused by a fronto-olfactory extra-axial mass diagnosed by using MRI.

Methods: The dog underwent bilateral transfrontal craniotomy for excision of the mass by using ultrasonic aspiration. Immediate postsurgical MRI revealed complete gross resection with no evidence of early-onset complications such as edema, hemorrhage, mass effect, or pneumoencephalus. However, diffuse symmetric meningeal thickening and contrast enhancement were noted. No complications were noted during surgery or while under anesthesia.

Results: Neurological deterioration was observed postoperatively. No abnormalities were detected systemically. Thus, early MRI-confirmed findings and neurological deterioration were suspected to be caused by IH. Conservative treatment consisting of bed rest, gabapentin, and intravenous theophylline was then initiated in addition to steroids, antiepileptic drugs, and antibiotics. A gradual neurological improvement was observed, and the dog was discharged completely ambulatory with moderate proprioceptive ataxia 15 days after surgery.

Conclusion: The clinical and MRI-confirmed findings reported here are consistent with IH, a well-described syndrome in man. This is the first report of a dog with MRI-confirmed findings consistent with IH describing subsequent response to medical management.

Clinical significance: Intracranial hypovolemia after craniotomy should be considered when there is neurological deterioration and characteristic MRI-confirmed findings.

1 | INTRODUCTION

Normal and abnormal computed tomographic- (CT) and MRI-confirmed findings after surgery for intracranial pathology in human medicine[1-3] and in dogs have been described in several studies and case reports.[4-6] Meningeal enhancement after craniotomy is variable depending on the extension (focal or diffuse), shape (nodular or linear), and location (pachymeningeal, leptomeningeal, or both). Meningeal enhancement is considered normal after craniotomy when characterized by a thin linear leptomeningeal and pachymeningeal involvement conforming to the margins of the resection bed or regional meninges (Table 1).[5-7]

Intracranial hypovolemia (IH) is a well-characterized syndrome in human medicine that results from abnormally low intracranial volume,[8] commonly secondary to a low cerebrospinal fluid (CSF) volume, and is associated with variable neurological signs and typical MRI-confirmed abnormalities.[9,10] Intracranial hypovolemia is usually associated with CSF leakage,[11,12] for example, secondary to over-shunting after ventriculoperitoneal shunt placement[13] or after brain[14] or spinal procedures including lumbar drainage, spinal anesthesia, or surgery.[9] Intracranial hypovolemia has not been described previously in nonexperimental dogs.

Magnetic resonance imaging-confirmed findings in human patients with IH are broadly described in the literature and can include (1) diffuse thick pachymeningeal enhancement after gadolinium administration[8-13]; (2) subtle changes of downward displacement of the brainstem and cerebellum, with third ventricle collapse[8-12]; (3) occasional focal subdural fluid collections (named *subdural hygroma*)[8-10,12]; and (4) increased crowding of the posterior fossa if IH progresses, resulting in tonsillar herniation.[9,11]

To the best of the authors' knowledge, this is the first report of suspected postoperative IH in a dog. This report describes the clinical signs and MRI-confirmed findings compatible with IH in a dog after resection of a brain tumor. A review of the pathophysiology and description of the treatment and short-term outcome is also reported.

TABLE 1 Normal and early postoperative meningeal findings according to CT/MRI after brain surgery for neoplastic and nonneoplastic conditions

Findings	Pathophysiology
1. Focal meningeal contrast enhancement at or adjacent to the site of the surgical procedure	1. Associated with the breakdown of the BBB with subsequent extrusion of contrast material into the surrounding extravascular space, neovascularization, an increased volume of blood flow to tissues resulting from a pathologic process or after surgical manipulation[4-6]
2. Remote tissue contrast enhancement	2. Related to disruption of the white matter BBB caused by brain retraction[6]
3. Remote meningeal or choroid plexus contrast enhancement	3. Common after surgery in which breaching of the lateral ventricle occurs.[2,5]
4. Focal meningeal thickening	4. Secondary to meningeal inflammation due to hemorrhage or manipulation, with subsequent meningeal thickening and eventually fibrosis[5]
5. Nodular contrast enhancement at the previous site of the tumor	5. [Related to] residual neoplastic tissue, presence of focal hemorrhage or granulation tissue[5], [or] some types of synthetic hemostatic materials[1]

