Hydrocephalus in dogs: a review

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ABSTRACT: Hydrocephalus is a multifactoral disorder that was rarely diagnosed in dogs until the availability of advanced imaging techniques in veterinary practice. This article reviews recent advances in the understanding of canine hydrocephalus including pathogenesis, clinical symptoms, diagnostic methods, and treatment solutions. The advantages and disadvantages of USG, RTG, CT and MRI as advanced diagnostic methods are discussed. For now Low-field Magnetic Resonance Imaging is the most useful tool in investigating hydrocephalus. The recommended sequences for MRI are T1-weighting images Spin echo, Field echo 3D with TR 380–750 ms, TE 12–25 ms, slice thickness 1–6 mm and with an interslice gap of 0–2 mm. The evaluation of cerebral ventricular system morphology in obtained MRI scans involves measuring the height, area and volume of the brain and lateral ventricles. The results are classified as normal state if the ratio of ventricular height to the brain height is above 14%, the ratio of ventricular area to the brain area amounts to above 7%, and the ventricular to brain volume ratio is above 5%. However, there are still problems relating to inter- and intrabreed comparison among examined dogs. Treatment solutions in hydrocephalus are also discussed in this review. The medical treatment of hydrocephalus aims to decrease CSF production and is based on using acetazolamide, furosemide and prednisone. Surgical management aims to place the ventriculoperitoneal shunt for CSF flow control. Postsurgical complications are also described in this review.

Keywords: canine hydrocephalus; cerebrospinal fluid; magnetic resonance imaging; ventriculomegaly; ventricular asymmetry; medical treatment; ventriculoperitoneal shunt

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1. Introduction

Hydrocephalus describes the abnormal accumulation of cerebrospinal fluid (CSF) within the cranium. It is not a specific disease, but rather a multifactorial disorder with a variety of pathophysiological mechanisms (Thomas 1999, 2010; Hecht and Adams 2010).

2. Pathogenesis

CSF is produced at a constant rate of about 0.03 to 0.5 ml/min by the choroid plexuses in the lateral, third and fourth ventricle in an energy-dependent process (Rekate 1997; Thomas 1999, 2010). This production is independent of hydrostatic pressure within the ventricular system but is influenced by

the osmotic pressure of the blood (Thomas 2010). After production CSF circulates through the ventricular system, passing through the lateral ventricles, third ventricle, mesencephalic aqueduct, fourth ventricle and finally through the lateral apertures into the subarachnoid space of the brain and spinal cord (Thomas 1999, 2010). The CSF is absorbed by the arachnoid villi located in the venous sinuses in the subarachnoid space in a passive, energy-independent process (Thomas 1999, 2010). The major mechanism of CSF drainage involves absorption through the arachnoid villi, but alternative pathways include absorption through the arachnoid surface, capillary walls and extracranial lymphatic system (Rekate 1997; Zhao et al. 2010).

Normally the production and flow of CSF is relatively slow and the brain's visco-elastic properties ensure that there is no measurable pressure difference within the ventricular system (Rekate 1997; Thomas 2010).

The balance between the rate of formation and the rate of absorption determines the volume of CSF within the skull (Thomas 2010). The rate of CSF production is rather constant and independent of intracranial pressure. The normal intracranial pressure amounts to 7–10 cm H_2O. The CSF absorption depends on the pressure difference across the arachnoid villi which act as a valve system to keep intracranial pressure in the normal range. When intracranial pressure decreases below 7 cm H_2O, there is no CSF absorption. When this pressure increases, absorption increases in proportion to the pressure within the ventricles (Rekate 1997; Thomas 2010). However, arachnoid villi have a partial compensatory ability to reduce overproduced CSF (Zhao et al. 2010). Hydrocephalus occurs when there is resistance in the CSF passage that causes a higher pressure gradient between CSF proximal and distal to the obstruction and, moreover, alternate pathways of CSF absorption are unable to reduce the increased CSF volume within the ventricles and return it to normal range (Thomas 2010; Zhao et al. 2010). It has been reported that the disruption of CSF absorption is connected with the duration of hydrocephalus (Zhao et al. 2010).

