VETERINARSKI ARHIV 86 (5), 743-752, 2016

Clinical presentation and phenotypic characteristics of severe cavitatory lesions in the brains of three dogs - case report

Andrea Gudan Kurilj[1], Marija Lipar[2], Marko Hohšteter[1], and Boris Pirkić[2]

[1]*Department of Veterinary Pathology, Faculty of Veterinary Medicine, University of Zagreb, Zagreb, Croatia*

[2]*Clinic of Surgery, Orthopaedics and Ophtalmology, Faculty of Veterinary Medicine, University of Zagreb, Zagreb, Croatia*

GUDAN KURILJ, A., M. LIPAR, M. HOHŠTETER, B. PIRKIĆ: Clinical presentation and phenotypic characteristics of severe cavitatory lesions in the brains of three dogs - case report. Vet. arhiv 86, 743-752, 2016.

ABSTRACT

Marked lateral ventricular enlargement associated with atrophic cerebral cortex is described in three dogs. In case 1 (Irish terrier) and case 2 (German shepherd), hydranencephaly was diagnosed and it was characterized by the complete loss of cerebral hemispheres, leaving only about 2 mm of brain tissue that encircled fluid filled sacs. Clinically, the Irish terrier showed only sleepines and retroflexion of the head during sleeping, while the German shepherd showed no neurological or any other symptoms. In the third case (English setter), internal hydrocephalus was found, probably secondary to the severe periventricular lesions characterized by neutrophilic/mononuclear cell infiltration. This lesion was diagnosed as hydrocephalus associated with periventricular encephalitis, and this dog showed more severe neurological symptoms, such as: somnolence, opisthotonus, rigor of muscles due to seizures, poor eye-sight, stager, ataxia, and unresponsivness to vocalisation. In all cases, no infectious agent or specific cause was determined.

Key words: dog, cavitatory lesions, hydranencephaly, hydrocephalus, periventricular encephalitis

Introduction

One of the most common anomalies in the central nervous system (CNS) in all domestic animals, and especially in the dog, is hydrocephalus. Hydrocephalus is defined as a distension of the ventricular system of the brain, including or not including the arachnoid space around the brain, caused by obstruction of the normal flow of cerebrospinal fluid (CSF), increased production of CSF, or altered absorption of CSF at the arachnoid villi in the venous sinuses (JOHNSTON and TEO, 2000; REKATE, 2008). Hydrocephalus may be

*Corresponding author:

Andrea Gudan Kurilj, DVM, PhD, Dipl. ECVP, Department of Veterinary Pathology, Faculty of Veterinary Medicine, University of Zagreb, Heinzelova 55, 10000 Zagreb, Croatia, Phone: +385 1 2390 311; E-mail: agudan@vef.hr

ISSN 0372-5480
Printed in Croatia

classified by different criteria, such as: communicating with no detectable pathological lesions, or noncommunicating due to defined lesions (VANDEVELDE et al., 2012).

Another types of cavitatory brain lesion are encephaloclastic defects (porencephaly and hydranencephaly), which probably occur as a result of an acquired transplacental and destructive process in the pre-existing brain tissue (VANDEVELDE et al., 2012). In hydranencephaly, there is almost complete loss of one or both cerebral hemispheres, leaving a thin-walled membranous cavity. Porencephaly describes a less extensive defect in the cerebral parenchyma, that may not communicate with the lateral ventricle or the subarachnoid space (MAXIE and YOUSSEF, 2007; DE LAHUNTA and GLASS, 2009; VANDEVELDE et al., 2012). A special type of congenital or post-natal hydrocephalus is classified as compensatory or hydrocephalus ex vacuo, in which primary loss of brain tissue leads to the local distention of the ventricle into the lesion site of the cavitation. This form of hydrocephalus is not necessarily dependent on any change in hydrostatic pressure from obstruction/production of CSF (VANDEVELDE et al., 2012), and some authors classify hydranencephaly as this type of cavitatory lesion (ZACHARY, 2012).

This disorder may be asymptomatic or can cause minor to severe neurological disturbances, depending on the structures of brain affected and the severity of the lesions (SCHMIDT et al, 2012). Symptoms may be irritability, seizures, sleepiness, high-pitched vocalisation, tunnel vision, uncontroled eye movements, muscle spasms, slow or restricted movement, dysmetria and spontaneous reflexes (DE LAHUNTA and GLASS, 2009).

