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A rare case of post ictal blindness in a Labrador dog suffering from status epilepsy

Desh Deepak, Vipul Thakur, Naresh Chandra and Dr. Vinod Kumar Varun

Abstract
The present study reports an unusual complication of Post ictus bilateral visual loss for a transient period associated with status epilepticus in a four-year Labrador dog presented to Veterinary clinical complex, SVPUAT, Meerut. The case was presented with a history of seizures for the last two years and was undergoing treatment with oral phenobarbitone therapy. At the time of presentation, the dog was in a continuous state of seizures for the last 35 minutes therefore, the case was tentatively diagnosed as status epilepticus and antiepileptic therapy was initiated using intravenous diazepam bolus @1.0 mg/kg body weight. Seizures were nonresponsive therefore second bolus of intravenous diazepam was administered @ 2.0 mg/kg body weight in combination with intravenous mannitol @1.0 mg/kg body weight. Seizures were responsive to high-dose intravenous diazepam therapy @ 2.0 mg/kg body weight and were further managed with a combination of phenobarbitone and valproic acid. The dog was observed on the second day showing the state of delirium and walking into obstacles. Ocular examination reveals no ocular abnormality. Menace test and obstacle test suggest loss of vision but pupillary light reflex was intact. Examination of CSF reveals normal appearance and normal cell count. Based on history and clinical evidence, the condition was tentatively diagnosed as post ictalblindness which resolves within 48 hours. The Drugs were well tolerated by the dog with recovery from status epilepsy and subsidence of post-ictus blindness within 48 hours.

Keywords: Epilepsy, blindness, diazepam, post ictal, seizures

Introduction
Epilepsy is considered as most common nervous disorder in clinical veterinary practice (Charalambous*et al*., 2014) [4]. Epilepsy refers to heterogeneous pathology characterised by the appearance of recurrent seizures (Stafstrom and Carmant, 2015) [8]. Seizures refer to paroxysmal neurological alteration resulting from the hypersynchronous firing of neurons in the brain (Stafstrom and Carmant, 2015) [8`]. Seizures can be epileptic or non-epileptic/psychogenic. Epileptic seizures result from abnormal firing of neurons while non-epileptic seizures are psychogenic in origin. A seizure can be classified into three types *viz* focal, generalised, and focal seizures which tend to generalize later. Focal seizures originate from neuronal firing restricted to localized areas limited to one cerebral hemisphere while generalised seizures are associated with the abnormal firing of neuronal networks distributed over both cerebral hemisphere (Stafstrom and Carmant, 2015) [8]. Clinical manifestations of focal seizures depend upon the area of the firing neuronal network for example focal seizures associated with the occipital lobe are presented with a visual malfunction, temporal lobes with dyscognition and seizures involving post-central gyrus with paresthesia. Post-seizure manifestations can last for a few minutes to a few hours in most cases of canine and include disorientation, restlessness, weakness, hyperactivity, and aggression. Status epilepticus refers to prolonged epileptic seizures that continue for more than five minutes without recovery or occurrence of more than one seizure in quick succession without any inter-seizure recovery and extend up to 30 minutes and even more (Berendt *et al*., 2015) [5]. Status epilepticus is associated with various systemic complications and can cause irreversible neurological damage, if not treated properly within adequate time (Blades and Rossmeisl, 2017) [6]. Such systemic complications include shock, cardiac arrest, and respiratory collapse. The intensity of all status epilepticus associated complications shows a correlation with the duration of episodes of status epilepticus (Costello and Cole, 2007) [7]. The present case reports transient blindness precipitating after an episode of status epilepticus and continued for 48 hours.

Materials and Methods

History, clinical observation, and diagnosis

A four-year Labrador dog was presented to the veterinary clinical complex, Sardar Vallabhbhai Patel University of Agriculture and Technology, Meerut, UP with a history of epilepsy for the last two years. The dog has suffered four to five episodes of seizures during the last three months. At the time of presentation, the dog was in a state of continuous fits for the last 35 minutes. Clinical examination reveals elevated rectal temperature up to 104o F and normal lymph nodes. Blood sample was collected for hematological and biochemical analysis revealing normal blood count, normal organ functions, and the absence of any haemoprotozan infection. Examination of cerebrospinal fluid revealed the absence of pleocytosis. Based on clinical manifestations and laboratory examination, the condition was tentatively diagnosed as status epilepticus. Status epilepticus was successfully managed with diazepam but after recovery, the dog showed signs of delirium and moving into obstacles. Dog's eye was clinically examined for any evident lesion. The dog didn't blink on the menace test and failed to appreciate the obstacles establishing loss of vision. Positive pupillary light reflex confirms intactness of reflex arc. Since the eyes were fully intact with positive pupillary light reflex, the condition was diagnosed as post ictal cortical blindness.

