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Case Reports

MAGNETIC RESONANCE IMAGING FINDINGS OF INTRACRANIAL GLIOMA IN A TIGER-BOXER BREED DOG WITH SEIZURES

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INTRODUCTION

Seizures are widespread neurological anomalies observed in dogs and as understood, tumor localization is one of the most important factors related to the development of seizures (Bush *et al.*, 2002). Seizures can be seen as reactive epileptic episodes from extracranial sources such as metabolic disorders and toxications or from intracranial diseases such as primary epilepsy, encephalitis, neoplasia, degeneration, malformation and trauma that are from idiopathic sources (Oliver and Lorenz, 1993; Aslan, 1998; Bush *et al.*, 2002). Seizures are commonly a widespread reflection of various intracranial physiological anomalies (Oliver and Lorenz, 1993; Bagley and Gavin, 1998 and Bush *et al.*, 2002). Seizures can occur as focal or generalized, but can also be the single symptom of intracranial tumors (Oliver and Lorenz, 1993; Bagley and Gavin, 1998; LeCouteur, 2001 and Taylor, 2003). Seizures seen in dogs with a brain tumor are evidences that indicative of an underlying structural brain disease (Oliver and Lorenz, 1993; Bagley and Gavin, 1998 and LeCouteur, 2001). Tumor localization is one of the most important factors related to the development of seizures (Bagley and Gavin, 1998; Lipsitz *et al.*, 2003). Seizures are commonly related to tumors that involve the cerebral hemispheres and the diencephalons (forebrain or the supratentorial structures). However, some seizures are related to the frontal lobes, olfactory zone, parietal lobes or brain stem (Bagley and Gavin, 1998). Developed imaging techniques such as computed tomography (CT) or MRI are required to diagnose and determine the structural anomalies of these tumors (Bagley and Gavin, 1998; Bush *et al.*, 2002; Lipsitz *et al.*, 2003; McConnell *et al.*, 2004 and Ohlerth and Scharf, 2006). In the light of this information, we studied the brain of a dog with a suspicious cranial neoplasia that was referred to our clinic due to complaints of seizures. In the MRI studies, it was concluded that the causes of epileptoid seizures was due to neoplastic process suggestive of an existing primary glial neoplasm.

KEY WORDS: MRI, Seizure, Dogs, Brain, Tumor

CASE HISTORY

A 12-year old, male, Tiger Boxer dog was referred to our clinic in Istanbul University, Veterinary Faculty, Department of Internal Medicine due to epileptoid seizures. To determine the cause of seizures, as we assumed that metabolic factors could lead to the episodes of epilepsy. Blood profile, urine tests, electrocardiography, thoracic radiography and abdominal ultrasonography were carried out. After these, a brain Magnetic Resonance Imaging (MRI) was performed as we thought seizures may originate from a lesion located in the brain. For this purpose, T1-weighted (T1W) images, T2-weighted (T2W) images and FLAIR at the axial plane, T1-weighted (T1W) images at the sagittal plane and FLAIR cross-sections at the coronal plane were obtained without any contrast medium. As the seizures showed a tendency to increase even though antiepileptic agents and corticosteroid therapy were administered, the owners consented to euthanizing the dog, but was not favourably disposed to a post mortem being performed. The biochemical parameters in a dog with a cranial tumor were as follows:

PARAMETER/UNIT	RESULT	NORMAL RANGES
Alkaline Phosphatase ALP (IU/L)	987	10-150
Alanine Amino Transferase (ALT) (IU/L)	166	5-60
Aspartate amino transferase (AST) (IU/L)	45	5-55
Blood Urea Nitrogen (BUN) (mg/dL)	11	7-27
Creatinine kinase (CK) (IU/L)	104	10-200
Creatinine (mg/dL)	1,6	0.4-1.8
Lactate Dehydrogenase (LDH) (IU/L)	139	50-380
NH3 (μmol/L)	9	0-40
Creatine kinaze-MB (CK-MB) (IU/L)	18	0-35
Free thyroxine (fT4) (ng/dL)	0,7	0.7-2.1
Cholesterol (mg/dL)	369	112-328
Triglyceride (mg/dL)	110	20-150
Parathormone (pg/ml)	3,0	15-150
Albumin (g/dL)	2,9	2.6-4.3
Calcium (mg/dL)	13,4	7.5-11.3
T. Protein (g/dL)	7,6	5.1-7.8
Globulin (g/dL)	4,7 g/dl	2.3-5.2
Phosphorus (P) (mg/dL)	4,5 mg/dl	2.1-6.3