3. Classification and causes

Hydrocephalus can be classified in various ways (Thomas 1999, 2010; Hecht and Adams 2010).

Depending on the location of the accumulated CSF, hydrocephalus is classified further as internal, in which the ventricle's enlargements are apparent, or external, with an enlarged subarachnoid space (Thomas 1999; Hecht and Adams 2010). The increased volume of CSF in hydrocephalus is rather caused by a decreased resorption (secondary to underdevelopment of arachnoid villi or inflammatory processes) than increased production of CSF (apparent in choroid plexus tumours) (Thomas 1999; Hecht and Adams 2010).

Depending on the aetiology, general classification divides hydrocephalus into congenital or acquired forms (Thomas 1999; Hecht and Adams 2010). Congenital hydrocephalus is most common in toy-breed dogs such as the Maltese, English bulldog, Pug, Pomeranian, Yorkshire terrier, Chihuahua, Lhasa apso, Toy poodle, Boston terrier and Pekingese (Vullo et al. 1997; Thomas 1999; Esteve-Ratsch et al. 2001; Ohlerth and Scharf 2007; Woo et al. 2010). The causes are diverse and include genetic factors, developmental anomalies, intrauterine or prenatal infection or bleeding in the brain (Thomas 1999). However, the enlargement of cerebral ventricles and asymmetry observed in those breeds are independent of the head shape (Kii et al. 1997, Esteve-Ratsch et al. 2001; Woo et al. 2010). Congenital hydrocephalus also occurs secondary to a wide range of nervous system anomalies, including meningomyelocele, Chiari malformation, Dandy-Walker syndrome and cerebral hypoplasia (Thomas 1999, 2010).

Secondary to decreased brain parenchyma there is compensatory hydrocephalus (ex vaccuo). A decrease in parenchyma can occur following trauma, infraction or necrosis. Hydrocephalus can also be caused by a blockage of CSF flow, so-called obstructive hydrocephalus. Obstruction within the ventricular system or at the outflow through the lateral apertures is called noncomunicating hydrocephalus because there is no communication between the ventricular system and subarachnoid space. Obstruction within the subarachnoid space or at the level of absorption in the arachnoid villi is termed communicating hydrocephalus because communication between the ventricular system and subarachnoid space persists (Thomas 1999; Hecht and Adams 2010).

Obstructions may become apparent secondary to congenital stenosis of mesencephalic aqueducts or lateral apertures (Hecht and Adams 2010). In mature dogs diseases such as tumours and intracranial inflammations frequently cause acquired obstructive hydrocephalus (Thomas 1999, 2010).

Depending on the pressure, hydrocephalus can be hypertensive, with increased pressure within the

dilated CSF-filled space, or normotensive (Thomas 1999; Hecht and Adams 2010).

Depending on the time of onset, hydrocephalus is divided into acute and chronic (Thomas 1999; Hecht and Adams 2010). Acute hydrocephalus, rather than chronic, relatively compensated normotensive hydrocephalus, is most frequently associated with inflammatory disease, periventricular oedema, subarachnoid haemorrhage and increased intraventricular pressure (Suarez-Rivera 1998; Vullo et al. 1998; Thomas 1999). Changes in ventricular pressure and volume occur in the early stage of hydrocephalus (Vullo et al. 1998).

4. Clinical signs

Morphological malformations of congenital hydrocephalus include an enlarged, dome-shaped head with persistent fontanelles and open cranial sutures. If hydrocephalus develops after the cranial sutures have closed, there is no skull malformation (Vite et al. 1997; Thomas 1999, 2010; Hecht and Adams 2010).