Note: Other normal nonmeningeal changes observed in the early postoperative CT/MRI were small amount of air (pneumoencephalus), edema, hemorrhage (normal when blood or fluid collection is 1-3 mm in thickness), hemostatic agents, focal ischemia, and artifacts from metallic surgical instrumentation.[1,7]

Abbreviations: BBB, blood–brain barrier; CT, computed tomography.

2 | MATERIALS AND METHODS

2.1 | History

An 8-year-old 29-kg male neutered border collie was evaluated by the University of Glasgow Neurology Service for undergoing surgical debulking of a fronto-olfactory tumor diagnosed by means of MRI at the referring veterinary center. The dog had a 6-week history of acute onset generalized tonic–clonic seizures. Additional clinical signs consisted of compulsive pacing, ataxia, restlessness, vocalization, aggression toward other dogs, and loss of some learned behavior since the onset of seizures. General physical examination results were within normal limits. Neurological examination revealed a slightly disoriented mental status and compulsive pacing in the consult room. Postural reactions were decreased in the right pelvic and thoracic limbs but were normal on the left side. Segmental spinal reflexes and cranial nerve examination results were normal. No pain could be elicited by palpation of the cranium or the cervical spine. Neuroanatomical localization was consistent with left forebrain. Complete blood cell count and serum biochemistry results were within reference limits.

The MRI performed at the referring veterinary center revealed a T2-weighted and fluid-attenuated inversion

recovery heterogeneously hyperintense and T1-weighted hypointense contrast enhancing extra-axial mass in the fronto-olfactory lobes. There was severe peritumoral edema and marked mass effect and extension into the nasal cavity through the cribriform plate, more lateralized to the left (Figure 1A,B). Main differential diagnoses included esthesioneuroblastoma, meningioma, histiocytic sarcoma, nasal carcinoma, and lymphoma.

Abdominal ultrasound and thoracic CT revealed no signs of metastatic disease. Treatment with phenobarbital (4 mg/kg orally every 12 hours), levetiracetam (20 mg/kg orally every 8 hours), and prednisolone (0.5 mg/kg orally every 12 hours) had been initiated by the referring veterinarian.

2.2 | Surgery

The dog was admitted to undergo a modified bilateral transfrontal craniotomy as described by Glass et al.[15] The dog was premedicated with methadone (0.2 mg/kg IV). Prior to induction, the dog received an IV bolus of lidocaine (1 mg/kg), dexamethasone (0.2 mg/kg IV), and mannitol (0.5 g/kg), followed by constant rate infusions (CRI) of fentanyl (0.005–0.01 mg/kg/hour) and lidocaine (0.02-0.03 mg/kg/minute). Anesthesia was induced with propofol (2.3 mg/kg IV) and maintained with isoflurane and oxygen mixture.

Cefuroxime was administered at 20 mg/kg IV preoperatively and repeated every 60 minutes throughout the procedure. The dog was placed on a padded operating table in sternal recumbency and was aseptically prepared. The head was secured with a vacuum beanbag positioner with the mandible parallel to the table without pressure on the jugular veins. After a longitudinal skin incision and removal of the periosteum, the internal table of the frontal sinus was drilled to access the cranial vault. The dura mater was resected to expose a large intradural gray mass that appeared well differentiated from the normal brain parenchyma. Satisfactory resection was eventually achieved by a combination of en bloc resection and ultrasonic aspiration. The internal table of the frontal bone defect was covered with artificial dura mater (Neuro-Patch; Aesculap AG, Tuttlingen, Germany), and the external frontal bone flap was replaced and secured by using nonabsorbable monofilament suture material. Skin closure was routine. Postoperative MRI with a 1.5-T magnet (Magnetom Essenza 1.5 MRI; Siemens AG, Erlangen, Germany) was performed. A T2-weighted and T1-weighted images were acquired in the transverse, sagittal and dorsal planes. Postcontrast T1-weighted images were acquired in transverse, sagittal and dorsal planes after administration of 0.1 mmol/kg, IV bolus of gadopentetate dimeglumine (Magnevist; Bayer Schering Pharma AG, Berlin, Germany). The MRI images were consistent with gross total mass resection (Figure 1C,D) associated to