Results

Status epilepticus was successfully managed with intravenous diazepam. The epilepsy was maintained with oral phenobarbitone and valproic acid. Post ictal blindness was transient and resolved within 48 hours. The hematology and biochemical evaluation was performed on the day the case was presented to the veterinary clinical complex after initiation of treatment. The findings of hematological and biochemical evaluation have been presented in Tables 1.1 and 1.2.

Table 1: Hematological analytes of epileptic dog

Erythrogram analytes		
Parameters	**0 Day**	**References range**
TEC (× 10 6 /µl)	5.2	5.5-8.5
HB (gm/dl)	12	12-18
PCV (%)	40	37-55
MCV (%)	71	62-77
MCH (Pcg)	22	21-26.2
MCHC (gm/dL)	33	32-36
Leucogram analytes		
WBC (/µL)	13500	6000-17000
Neutrophils (%)	72	62-80
Lymphocytes (%)	17	10-28
Monocytes (%)	8	3-9
Eosinophils (%)	2	2-12
Basophils (%)	1	0-2

Table 2: Biochemical analytes of epileptic dog

Biochemical Parameters		
ALT(U/L)	48	60
AST(U/L)	62	50
ALP(U/L)	168	150
Total protein(g/dL)	7.7	6-8
Albumen(g/dL)	4.1	5-6
Globulin(g/dL)	3.6	3-5
A/G	1.13	0.59-1.11
Total Bilirubin(mg/dL)	0.09	0.07-0.061
Direct Bilirubin(mg/dL)	0.06	0.06-0.012
Indirect Bilirubin(mg/dL)	0.03	0.01-0.049
T4 (nmol/L)	42.4	19-58
TSH (nmol/L)	0.068	0.04-0.35
BUN (mg/dL)	18.8	10-20
Creatinine(mg/dL)	1.2	0.5-1.5

Discussion

Cortical blindness refers to loss of vision as a result of brain lesions (Kumar and Muppala, 2021) [3]. Cortical blindness is an unusual post ictal manifestation in humans (Badran *et al*., 2018) [1]. Most cases of post ictal blindness in humans are associated with generalised or focal occipital seizures of children (Badran *et al*., 2018) [1]. The occipital lobe receives visual input from the eyes and processes them therefore affection of the occipital lobe results in decreased vision, nystagmus, and transient or permanent loss of vision. This blindness can persist for minutes to days or can be permanent.

Excessive recurrent seizures *viz* cluster seizures and status epilepticus are known to induce secondary epileptic brain damage (EBD) in both animals and humans (Hasegawa *et al*., 2005) [2]. EBD precipitates various neurological and psychological aberrations in humans and aggression in dogs (Hasegawa *et al*., 2005) [2]. Epileptic brain damage in the region of the occipital lobe will result in transient or permanent loss of vision. Other than EBD there are various explanations proposed by various researchers from time to time to explain post ictal complications like paralysis. In 1856, Todd proposed his exhaustion theory to explain the postictal paralysis but it was rejected by Efron as an explanation for post ictal blindness. Efron proposed the theory of hyperpolarization induced post ictal inhibition to explain post ictal blindness. Olurin credited interictal brain anoxia induced lactic acidemia as the cause of bilateral loss of vision. Nowadays, inter ictal brain anoxia is credited as the chief cause of post ictal blindness.

Cortical blindness will be presented with normal pupillary light reflex since the visual pathway anterior to the geniculate nuclei occipital lobe is normal. The present case also exhibited loss of vision with intact pupillary light reflex. This loss can be explained by cerebrovascular anoxia produced as a result of status epilepticus.

Conclusion

Epilepsy is one of the most common neurological pathologies encountered by clinical veterinary practitioners and is associated with several post seizures complications. Transient loss of vision can occur during the ictal or post ictal period.

Most of the owners and clinicians probably fail to appreciate post ictal blindness and confuse it with delirium and depression, therefore the incidence of transient post ictal blindness in canines can be anunder diagnosed and underreported condition. Oxygen administration should be instituted as soon as possible for the cases presented with status epilepticus as it will prevent ictal or post ictal cerebral anoxia. Such unusual cases warrant diagnostic imaging and electrodiagnostic tests like Encephalogram to establish the underlying structural cause which were limitations of our study.

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