The excessive accumulation of CSF is responsible for neurologic deficits due to an increase in intracranial pressure and the loss of brain parenchyma (Vite et al. 1997; Vullo et al. 1998). Also, brain atrophy or developmental abnormalities can result in neurological symptoms (Vite et al. 1997). Hydrocephalic animals exhibit altered mental states ranging from depression to hyperexcitatability, disturbed consciousness, visual and auditory impairment, incoordination, circling, seizures, as well as symptoms such as dilated and fixed pupils, blindness, ventro or ventrolateral strabismus and abnormal shape of the skull. Some animals also exhibit no overt clinical signs at all (Vullo et al. 1997; Thomas 2010; Adamiak et al. 2012). There is a poor correlation between clinical signs and ventricular enlargement (Vite et al. 1997; Esteve-Ratsch et al. 2001; Thomas 2010).

It has been reported that the intensity of clinical signs is depended on the increased intracranial pressure. In obstructive hypertensive hydrocephalus clinical signs are far more apparent than in normotensive hydrocephalus (Vullo et al. 1998; Hecht and Adams 2010; Thomas 2010). Neurological deficits may progress over time, remain static or improve after a while (Thomas 2010).

Affected patients are frequently smaller than average (Vite et al. 1997; Thomas 2010; Woo et al. 2010). Vite et al. (1997) compared ventricular volume with sex, age, and body weight. The results indicated that a relationship between ventricular volume and body weight may exist and showed that body weight is inversely proportional to the percentage of intracranial volume occupied by ventricles in hydrocephalic dogs (Vite et al. 1997).

5. Advanced diagnostic methods

Diagnosis of hydrocephalus is based on identifying the clinical features and imaging the brain using advanced imaging techniques to assess ventricular size and recognise any specific prime causes (Thomas 2010).

5.1. RTG

RTG imaging may suggest hydrocephalus in puppies because of an abnormal dome-shaped head, skull bones thinness, persistent fontanelle, open cranial sutures and lack of cerebral impressions on the internal side of the skull. In adult dogs RTG imaging is superfluous because any skull malformations are readily apparent (Thomas 1999, 2010; Hecht and Adams 2010).

5.2. USG

Ultrasound of the brain can be performed only on young dogs through a persistent dorsal midline fontanelle (Brown et al. 1984; Esteve-Ratsch et al. 2001; Thomas 2010; Adamiak et al. 2012). The anatomy of the cerebral ventricular system can be easily identified, especially the lateral ventricles, third ventricle and the mesencephalic aqueduct (Brown et al. 1984). USG as a non-invasive method is helpful in assessing the range of ventriculomegaly and monitoring changes over time (Thomas 2010). In addition, the ability of performing the procedure without sedation or anaesthesia is another great advantage (Brown et al. 1984; Thomas 2010). Optimal resolution is acquired using a high-frequency probe (7–12 MHz). Normal-sized ventricles are seen as paired, slit-like anechoic structures, ventral to the longitudinal fissure, on the side of the midline. Enlarged ventricles appear either as paired anechoic regions or as a single, large anechoic structure in cases where the septum pellucidum that normally separates the lateral ventricles is absent (Thomas 2010).

5.3. CT

In small animal practice, particularly in complex anatomical areas such as the head, CT is established as a standard imaging method and can be considered as valuable tool for diagnosing neurological defects (Adamiak et al. 2012). Compared to conventional radiology, CT has higher tissue contrast. Black, white and shades of grey gradient are assigned to display the images. In hydrocephalus, the ventricular size, a mainstay for the diagnosis of internal hydrocephalus, may be accurately assessed using CT. In the case of external hydrocephalus, the hypodense CSF which is easily detected within the ventricular system is seen in enlarged cortical sulci and the subarachnoid space. Focal lesions and cortical atrophy causing obstructive hydrocephalus may be observed as well. A periventricular oedema is detected in dogs particularly with acute hydrocephalus, characterised by blurred ventricular margins and hypodense brain tissue around the ventricles (Ohlerth and Scharf 2007).

5.4. MRI

Magnetic Resonance Imaging has revolutionised the diagnosis of intracranial malformations in small animal practice (Kraft et al. 1989; Gavin 2011; MacKillop 2011).