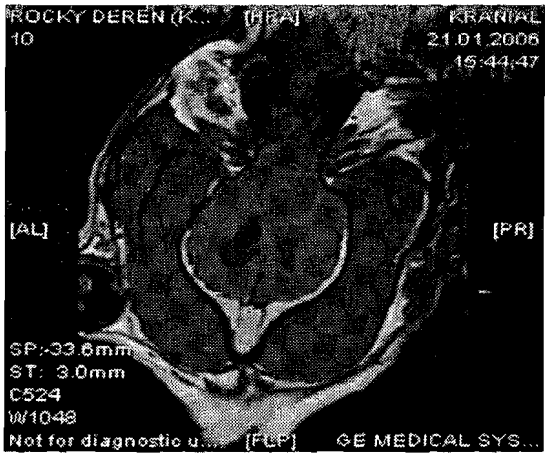

PLATE 1: Axial Plane, T1W, MRI findings of intracranial glioma in a dog

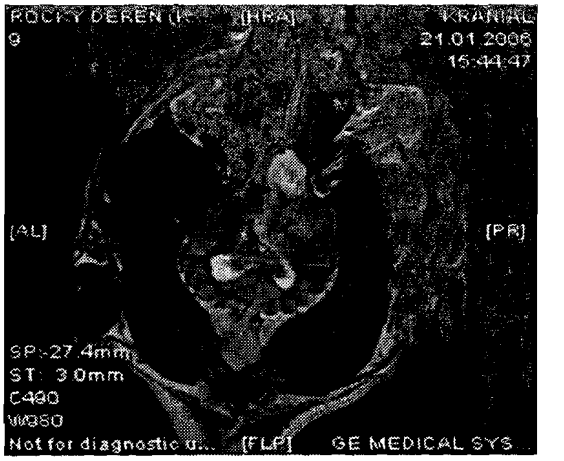

PLATE 2: Axial Plane, T2W, MRI Findings of intracranial glioma in a dog.

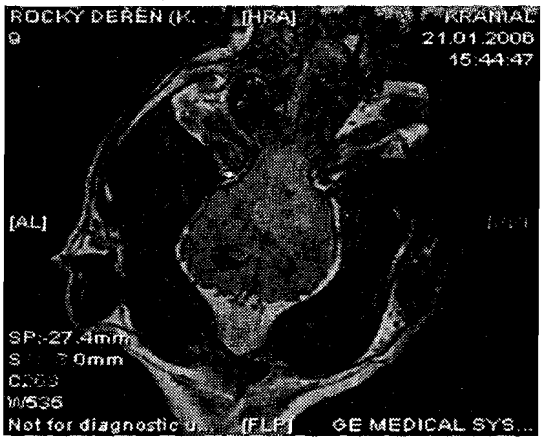

PLATE 3: Axial Plane, FLAIR, MRI findings of intracranial gloom in a dog,

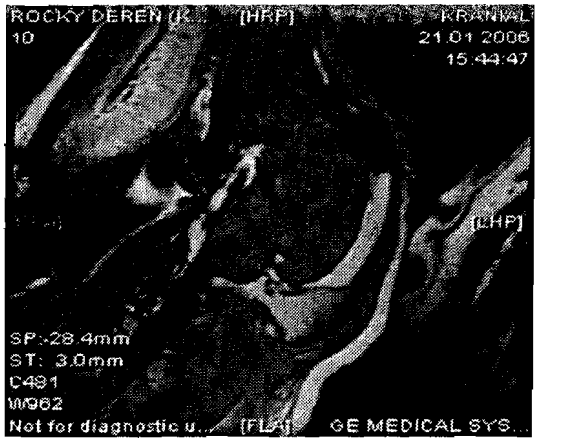

PLATE 4: Sagittal Plane, T1W, MRI findings of intracranial glioma in a dog,

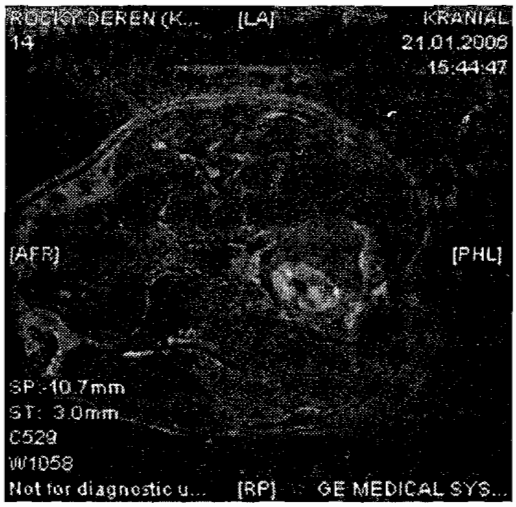

PLATE 5: Coronal Plane, FLAIR, MRI findings of intracranial glioma in a dog.