In veterinary medicine, such severe cavitatory lesions, such as hydranencephaly or hydrocephalus ex vacuo, have mostly been reported in ruminants, and they are usually caused by viruses, such as herpesviruses, orbiviruses or bunyaviruses (MAXIE and YOUSSEF, 2007). In dogs, these pathological conditions, as well as some unusual types of hydrocephalus, such as that associated with periventricular encephalitis, have been reported rarely (HIGGINS et al., 1977; CANTILE et al., 1997; DAVIES et al. 2012). Moreover, these disorders can have different clinical manifestations, which can make it difficult to set the right diagnosis. Therefore, the objective of the current case report is to describe the clinical signs, and diagnostic and pathological findings in three dogs with severe cavitatory lesions in the brain.

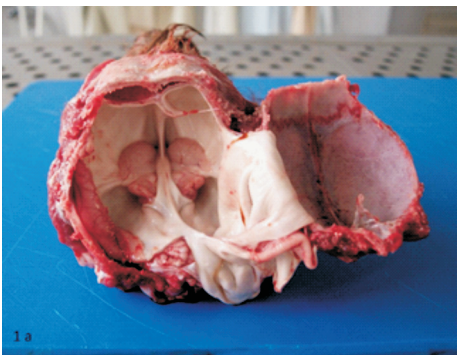
Case presentation

The first case (case 1) was a 7 month old, male Irish terrier, weighing 15 kg. The dog was vaccinated against rabies, canine distemper virus (CDV), canine adenovirus type 2 (CAV 2), canine parvovirus (CPV) and canine parainfluenzavirus (CPi). At the age of 3 months he was very sleepy, and during sleeping the owner noticed retroflexion of the head. When he was 7 months old, during walking he suddenly vomited a huge amount of foamy content and rapidly become somnolent. In addition there was abundant

salivation. The referral veterinarian assumed the puppy had taken poison when it was out of the owner's control. Hematology tests (LaseCyte, Idexx, USA) were performed and all parameters were within normal ranges. During the same day he become somnolent again, tachicardic, tachipnea, opistotonus, and rigor of all visible body muscles occured and the dog suddenly died and was submitted for necropsy at the Department of Veterinary Pathology. Another dog from the same litter, who lived together with this dog, did not show any signs of disease.

The second case (case 2) was a 2 year old male German shepherd, weighing 35 kg. According to the owner's statement, this dog never showed any signs of illness or behavioral changes. The dog was found dead in the garden two hours after a walk with the owner. Due to the sudden death, the dog was submitted for necropsy. Examining the vaccination book it was determined that the dog had also been regularly vaccinated against canine infectious diseases and rabies, like the dog in case 1.

The third case (case 3) was a female English setter puppy, four months old, weighing 8.7 kg. This dog was vaccinated against canine contagious viral diseases and rabies. At the age of 3 months the owner noticed weakness of the hind limbs, stagger and that it was bumping into objects, it had ataxia, was irresponsive to vocalisation, and avoided taking food and water. The referral veterinarian did a blood test related to tickborne diseases, which was negative, and applied 0.9% saline solution and midazolam intravenously. Finally, the dog was presented at the Veterinary Faculty, due to seizures, languor and poor eye-sight. Clinical examination revealed proper grading, the mucous membranes were glowing, and the lymph nodes were normal. Depression, hypermetric gait, a wide posture, inclined to the left side, and tremor were noted. Proprioceptive reaction in the left front leg was absent. Palpebral reflex was present. During dorsal head flexion the left eye turned ventrally. Mentation disturbances, circling and compulsively barking were detected. A routine hematology count revealed a normal cell count. Serum biochemistry was also performed. Blood nitrogen urea (BUN), creatinine (CREA), total proteine (TP), albumin (ALB), globulins (GLOB), alanine aminotransferase (ALT), aspartate aminotransferas (AST) and alkaline phosphatase (AP) were within normal ranges, wherease serum glucose was low (3.8 mmol/L) due to feesting. A cerebrospinal fluid sample was taken from the cisterna magna under general anaesthesia. The colour of the cerebrospinal fluid (CSF) was yellow (xanthocromia). In the CSF there were 114 cells in mm^3; 90% were neutrophils and the rest were mononuclear cells, macrophages and 3 red blood cells. Bacteriological examination of the CSF was negative. The Pandy test was positive (+++), TP was 180 mg/dL. CSF contained 2.14 mmol/L of glucose and 0.78 mmol/L of lactate. There was a lack of clinical improvement and the prognosis was poor, so the animal was euthanized and submitted for necropsy.