Low-field MRI predominates in investigating hydrocephalus because it has the best diagnostic volume, is a sensitive method (increased spatial resolution and tissue contrast), and produces high quality images with minimum artefacts (Kraft et al. 1989; Kang et al. 2009; Konar and Lang 2011; Robertson 2011; Vaquero et al. 2011). Furthermore, it allows the acquiring of numerous scans of the same brain fragment in different planes and sequences. It also allows effective diagnosis of hydrocephalus and its primary causes (Adamiak et al. 2012). Obstructing masses, such as tumours, granulomas and cysts may be identified, especially in post-contrast images (Thomas 2010). The main disadvantage of this technique is the necessity for general anaesthesia which carries risks, especially for animals with cardio-pulmonary disorders (Hecht and Adams 2010).

The advantages of MRI over CT include an improved contrast gradient, acquisition of multiplane images, the availability of specialized sequences, and the use of non-ionizing radiation. A longer acquisition time and lower spatial resolution are the disadvantages of MRI compared with CT. However, CT is preferred over MRI when time under anaesthesia must be kept to a minimum (Hecht and Adams 2010).

Both CT and MRI enable accurate assessment of ventricular size, extent of cortical atrophy, and the presence of focal lesions that can be observed in hydrocephalus. Furthermore, MRI is more sensitive than CT in imaging small focal lesions, especially those in the caudal fossa (Thomas 1999, 2010). Both imaging methods are also useful in monitoring patients with ventriculoperitoneal shunts after surgical placement (Thomas 1999, 2010; Pomianowski and Adamiak 2012). Alterations in ventricular size may also be monitored and the presence of complications, for example subdural haematoma or hygroma, can be evaluated (Thomas 1999, 2010).

MRI findings in hydrocephalus include gradual narrowing of the subarachnoid space, progressive dilation of the cerebral ventricle and thinning of the cerebral cortex (Zhao et al. 2010). Abnormal CSF accumulation mainly appears hyper-intense on T2-weighting images, hypo-intense on T1-weighting images and attenuated in FLAIR (Hecht and Adams 2010; MacKillop 2011; Robertson 2011). If the CSF contains abnormal cells or proteins, in the case of inflammation or intraventricular haemorrhage, altered signal intensity may be observed. Hypertensive hydrocephalus may in conjunction with periventricular oedema be visualised as a periventricular rim of hyper-intensity surrounding the ventricular system of T2 weighting and FLAIR images (Hecht and Adams, 2010; MacKillop, 2011).

6. Challenges associated with the diagnosis of hydrocephalus

Cerebral ventricular dilation, especially of the lateral ventricles, is the most symptomatic variation in internal hydrocephalus. The diagnosis should be confirmed by MR imaging in conjunction with clinical features (Hecht and Adams 2010; Thomas 2010; Vite and Cross 2011).

However, imaging diagnoses of hydrocephalus may be challenging (Hecht and Adams 2010). It has been reported that lateral ventricle dilatation and asymmetry was observed in physiological and pathological cases (Kii et al. 1997).

Symmetric or asymmetric enlargement of the lateral ventricles is relatively common in normal adult dogs and puppies (Vullo et al. 1997; Thomas 1999). Moreover, minimal or mild ventriculomegaly

as well as asymmetry may be correlated with breed, gender or body weight among healthy dogs (Vite et al. 1997; Woo et al. 2010). Furthermore, progressive dilation of ventricles and the subarachnoid space are also anticipated with increasing age (Hecht and Adams 2010). Therefore, measurement of the ventricular volume can be useful for studying changes associated with age, degenerative disease, and ventricular obstruction (Vite et al. 1997). There are visible differences in the normal neonatal canine brain compared with the mature brain, and these can be observed using magnetic resonance imaging (Kii et al. 1998; Gross et al. 2010).