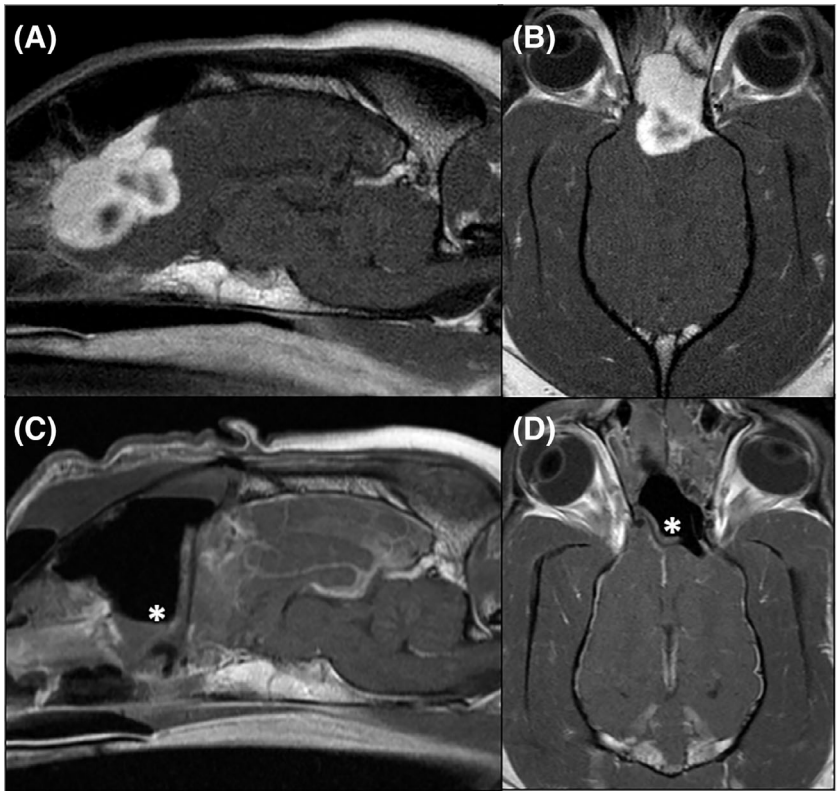

FIGURE 1 Postcontrast T1-weighted sagittal (A,C) and dorsal (B,D) images at diagnosis (A,B) and immediately after tumor resection (C,D). The macroscopic resection of the extra-axial, well-demarcated, strongly contrast enhancing, left-sided mass on the fronto-olfactory lobe can be seen. The large defect after the resection tissue is replaced by a moderate amount of gas and fluid (*)

diffuse areas of T2-weighted hyperintensity affecting the rostral portion of the remaining frontal lobes, most significant on the left side and affecting both gray and white matter, consistent with edema. Diffuse, bilateral, and symmetrical contrast enhancement and mild thickening of the pachymeninges were noted on T1-weighted postcontrast images (Figure 2). Mild and linear contrast enhancement extending into the cerebral sulci of the frontal lobes was also seen and considered consistent with bilateral frontal lobe leptomeningeal involvement. In addition, normal postsurgical changes in the caudal nasal cavity consisting of moderate amount of fluid and gas were observed (Figure 1C,D).