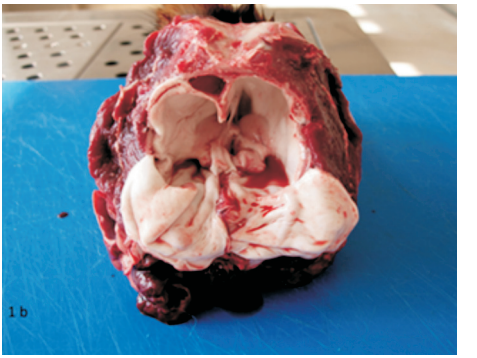


Fig. 1a and b. Dogs (cases 1 and 2), opened calvarium; hemispheres reveal significantly dilated ventricles and loss of brain tissue; in the ventral part of the ventricles there are well developed caudate nuclei and hippocampus.

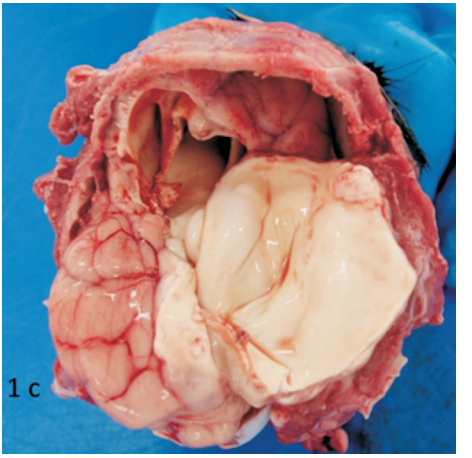


Fig. 1c. Dog (case 3), opened calvarium; slightly thicker remnants of brain parenchyma surrounding the lateral ventricles

Necropsy examination in all cases revealed that the cranial cavity was complete and of normal conformation. During the opening of the cranial cavity, in all cases a large amount of reddish fluid began to leak out because the hemispheres were also partialy cut. Following the complete opening of the calvarium and the hemispheres, it was visible that the brains had turned into a sacs filled with lightly reddish CSF and marginated with leptomeninges and discrete remnants of the brain parenchyma. In cases 1 and 2 these remnants included 1.5 mm of the brain cortex and very thin (0.5 - 1 mm) attenuated white matter (Fig. 1a and b). In case 3 the remnants of the brain parenchyma consisted of 2 - 3

mm of brain cortex and 1-2 mm of white matter (Fig. 1c). It was apparent that in cases 1 and 2 all parts of the hemispheres (including the frontal, parietal, temporal and occipital lobes) were uniformly defective, gyri were absent and the brain cortex looked smooth, while in case 3 the parietal and occipital parts of the hemispheres were most severely defective and the gyri were flattened. In all cases the brain stem was of near-normal conformation with well-developed caudate nuclei and hippocampus. Regarding lesions in other organs, in cases 1 and 2 severe congestion and oedema in the lungs were noticed, while in case 3 no macroscopic lesions in other organs were evident.

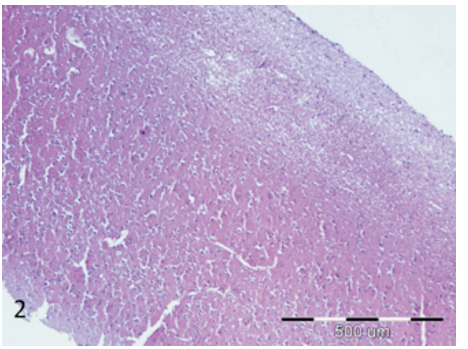


Fig. 2. Dog - case 1, brain; the residual brain cortex with a very thin layer of vacuolated white matter and attenuated ependyma. H&E; ×10.