Furthermore, anatomical alterations in the ventricular system of the brain within each breed warrant investigation because various patterns of results in each may be observed (Kii et al. 1997; Vullo et al. 1997; Woo et al. 2010). According to Kii et al. (1997), Vullo et al. (1997) and Esteve-Ratsch et al. (2001), clinically insignificant moderate ventricular enlargement and minimal to mild asymmetry was common in examined groups of Beagles, Yorkshire Terriers and German shepherds. The percentage of intracranial volume occupied by the ventricle can provide a quantitative measure of the degree of ventricular dilation, which can be used to evaluate variability between individual dogs within a given breed (Vite et al. 1997; Esteve-Ratsch et al. 2001).

It is also difficult to compare ventricular volume between breeds because variations in the anatomy of the ventricular volume revealed by MR images of the canine brain may reflect large differences among dog breeds (Kii et al. 1997; Vite et al. 1997; Esteve-Ratsch et al. 2001).

On the other hand, a significant volume of CSF can accumulate in the cerebral ventricles without any overt signs of disease. Therefore, ventriculomegaly and ventricular asymmetry are common findings in asymptomatic animals and may or may not represent a clinically significant change (Kii et al. 1997; Hecht and Adams 2010). The quantification of ventricular volume can be used to make a comparison between the degree of hydrocephalus and the presence of clinical signs thought to be due to hydrocephalus (Vite et al. 1997).

All the above caveats demonstrate that a large sample group of dogs should be examined before a normal range of ventricular volume within a given breed can be determined. Moreover the range of normal percentage of intracranial volume occupied by the ventricle should be determined for various breeds to simplify the diagnosis of hydrocephalus. Diagnosis in this case will be based on quantitative measurements. Finally, a large sample size is needed before the correlation between ventricular volume and the occurrence of neurological dysfunction in dogs can be determined (Vite et al. 1997).

7. Taking measurements

Before MR scanning it is recommended to make a complete physical and neurological examination (Kii et al. 1997; Esteve-Ratsch et al. 2001; Woo et al. 2010).

The MRI examination is performed on dogs under general anaesthesia induced with ketamine at 5–15 mg/kg administrated intravenously, followed by intramuscular injection of atropine at 0.05 mg/kg and xylazine at 1 mg/kg (Kii et al. 1997; Vullo et al. 1997). Premedication with butorphanol at 0.2 mg/kg and then propofol at 5–6 mg/kg is also recommended (Esteve-Ratsch et al. 2001; Woo et al. 2010; Adamiak et al. 2012).

MR images are acquired using a magnetic resonance scanner after dogs are placed in sternal recumbency on the scanning tube (Kii et al. 1997; Woo et al. 2010).

Kii et al. (1997) recommended using T1-weighting images with a repetition time (TR) of 500 ms and echo delay time (TE) of 25 ms, slice thickness of 5.0 mm or 6.0 mm with an interslice gap of 0 mm. Vullo et al. (1997) recommend using T1-weighting images with a TR of 750 ms, TE 25 ms, slice thickness of 2.0 mm with interslice gap of 1.0 mm and intraplane resolution of 0.5 mm. Esteve-Ratsch et al. (2001) recommend using T1-weighting images Spin echo, with TR of 380–650 ms, TE 12–25 ms and Field echo 3D, with TR 23–40 ms, TE 12–15 ms, and slice thickness of 1–3 mm. Finally, Woo et al. (2010) recommend using T1-weighting images with a TR of 650 ms, TE of 25 ms and slice thickness of 4–6mm with no gap.

All imaging protocols should obtain images in transverse and dorsal positions (Kii et al. 1997; Vullo et al. 1997; Esteva-Ratsch et al. 2001; Woo et al. 2010).

Cerebral and ventricular parameters are quantified using computer image processing algorithms (Kii et al. 1997; Vullo et al. 1997; Esteve-Ratsch et al. 2001). To standardise differences in measurements among the dogs, they are expressed as percentages (Kii et al. 1997; Vite et al. 1997).