3 | RESULTS

3.1 | Postoperative management

There were no documented hypoxic events during surgery, and intubation and recovery procedures occurred without incident. Despite exhibiting a bright and responsive mentation immediately postoperatively and tolerating oral feeding from 24 hours postoperatively, the dog remained nonambulatory tetraparetic. This was accompanied by fluctuating mentation with occasional periods of moderate obtundation. Cranial nerve examination results and spinal reflexes were normal, but postural reaction deficits were present in all four limbs. These deficits provided evidence of a diffuse intracranial affection. An intermittent but profuse clear nasal leakage (rhinorrhea) that was compatible with CSF nasal discharge was observed (Figure 3), but it could not be confirmed. The rhinorrhea was noted especially during rehabilitation standing exercises and assisted walking. Main differential diagnoses for the potential cause of postoperative deterioration included metabolic/toxic (electrolytic disorder, hypoglycemia, systemic hypotension or hypertension, drug overdose), vascular (intracranial hemorrhage, encephalopathy related with a hypoxic event or global brain ischemia), iatrogenic (pneumoencephalus, extensive brain edema), or inflammatory/infectious disorders (considered less likely because of the early onset). Main metabolic disturbances were ruled out after results of complete blood cell count, serum biochemistry, blood gas analysis, glucose curve, systemic blood pressure and electrocardiogram monitoring, and phenobarbital serum levels measurement. Dosages of antiepileptic drugs and analgesics were also reviewed. Because the dog's neurological status remained unchanged, 4 days after surgery, plain CT (Siemens Multix Top, AGFA CR 15X CR, Munich, Germany) of the head was performed. Normal postoperative changes were observed, consisting of frontal bone craniotomy defect, endoturbinate removal, and fluid fixed on caudal nasal cavity and in areas adjacent to the surgical site. A normal attenuation of the brain was observed, with no signs of midline shift, ventricular collapse, pneumoencephalus,