At necropsy, in all cases, part of the fresh brain was submitted for direct immunofluorescence for rabies virus, and the brain, heart, lungs, liver, spleen, kidneys and intestine were fixed in 10% neutral buffered formalin and embedded in paraffin. Tissue samples were cut into 3 to 5 µm thick sections and stained with haematoxylin and eosin (H&E), Gram and Giemsa methods. Additionally, immunohistochemistry was performed on paraffin embeded tissue using streptavidin-peroxidase complex staining for canine distemper virus (anti-CDV monoclonal antibody, 1 : 800, VMRD Inc, Pullman, WA), and canine parvovirus (anti-CPV monoclonal antibody, 1 : 100, VMRD Inc, Pullman, WA).

In cases 1 and 2 histopathology revealed that the remnants of the hemispheres comprised the meninges, attenuated cortex and a very thin zone of vacuolated white matter (Fig. 2); this residual tissue was lined by attenuated ependyma. There was no evidence of inflammation. In case 3, the cortex, which was partly attenuated, was also covered with meninges and lined with a thin zone of white matter, lined by attenuated ependyma which was frequently incomplete or absent. In a narrow band of tissue that bordered the cavity, particulary in the ventral part, vasculitis was seen, characterised by infiltration of lymphocytes and plasma cells admixed with a few neutrophils in and arround the walls of the small blood vessels; multifocally there were also areas of acute hemorrhage

(Fig. 3a) that was occasionally accompanied by infiltration of the reactive macrophages (Fig. 3b) and few neutrophils which phagocytosed necrotic brain tissue; multifocally there were also large axonal spheroids (Fig. 3c). In all cases, Giemsa and Gram stains, performed on tissue embedded in paraffin, revealed no bacteria or protozoal structures. Immunolabeling for canine distemper virus and parvovirus and immunofluorescence for rabies were negative. Histopathological analysis of other organs revealed only severe edema and congestin in the lungs of the dogs in cases 1 and 2.

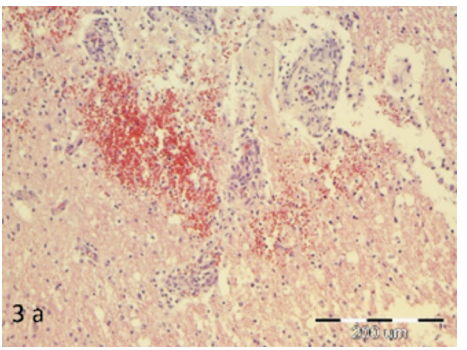


Fig. 3a. Dog (case 3), brain, ventral part of the hemispheres; vasculitis of small blood vessels accompanied by areas of acute hemorrhage. H&E; ×20.

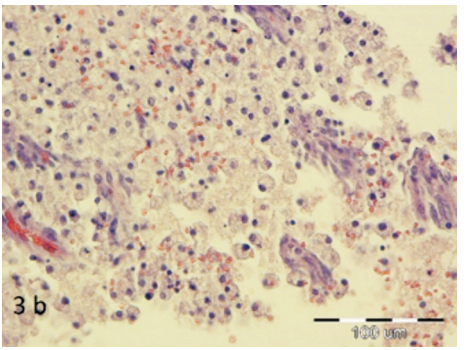


Fig. 3b. Dog (case 3), reactive macrophages (gitter cells) in the area of vasculitis and hemorrhage. H&E; ×40.

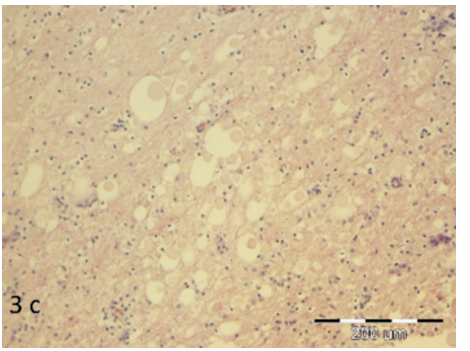


Fig. 3c. Dog (case 3), large eosinophilic axonal swellings (axonal spheroids) near the area of vasculitis and hemorrhage. H&E; ×20.

According to these findings, cases 1 and 2 were diagnosed as hydranencephaly, and case 3 was diagnosed as hydrocephalus associated with periventricular encephalitis.