DISCUSSION

An increase is seen in the incidence of seizures related to primary brain tumors in humans (Le Couteur, 2001). Brain tumors are seen in dogs at a 14.5 risk ratio in 100.000, while these seizures are reported as clinical symptoms of tumors (Bagley and Gavin, 1998 and Le Couteur, 2001). However, the actual incidences of seizures that indicate a relationship with intracranial tumors are not completely known (Bagley and Gavin, 1998). The anamnesis of the dog indicated that the animal was undergoing epileptoid type generalized seizures for a period of 3 months which were occurring frequently, and that the dog's general condition was good. The dog however developed a noticeable severe uneasiness and behavioural disorders were also observed. However, excluding epileptoid seizures, no other clinical neurological symptoms that indicated a focal lesion in the brain of the dog was observed.

Some researchers (Triolo *et al.*, 1994 and Taylor, 2003) have reported that neurological symptoms that develop due to brain tumors may differ day by day and that masses that destroy the adjacent tissues and increase the intracranial pressure (edema, hemorrhage, obstructive hydrocephalus) may have an impact on the appearance of the neurologic symptoms.

In this case report the patient had an increase in serum Alkaline Phosphatase (ALP) and Calcium levels and the decrease in the parathormone level obtained from laboratory analyses were compatible with paraneoplastic syndromes as reported in the literature (Bergman, 2001). The absence of anomalies in other, biochemical, urologic, electrocardiographic, radiographic and ultrasonographic tests allowed us to eliminate extracranial causes of cerebral functional disorders similar to the disorders reported by other researchers (LeCouteur, 2001; Couto, 2003 and Lipsitz *et al.*, 2003).

The diagnosis of the intracranial masses can be done by advanced devices such as CT or MRI (Bagley and Gavin, 1998; LeCouteur, 2001; Bush *et al.*, 2002; Lipsitz *et al.*, 2003; Taylor, 2003 and Ohlerth and Scharf, 2006). When, MRI studies were performed in the dog with seizures, a mass was seen at the right frontal lobe of the cranium, approximately 4x2.5x2 cm in dimensions, ovoid structured and characterized mixed intensities (hypo and hyper intensity). The mass appears hemorrhagic at the central cross section, and therefore a presence of a calcified component can be considered, whereas the mass was millimetric in dimension, characterized with a T2W hypointensity nodular signaling area at the superior and peripheral cross sections and contained a few necrotic/cystic components. There is also mass effect with shift of midline structures to the left side. The radiological appearance of the diseased dog suggested an existence of a primary glial neoplastic process at the frontier plan (PLATE 1, 2, 3, 4 and 5).

Some researchers (Bagley and Gavin, 1998 and Le Couteur, 2001) reported that, in dogs with a brain tumor, according to localization and character of the tumor, the tumor could be removed by surgical intervention or by chemotherapy/radiotherapy, but that unfortunately the success rates with such treatments especially in gliomas were slim. It was also reported that, seizure disorders could be managed by antiepileptic agents that are symptomatic and supportive drugs in the primary treatment of brain tumors and that corticosteroid administration should be carried out frequently in a simultaneously (Triolo *et al.*, 1994; Bagley and Gavin, 1998 and Taylor, 2003). However, there

have been reports that standard antiepileptic agents administered in the treatment of seizures resulting from brain tumors were inadequate in the control or improvement of the disease (Triolo *et al.,* 1994, Bagley and Gavin, 1998). Dogs with a brain tumor frequently are euthanized due to the difficulties in controlling seizures, because of such clinical problems that are a significant marker of mortality and morbidity in the dog (Bagley and Gavin, 1998 and McConnell *et al.,* 2004).

Lipsitz *et al* (2003) reported that the MRI findings of gliomas were almost similar to the MRI findings in humans, but nevertheless there were only few studies on glioblastomas in dogs and therefore, in order to achieve definite statistical data, it was necessary to perform a considerable number of animal studies.

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