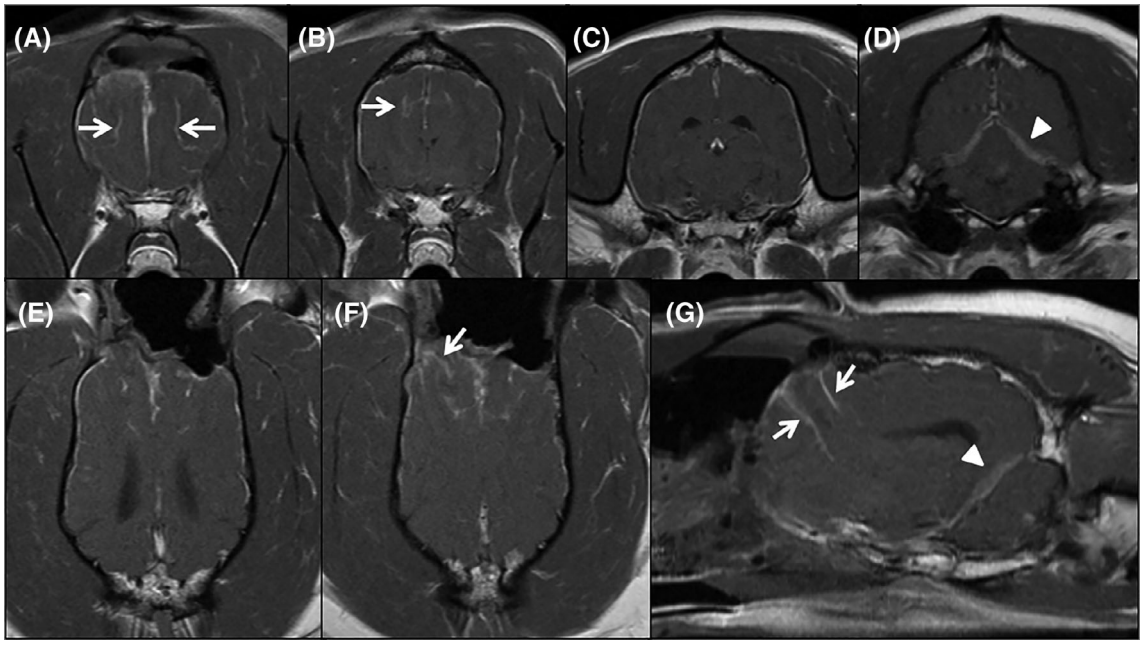

FIGURE 2 Immediately postoperative postcontrast T1-weighted transverse (A-D), dorsal (E,F), and parasagittal (G) images illustrating diffuse pachymeningeal contrast enhancement. Mild thickening of the pachymeninges is present, more evident at the tentorium (arrowheads). Contrast enhancement extends into both frontal lobes' sulci, corresponding with leptomeningeal enhancement (arrows)

hemorrhage, extensive edema, or craniotomy-bone displacement. The frontal lobes morphology was asymmetrical due to partial lobectomy on the left. Furthermore, there was moderate brain parenchyma concavity, with sagging appearance, of the frontal lobes under the skull limits on sagittal and transverse images (Figure 4); however, this was initially considered an expected finding after the large mass removal. Cerebellomedullary cistern CSF collection was attempted, but a sample could not be obtained.

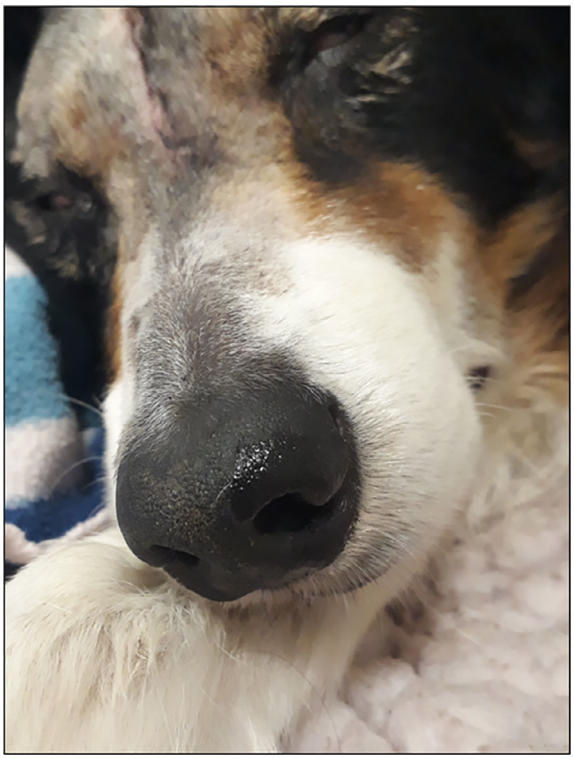

FIGURE 3 Detail of clear and unilateral nasal discharge after craniotomy. Cerebrospinal fluid rhinorrhea was suspected but could not be completely confirmed. The discharge was intermittent, becoming more profuse during the dog's mobilization for physiotherapy

Because no other systemic or intracranial disorder could be found, the alternating but persistent neurological deterioration (consistent with a diffuse intracranial localization rather than frontal lobe, which would be expected) 6 days after surgery and the diffuse meningeal changes seen on MRI were suspected to be caused by IH secondary to surgical debulking and CSF volume loss. Medical management with bed rest, gabapentin (20 mg/kg, orally every 8 hours), and theophylline (7 mg/kg orally every 24 hours) was started on day 6 after craniotomy. The rhinorrhea subsided gradually, and the dog exhibited progressive signs of improvement, being able to weight bear 10 days after surgery and ambulate with moderate proprioceptive ataxia at discharge 15 days postcraniotomy.

3.2 | Follow-up

At reexamination 2 weeks after discharge, the dog's mental status, postural reactions, and cranial nerve examination were normal, and only very mild proprioceptive ataxia was detected. No seizure activity was noted postoperatively. Histopathological analysis of the resected mass and overlying meninges, fixed in 10% buffered formalin, embedded in paraffin, sectioned at 4 μm and stained with hematoxylin and eosin, confirmed a diagnosis of grade II chordoid meningioma. After the reexamination, the dog underwent adjuvant radiotherapy, and his neurological status remains normal with no additional seizures at the time of writing, 5 months after surgery.