Discussion

In the veterinary literature, there have only been a few documented spontaneous cases of severe cavitatory lesions of the brain in dogs. Here we describe three cases of such lesions with clinical findings that preceded the death of the animals. When evaluating hydrocephalus and other cavitatory lesions of the brain in any species, it is important to differentiate developmental and inflammatory origins (SIPMA et al., 2013). Congenital malformations are abnormalities that are present at the time of birth, but there is little information regarding their incidence in animals (DAVIES et al., 2012). In veterinary medicine, secondary malformations are most commonly reported, such as porencephaly and hydranencephaly, associated with maternal infections during fetal development. These infections are most common in ruminants, and are usually caused by viruses, such as herpesviruses, orbiviruses or bunyaviruses (WOUDA et al., 2008). Furthermore, similar lesions have been described in cats infected with panleucopenia virus (SHARP et al., 1999), and in dogs, severe acute encephalitis and hydrocephalus were experimentaly induced by the canine parainfluenza virus (BAUMGARTNER et al., 1982). In the Irish terrier (case 1) and German shepherd (case 2) in which hydranencephaly was described, no inflammation or evidence of infection was identified in histopathological sections. Also, another dog from the same litter as the Irish terrier did not show any signs of disease or neurological disorders, which also supports the fact that infection was not likely. We also did not notice any anomaly in the bone structures of the skull and neck vertebrae that could interfere with the flow of cerebrospinal fluid. Another possible cause of such lesions is decreased cerebral perfusion and the subsequent loss of brain tissue, which was also considered in similar cases described earlier (DAVIES et al., 2012). However, anatomical or histological changes in brain vessels were not noticed, but it is important to emphasize that a brain with such severe cavitatory lesions is extremely difficult for postmortem analysis, because it is prone to desintegration.

The most striking features in the English setter (case 3) were the inflammatory and degenerative changes in the subependymal and periventricular white matter of the lateral ventricles. The sudden onset of rapidly progressing neurological signs and severe pathological process associated with the cerebrospinal fluid pathways indicated an acquired type of hydrocephalus. This is in accordance with the cases in dogs previously described by HIGGINS at al. (1977), CANTILE et al. (1997) and in a fox MANDARA et al., (2007). Due to the presence of periventricular neutrophilic/mononuclear cell infiltration, it is thought that a bacterial infection may have been the cause of the inflammatory lesions, with internal hydrocephalus secondary to the severe periventricular lesions. However, we did not identify any infectious agent and similar conditions were previously reported in the pathogenesis of spontaneously occurring acquired canine and fox hydrocephalus, where no viral or bacterial causes had been identified, except *Staphylococcus aureus* and *Pasteurella multocida* in one case (HIGGINS at al., 1977; CANTILE et al., 1997; MANDARA

et al., 2007). Hovewer, this does not exclude an infectious etiology, and suggests that a larger number of infectious agents should be investigated.

Neurological disturbances in dogs suffering from cavitatory lesions in the brain are nonspecific, and it may be assumed that they depend on the severity of the affected brain tissue. However, in the cases presented, the dog in case 1, despite the severe loss of brain tissue, showed only sleepiness and retroflexion of the head during sleeping, while the dog in case 2 did not show any symptoms. Both dogs died suddenly, with signs of severe lung congestion and edema. These findings are quite surprising in relation to the previously described cases, where dogs with total loss of the temporal and parietal lobes had behavioral changes and circular movements (DAVIES et al., 2012). The same authors suggested that dogs with hydranencephaly had insufficient abnormal cortical tissue remaining for it to act as a seizure focus (DAVIES et al., 2012). In contrast, the dog in case 3 had slightly more brain tissue preserved and showed more severe nervous simptoms. Similar simptoms in dogs with hydrocephalus and associated periventricular encephalitis were described by HIGGINS et al. (1977). This suggests that the reason for the more severe neurological signs, apart from more preserved brain tissue, could also be the accompaning inflammation and rapid progress of disease.

Xanthocromia derives from bilirubin originated from hemoglobin, which is formed by macrophages and leptomeningeal cells that degrade hemoglobin from red blood cells (VERNAU et al., 2008). This indicates damage of the vascular system in the central nervous system and the loss of the blood-brain barrier (DE LAHUNTA and GLASS, 2009). The source of the red blood cells in CSF could be from contamination during sampling due to traumatic puncture or subarachnoid hemorrhage. In the case of contamination, RBC are predominant in the total cell count. In case 3 here contamination of the CSF with blood is excluded because 90% of the cells were neutropils. Xanthocromia and neutrophilia were the result of vasculitis, encephalitis and hemorrhage in the periventricular area, which was detected histologically. Futhermore, collection of CSF was performed from the cisterna magna under general anaesthesia, which was less likely to cause contamination.