Ventricular height (Vh), brain height (Bh), ventricular area (VA), and hemisphere brain area (BA)

are measured on transverse images at the level of the interthalamic adhesion (Kii et al. 1997; Esteve-Ratsch et al. 2001; Woo et al. 2010). The ventricle to brain height ratio for both lateral ventricles is then calculated (Vh/Bh × 100%) (Kii et al. 1997; Woo et al. 2010).

The area of each lateral ventricle and hemisphere are manually outlined on the transverse MR image and automatically calculated by computer. The relative ventricle area is presented as the percentage of hemisphere occupied by the ventricle (VA/ BA × 100%) (Esteve-Ratsch et al. 2001; Woo et al. 2010).

Anatomical knowledge allows exclusion of the subarachnoid space from the calculation of ventricular volume (VV). Ventricular volume calculations include the volume of the lateral, third and fourth ventricles, the mesencephalic aqueduct and the inter-ventricular foramina (Vite et al. 1997).

Ventricular volume is calculated as a sum of the ventricular area on each transverse image multiplied by the slice thickness (Kii et al. 1997; Vite et al. 1997). A similar process is performed to determine the intracranial volume, where the contrast between the brain parenchyma and the cortical bone of the skull is used to segment the images into intractanial and extracranial tissue. The brain volume (BV) is obtained by subtracting ventricular volume from the intracranial volume (Vite et al. 1997).

As a next step, the ventricle to brain volume ratio is calculated (VV/BV × 100%) (Woo et al. 2010).

These three measurement methods, presenting ventricular alterations of hydrocephalic dogs using height, area and volume of the brain and the lateral ventricles, have statistical significance and are complementary (Woo et al. 2010).

8. Interpretation of measurements

According to Kii et al. (1997), the ratio of ventricular height to brain height is categorised as normal sized (0–14% Vh/Bh), moderately enlarged (15 to 25% Vh/Bh) or severely enlarged (> 25% Vh/Bh). Woo et al. (2010) classified Yorkshires as normal with Vh/Bh < 25% and as hydrocephalic with Vh/Bh > 25%.

According to Esteve-Ratsch et al. (2001), the ratio of the ventricle area to the hemisphere brain (VA/BA) is classified as normal range at 3.0–7.6% for Yorkshire terriers and 0.3–3.2% for German Shepherds. The VA/BA of normal Yorkshires is < 7% and of hydrocephalic Yorkshires it is > 7% according to Woo et al. (2010).

In addition, the degree of asymmetry between lateral ventricles is investigated and categorised on the basis of ventricular area (VA) (Kii et al. 1997). According to Esteve-Ratsch et al. (2001), asymmetry of the left versus right ventricle is categorised as slight (1 : 1.1–1.5), moderate (1 : 1.6–2) or extensive (> 2 : 1).

According to Woo et al. (2010) normal dogs have ventricle-to-brain volume ratios (VV/BV) of < 5% while dogs with hydrocephalus have VV/BV of > 5%.

9. Treatment of hydrocephalus

9.1. Medical treatment

Medical treatment is recommended to manage acute deterioration when surgery is not indicated and as a palliative treatment when surgery is not an option. The most frequent drugs used in therapy are acetazolamide (carbonic anhydrase inhibitor) at 10 mg/kg *per os* every 8 h, furosemide (a loop diuretic) at 1 mg/kg *per os* once daily, and glucocorticoids: prednisone at 0.25–0.5 mg/kg twice daily until features improve, followed by a reduction of the dose at weekly intervals down to 0.1 mg/kg every other day. These drugs all act to decrease CSF production. Medical therapy can provide temporary relief but does not provide long-term benefit and is associated with potential side effects such as electrolyte abnormalities (Thomas 2010).