4 | DISCUSSION

Meningeal enhancement after brain surgery is considered normal when it is focal and linear on the perisurgical edge and can affect both pachymeninges and

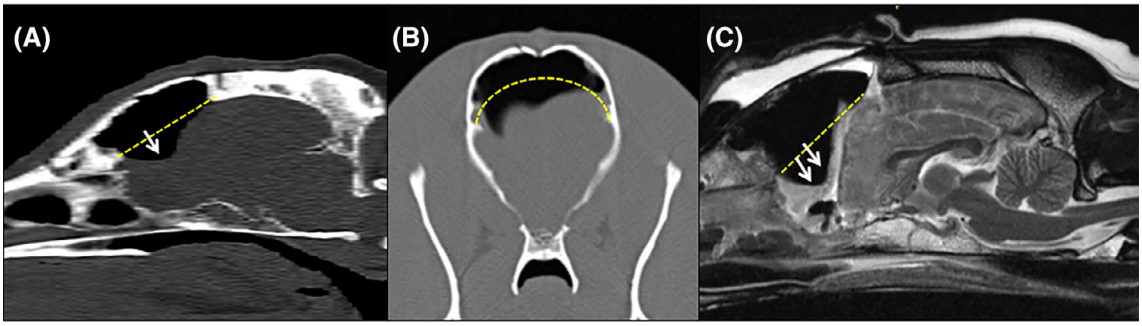

FIGURE 4 Four-day postoperative computed tomographic sagittal (A) and transverse (B) images and immediately postoperative MRI T2-weighted midsagittal (C) image illustrating the cranial defect after the large meningioma removal. The frontal cortex has a sagging appearance due to brain parenchyma displacement downward (arrows) under the normal limits of skull defect (dotted lines)

leptomeninges.[5-7] By contrast, the characteristic meningeal enhancement associated with IH is diffuse, extends symmetrically over the entire meningeal surface, and affects only the pachymeninges.[1,9-13,16] In the dog described in our case report, a diffuse pachymeningeal enhancement not limited to the surgical margins was present, compatible with the IH enhancement. However, a focal leptomeningeal enhancement restricted to the frontal lobes was also appreciated, which was associated with surgical manipulation.

Other etiologies which could cause a diffuse pachymeningeal enhancement in dogs include idiopathic hypertrophic pachymeningitis,[17] immune-mediated[18] or infectious meningitis (eg, distemper),[19] and neoplastic meningeal infiltration.[20] All these disorders were considered less likely because of the lack of diffuse pachymeningeal changes on the preoperative MRI, absence of other changes to the brain parenchyma, lack of evidence of inflammatory changes on the histopathological analysis, and favorable outcome in our dog without immunosuppressive doses of steroids.

The other change noted on both postoperative CT and MRI was the concave depression or sagging of the frontal brain parenchyma. This finding was initially considered normal because of the large volume of tissue removed. In addition, this finding is not a pathological change that has previously been reported in veterinary medicine. However, the sinking of the brain parenchyma after craniotomies/craniectomies has been described in man in relation to IH.[21]

In human medicine, clinical signs of IH could be variable and worsen during vigorous movements or while coughing or laughing.[13] The most common symptom is positional headache that worsens while standing and improves when lying down. Patients can also suffer from other associated symptoms including tinnitus, vertigo, nausea, and pain. In severe cases, the clinical course can result in deterioration of consciousness or coma.[8-10,12] Some of these signs will be unnoticed in dogs; therefore, IH would be evident only when neurological deficits are present. In our case, IH was clinically compatible because, as in man, our dog exhibited deteriorating neurological status after dynamic movements (after physiotherapy exercises) concomitant with increased nasal leak (compatible with CSF rhinorrhea), and the neurological recovery was not as expected with no other identified or suspected cause.