Normal ranges of total protein in the CSF are considered to be from 13 to 28 mg/dL according to VERNAU et al., 2008, whereas DE LAHUNTA and GLASS (2009) gave 25-30 mg/dL. In case 3 here the total protein concentration was 180 mg /dL, which excedeed normal values 6 times. The protein concentration along the neuroaxis is the lowest in the ventricular fluid and the highest in the lumbal sac (DEISENHAMMER et al., 2006). The possible reasons for the elevated total protein concentation are: the increased permeability of the blood/brain/CSF barrier, intrathecal production of proteins, interruption of CSF flow or absorption probably due to severe inflammation.

Glucose is an essential energy source for maintenance of neurotransmitter homeostasis during synaptic activity (BAK et al., 2006). In case 3, concentrations of CSF glucose and serum glucose were decreased, but the ratio between CSF and serum glucose

was 0.56, which may be considered to be slightly increased. Serum glucose concentration was decreased, probably due to food deprivation. The increased ratio between CSF and serum glucose could be the consequence of increased blood vessel permeability due to inflammation, loss of the blood-brain barrier or even because of decreased brain activity due to loss of tissue. Also, in bacterial meningitis a low CSF glucose level ranging from 0.55 to 2.15 mmol/L has been reported (EISENHUT et al., 2003). In case 3 the CSF lactate concentration was 0.78 mmol/L and in the serum it was 2.83 mmol/L. According to DEISENHAMMER et al. (2006) CSF lactate concentration is independent of blood concentration. The reason for the decreased lactate concentration in the CSF could be decreased oxydative phosphorilation due to severe neural tissue damage.

In conclusion, due to the low number of severe spontaneous cavitatory lesions in dogs, clinical signs and causes are still incompletely understood. Severe loss of cortical brain tissue could be the reason for the absence of some neurological symptoms in affected animals.

References

BAK, L. K., A. SCHOSUBE, U. SONENEWALD, H. WAAGEPETERSEN (2006): Glucose is necessary to maintain neurotransmitter homeostasis during synaptic activity in cultured glutamatic neurons. J. Cereb. Blood Flow Metab. 26, 1285-1297.

BAUMGARTNER, W. K., S. KRAKOWKA, K. OESTNEAR, J. EVERMANN (1982): Acute encephalitis and hydrocephalus in dogs caused by Canine Parainfluenza Virus. Vet. Pathol. 19, 79-92.

CANTILE, C., M. ARISPICI, M. MODENATO, R. FATZER (1997): Hydrocephalus with perivantricular encephalitis in the dog. Zentralbl. Veterinärmed. A. 44, 595-601.

DAVIES, E. S. S., H. A. VOLK, S. BEHR, B. SUMMERS, A. DE LAHUNTA, H. SYME, P. JULL, L. GAROSI (2012): Porencephaly and hydranencephaly in six dogs. Vet. Rec. 7, 170-179.

DEISENHAMMER, F., A. BARTOS, R. EGG, N. E. GILHUS, G. GIOVANNONI, S. RAUER, F. SELLEBJERG; EFNS TASK FORCE (2006): Guidelines on rutine cerebrospinal fluid analysis. Report from an EFNS task force. Eur. J. Neurol. 13, 913-922.

DE LAHUNTA, A., E. GLASS (2009): Cerebrospinal fluid and Hydrocephalus. In: Veterinary Neuroanatomy and Clinical Neurology. (De Lahunta, A., Ed.), Saunders Elsevier, St. Louis, pp. 54-76.

EISENHUT, M., T. MEEHAN, L. BATCHELOR (2003): Cerebrospinal glucose levels and sensorineural Hearing loss in bacterial meningitis. Infection. 4, 247-250.

HIGGINS, R. J., M. VANDEVELDE, K. B. BRAUND (1977): Internal hydrocephalus and associated periventricular encephalitis in dogs. Vet. Pathol. 14 236-246.

JOHNSTON, I., C. TEO (2000): Disorders of CSF hydrodynamics. Childs Nerv. Syst. 16, 776-799.