9.2. Surgical treatment

Surgical shunting procedures using systems designed for humans are also applied to young and adult hydrocephalic dogs (Platt et al. 2012). Surgical management aims to establish a controlled CSF flow from the ventricles into the peritoneal cavity. In animals this way of treatment is recommended when medical treatment fails or hydrocephalus results in severe or progressive neurological deficits. If the prime cause is identified (eg., an intracranial tumour) shunt placement can help palliate clinical signs. It is frequently performed for congenital hydrocephalus (Shihab et al. 2011). According to Shihab et al. (2011) for ventriculoperitoneal shunt placement dogs should be positioned in right pelvic lateral recumbency with head and shoulders in sternal recumbency. A hard palate should be placed horizontal to the table. A rostrotentorial

craniotomy is used. After making the burry hole in the caudal aspect of the parietal bone, lateral to the midline, a cross-shaped incision is made in the dura mater. The ventricular catheter is then introduced using the stylet provided with the shunt system. The anchor is fitted into the burry hole and sutured to the temporalis muscle. The valve is then placed in the cervical region ventrocaudal to the left ear. As a next step, a flank leading to the abdominal cavity is made. After skin incision, muscular layers are opened, and then the peritoneum is opened with a sharp incision. The distal part of the catheter is passed into the abdominal cavity and the muscular layers are sutured closed around the shunt. A subcutaneous tunnel is made from the site of entrance into the abdominal cavity up to the level of the first cervical vertebra and the distal shunt passed back toward and attached to the valve (Shihab et al. 2011).

9.3. Postoperative outcome and complications

Complications of ventriculo-peritoneal shunting are not well described in dogs (Platt et al. 2012). Observed complications of this procedure may include infection, undershunting caused by dislocation, kinking of the catheter, leakage, valve breakage, skin necrosis, craniostenosis, microencephaly, aqueduct stenosis or obstruction, headaches or overschunting resulting in ventricular collapse and subdural haematoma (Kitagawa et al. 2005; Shihab et al. 2011; Platt et al. 2012). According to Shihab et al. (2011), pain after shunt placement occurs because of intermittent obstruction of the ventricular catheter and intracranial hypotension. The possibility of overdrainage leading to intracranial hypotension may be considered when unexpected pain occurs (Shihab et al. 2011). Moreover, overshunting may lead to the development of subdural haematomas and hydromas (Kitagawa et al. 2005; Shihab et al. 2011; Platt et al. 2012). Overshunting results from a rapid decrease in intracranial pressure caused by overdrainage of CSF, and resultant injury to the veins in the subdural space. It also results in clinical signs when the haematoma is large or acute (Shihab et al. 2011).

Ventriculoperitoneal shunt infection is the most common complication in humans and it occurs within six months after surgery (Platt et al. 2012). According to Platt et al. (2012), the situation in dogs may be analogous. *Staphylococcus* spp. are the most frequently isolated bacterial species, less frequent are *Streptococcus pneumonia*, *Escherichia coli*, *Pseudomonas* spp., *Enterobacteriaceae* and *Candida* spp. Prophylactic antibiotic therapy may help reduce postoperative infections (Shihab et al. 2011). Post-shunting infection may give rise to clinical features like circling, generalised trembling, marked proprioceptive deficits, vertical nystagmus, head tilting, and cervical spinal pain (Platt et al. 2012). Platt et al. (2012) described the magnetic resonance imaging characteristics of cerebral infection in dogs as a complication after ventriculoperitoneal shunt placement. The MR changes acquired in dogs include intraventricular debris, ependymal hyper-intensity and contrast enhancement. The debris occur hypo-intense to CSF on T2-weighted images and hyper-intense on T1-weighted images; in T2* GRE images haemorrhage is hypo-intense, similarly to the changes described in humans (Platt et al. 2012).

10. Conclusions

This review aimed to describe the constantly expanding knowledge regarding canine hydrocephalus. The diagnosis of this disorder, frequent in toy-breeds, needs to be widely investigated using advanced imaging techniques in association with diagnosis of clinical features, because clinical symptoms do not always correspond with cerebral alterations. Furthermore, research should focus on evaluating quantitatively ventricular alterations in hydrocephalic dogs to calibrate the range of ventricular parameters for each breed. Surgical management as a treatment of choice in hydrocephalic dogs is based on human medicine. Postsurgical complications need to be more widely researched and described for canines.

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