A definitive diagnostic test for IH does not exist for man; furthermore, some patients may have mild clinical signs or unappreciated MRI changes. Hence, IH is commonly underdiagnosed in man.[14] Therefore, the final diagnosis of IH is obtained by combining clinical signs, MRI-confirmed findings, and identification of CSF leakage.[9,11] The CSF pressure could be measured as a complementary test; however, CSF pressure is normal in most people with IH.[8,22] A good response to treatment for IH can also be considered as an additional test to support the diagnosis.[8,9,11] On the basis of the clinical history of a large intracranial mass removal, suspected CSF rhinorrhea, neurological deterioration without justified cause after brain surgery, MRI-confirmed findings, and short-term outcome, we suspected that our dog developed IH as a surgical complication. Furthermore, decreased brain volume could be suspected on the basis of sagittal MRI and CT images on which frontal lobe concavity was observed.

The pathophysiology of the clinical and imaging findings in IH is explained by the Monro-Kellie doctrine. In a closed compartment (neurocranium) containing three components (brain parenchyma, blood, and CSF), the loss of volume of one component must be compensated by a corresponding increase in the volume of the other compartments. Because the central nervous system is generally considered to be invariable, when CSF leakage happens, compensation can occur only through increase of blood volume, mainly of venous blood, because veins are more easily expansible compared with arteries.[8,11] This compensatory enlargement of meningeal veins is associated with the diffuse pachymeningeal contrast enhancement.[8,11,22] Other authors[9] have suggested that the sagging of the brain places tension on engorged dural veins, which explains the meningeal contrast enhancement and may imply a risk for vessel rupture and hematomas. Furthermore, the IH results in loss of buoyancy and downward displacement of the brain.[14] The sagging brain could explain the neurological deterioration seen in cases of IH[11,12]; nevertheless, it also could be secondary to brain perfusion changes[12] (Figure 5).

Other physiological conditions have been associated with IH, including the fact that patients of slightly greater age may have a less compliant brain. Brain expansion after long-term compression (eg, slow growing tumor) is likely to be slower in elderly patients compared with younger patients.[14]

The scientific literature also refers to IH with variable terminology, such as *intracranial hypotension* or *brain sag syndrome*,[14] reflecting the complex pathophysiology. The term *intracranial hypotension* is the term most commonly used in the human medical literature; however, it is actually a misnomer because most patients with IH exhibit normal CSF pressures. The true pathophysiology is, therefore, more appropriately described as *reduced intracranial CSF volume*; thus, *CSF hypovolemia* is preferred rather than *CSF hypotension*.[8,22] More recently, other terms such as *postoperative hypotension-associated venous congestion* or *paradoxical brain herniation* have