MAXIE, G. M., S. YOUSSEF (2007): Nervous system. In: Jubb, Kennedy and Palmer's Pathology of Domestic Animals. (Maxie, G. M., Ed.), Saunders Elsevier, Philadelphia, pp. 281-457.

MANDARA, M. T., S. PAVONE, G. VITELLOZZI (2007): Internal hydrocephalus and associated periventricular encephalitis in a young fox. Vet. Pathol. 44, 713-716.

REKATE, H. L. (2008): The definition and classification of hydrocephalus: a personal recommendation to stimulate debate. Cerebrospinal Fluid Res. 5, 2.

SCHMIDT, M. J., S. KLUMPP, K. AMORT, S. JAWINSKI, M. KRAMER (2012): Porencephaly in dogs and cats: magnetic resonance imaging findings and clinical signs. Vet. Radiol. Ultrasound 53, 142-149.

SHARP, N. J., B. J. DAVIS, J. S. GUY, J. M. CULLEN, S. F. STEINGOLD, J. N. KORNEGAY (1999): Hydranencephaly and cerebellar hypoplasia in two kittens attributed to intrauterine parvovirus infection. J. Comp. Pathol. 121, 39-53.

SIPMA, K. D., P. CORNILLIE, M. N. SAULEZ, T. A. E. STOUT, G. VOORHOUT, W. BACK (2013): Phenotypic characteristics of hydrocephalus in stillborn friesian foals. Vet. Pathol. 50, 1037-1042.

VERNAU, W., K. A. VERNAU, C. S. BAILEY (2008): Cerebrospinal fluid. In: Clinical Biochemistry of Domestic Animals. (Kaneko, J. J., J. W. Harvey, M. L. Bruss, Eds.), Academic Press, Elsevier, Burlington, San Diego, London, pp. 769-808.

VANDEVELDE, M., R. J. HIGGINS, A. OEVERMANN (2012): Veterinary neuropathology. Essentials of theory and practice. 1st ed., Wiley-Blackwell, West Sussex, UK, pp. 92-105.

WOUDA, W., M. P. ROUMEN, N. H. PEPERKAMP, J. H. VOS, E. VANGARDEREN, J. MUSKEMS (2008): Hydranencephaly in calves following the bluetongue serotype 8 epidemic in the Netherlands. Vet. Rec. 162, 422-423.

ZACHARY, J. F. (2012): Nervous system. In: Pathologic Basis of Veterinary Disease. (Zachary, J. F., M. D. McGavin, Eds.), Elsevier Mosby, St. Louis, pp. 771-870.

Received: 8 July 2015
Accepted: 10 December 2015

GUDAN KURILJ, A., M. LIPAR, M. HOHŠTETER, B. PIRKIĆ: Klinička slika i fenotipska obilježja jakih kavitatornih lezija u mozgu pasa - prikaz slučaja. Vet. arhiv 86, 743-752, 2016.

SAŽETAK

Vrlo jako proširenje lateralnih moždanih klijetki praćeno s jakom atrofijom moždane kore prikazano je kod tri psa. U prvom slučaju (irski terijer) i drugom slučaju (njemački ovčar) opisana je hidranencefalija karakterizirana potpunim gubitkom moždanih polutki ostavljajući samo oko 2 mm moždanog tkiva što predstavlja tekućinom ispunjene vreće. Klinički je irski terijer pokazivao samo pospanost i retrofleksiju glave tijekom spavanja, a njemački ovčar nije pokazivao neurološke ni druge znakove bolesti. U trećem slučaju (engleski seter), unutarnji hidrocefalus vjerojatno je bio sekundaran zbog teškog periventrikularnog oštećenja karakteriziranog neutrofilnim/mononuklearnim staničnim infiltratom. Ta je lezija dijagnosticirana kao hidrocefalus povezan s periventrikularnim encefalitisom, a pas je pokazivao teške neurološke znakove kao što su pospanost, opistotonus, grčevi mišića, poteškoće s vidom, vrtoglavica, ataksija i nereagiranje na zvučne podražaje. U sva tri slučaja nije utvrđen infekciozni ni drugi specifičan uzrok.

Ključne riječi: pas, kavitatorne lezije, hidranencefalija, hidrocefalus, perivaskularni encefalitis