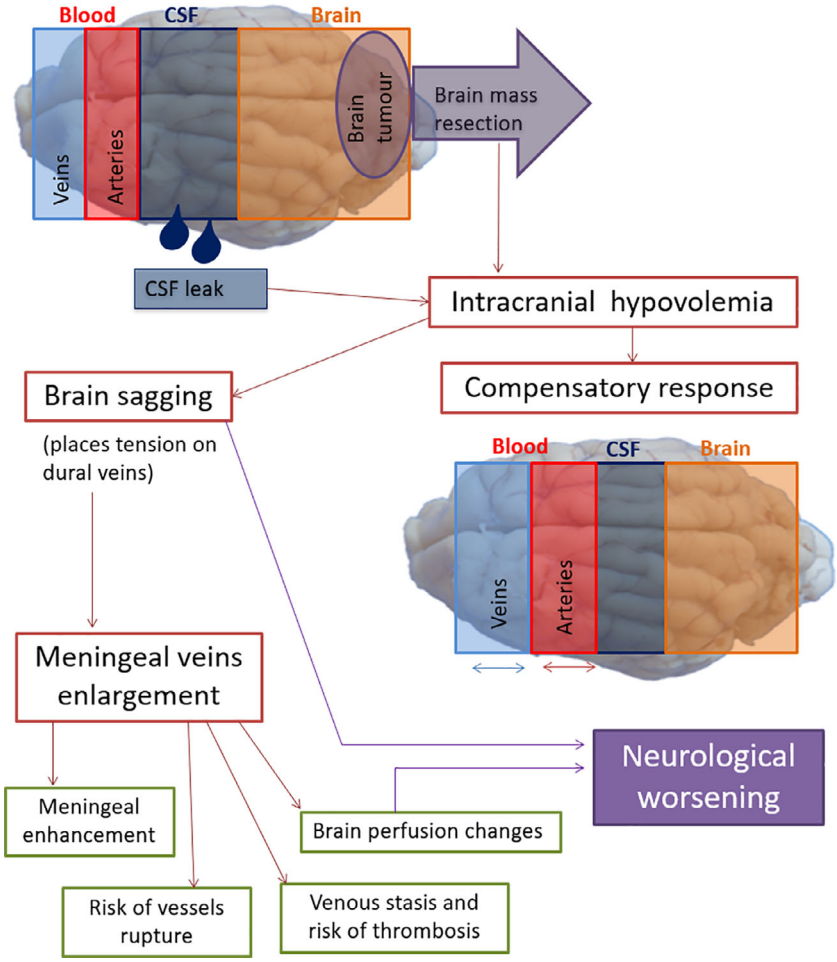

FIGURE 5 Pathophysiologic changes during intracranial hypovolemia after brain mass removal and CSF leak. CSF, cerebrospinal fluid

been used to describe presentations in which clinical and MRI descriptions are variable, but a decrease in the brain or CSF volume is common to all cases.[23]

The objective of the treatment of IH is to improve the intracranial volume and to stop the CSF leak. The initial medical treatment includes bed rest (flat posture), oral hydration,[9,12,22] head down and elevated feet posture (Trendelenburg position)[9], caffeine, corticosteroids,[8,22] and theophylline.[22,24] If medical therapy is not successful, surgical repair of the CSF leak is then indicated.[8,22] Action mechanisms of theophylline are not completely understood,[25] but it is thought to improve IH by inhibition of the compensatory vasodilatation by means of a dose dependent effect, which could also explain the effectiveness of other vasoconstrictive drugs such as caffeine.[24] The dog described in our case report experienced a remarkable improvement with medical treatment; however, the authors suspect that natural closure of dural defect helped decrease the amount of the suspected CSF loss. Because of the dog's gradual improvement with medical management, a second surgery to seal the neurocranium was not performed.

The limitations to obtain a final diagnosis in our case include the lack of CSF pressure measurement or a second postoperative MRI to assess the progress of the meningeal and parenchymal changes. However, there is no definitive test for the diagnosis of IH, and very few other entities result in a similar imaging appearance. Therefore, the presence on MRI of diffuse pachymeningeal contrast enhancement in a dog with brain volume loosening (brain tissue and/or CSF), the neurological signs previously described, and the response to therapy provided evidence to support our clinical suspicion of IH. Accordingly, IH after craniotomy should be considered in dogs with neurological deterioration and characteristic MRI-confirmed findings, especially when CSF leak is identified. Additional studies of CT- and MRI-confirmed findings in dogs after craniotomy or other procedures associated with CSF loss are required.

AUTHOR CONTRIBUTIONS

Cloquell A, DVM, MSc.: Conception of work, participation with the clinical role, data collection and interpretation, manuscript composition, accountable for all aspects of the work, revision and final approval of the submitted

manuscript. Kaczmarska A, DVM: participation with the clinical role, performed surgery, data collection, contributed to writing of manuscript and final approval of submitted manuscript. Gutierrez-Quintana R, MVZ, MVM, DECVN: participation with the clinical role and performed surgery, contributed to writing manuscript and supervision of the work, final approval of submitted manuscript. José-López R, DVM, DECVN: participation with the clinical role and performed surgery, contributed to writing of manuscript and supervision of the work, final approval of submitted manuscript.

CONFLICT OF INTEREST

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

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