Reactive seizures in 40 dogs: A clinical study

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Abstract
Seizures are the most common neurological disorders in dogs. Based on the etiology the epileptic dogs were classified as idiopathic epilepsy, reactive seizures and structural epilepsy. In this study 40/179 dogs were diagnosed with reactive seizures. They were grouped in to different age and sex. The different etiologies of reactive seizures in this study were hepatic encephalopathy, cardiomyopathy, uremic encephalopathy, infectious, hypothyroidism, hypocalcemia, verminous, hypoglycemia, tetanus and anemia. This categorisation will guide the clinician to treat for the primary cause and plan the antiepileptic treatment accordingly.

Keywords: Seizures, epilepsy, hepatic encephalopathy, uremic encephalopathy

Introduction
Seizures are the most common neurological disorders in dogs [1]. Based on etiology, seizures are grouped into three major categories – idiopathic, symptomatic and reactive [2]. A status of recurrent seizures is defined as epilepsy [3]. Reactive seizures have an extracranial origin, can be caused by variety of metabolic disturbances and intoxications [3], and can be virtually elicited by dysfunction of any organ system [4]. The metabolic causes of seizures include hypoglycaemia, hypoxia (anemia), hyperthermia, hyperosmolality, hypocalcemia, hypercalcaemia, hepatic encephalopathy, uremic encephalopathy, hypothyroidism [3]. Most of these conditions are reversible depending on the underlying disease. So, permanent antiepileptic drug therapy should only be initiated when the seizures are uncontrolled despite therapy or when an emergency situation such as status epilepticus occurs [4]. The present study evaluates the frequency of various reactive seizures in dogs.

Materials and Methods
A total of 179 dogs of various breeds, age and either sex presented to the tertiary care veterinary hospital with the history of epileptic seizures were considered for this study. Based on the preliminary investigations – complete detailed history of deworming and vaccination, clinical examination, neurological examination, complete blood count (CBC), serum biochemistry, radiodiagnosis and ultrasonography the etiological classification of the dogs were done. Among the 179 dogs 127 were idiopathic, 40 were reactive seizures and 12 were categorised as structural epilepsy.

Hypoglycemia was considered to be the cause of the seizure when dogs showed repeated low glucose levels or when the animals presented had low blood glucose levels during seizure and immediately responded to IV glucose administration.

Hepatic encephalopathy was diagnosed mainly through marked elevation in liver enzymes; diagnostic imaging of the liver and abdominal vessels by ultrasonography. Uremic encephalopathy was diagnosed by measuring plasma creatinine and urea concentrations. Electrolyte disorders were considered as the cause of seizure when at least one of the concentrations of calcium, sodium or potassium were increased or decreased. History of recent whelping in dogs with seizures and those which responded to intravenous calcium was considered as hypocalcemic tetany in female dogs (eclampsia). Hypothyroid dogs were categorised based on low T3 or T4 or elevated TSH.

Dogs with cardiac pathology confirmed by echocardiography were categorised as seizures due to cardiac origin. The blood smear of affected dogs positive for Ehrlichiosis or monocytic response were grouped as infectious cause. Undewormed puppies presented with seizures and did not develop seizures post deworming was grouped as verminous cause of seizures.
One dog was anemic and showed signs of epilepsy possibly due to hypoxia was categorised into anemic group while a dog developed tetanus after seizures was identified as seizures due to tetanus.

Results and Discussion

A total of 179 dogs were presented to the hospital with the history of seizures.

Table 1: Categories of seizures

<table>
<thead>
<tr>
<th>Category</th>
<th>No. of Animals</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic</td>
<td>127</td>
<td>70.9</td>
</tr>
<tr>
<td>Reactive</td>
<td>40</td>
<td>22.3</td>
</tr>
<tr>
<td>Structural</td>
<td>12</td>
<td>6.8</td>
</tr>
<tr>
<td>Total</td>
<td>179</td>
<td>100</td>
</tr>
</tbody>
</table>

Based on the complete clinical and neurological examination, CBC and Serum biochemistry results, radiography and ultrasonography the presented dogs were categorised into idiopathic if all the values were within normal limits. Those with change in this values and did not develop seizures after treatment for the primary cause were categorised as reactive seizures.

Those with structural cause as trauma, tumors and cysts were categorised as structural seizure. Idiopathic seizures constituted highest with 127 cases (70.9%) (Fig 1), Reactive were 40 cases (22.3%) and structural were 12 cases (6.8%) (Table 1).

Table 2: Demographic details of Reactive seizures

<table>
<thead>
<tr>
<th>Criteria</th>
<th>No. of Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>Less than 6 months</td>
<td>5 (12.5%)</td>
</tr>
<tr>
<td>6 months to 72 months</td>
<td>23(57.5%)</td>
</tr>
<tr>
<td>More than 72 months</td>
<td>12(30%)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>23(57.5%)</td>
</tr>
<tr>
<td>Female</td>
<td>17(42.5%)</td>
</tr>
</tbody>
</table>

Among the 40 dogs categorised as dogs with reactive seizures 5 (12.5%) were less than 6 months, 23 (57.5%) were between 6 months and 72 months and 12 (30%) were more than 72 months (Table 2). The age group between 6 months and 6 years is higher as the total surviving population is relatively higher in this age group.

The male dogs were 23 and female were 17 (Table 2). This might be because of relatively higher population of male dogs.

The dogs with hepatic encephalopathy constituted 12/40. This was diagnosed as discussed earlier and treated for the primary cause and the dogs responded uneventfully fulfilling these criteria. The 3/40 dogs with cardiomyopathy and 1/40 dog with anemia should have developed seizures due to hypoxia [5]. Uremic encephalopathy was diagnosed in 7/40 dogs and ultrasonography was performed to differentiate acute and chronic renal failure. Dogs with hypothyroidism were 2/40 (Table 3). The infectious cause was Ehrlichiosis in all the 5/40 dogs. The pathology of seizures in ehrlichiosis infection is attributed to non rerenerative anemia in dogs [6]. 2/40 dogs were hypocalcemic with normal values of ionised serum calcium but were nursing and didnot exhibit seizures after treating with calcium and weaning the puppies. The verminous cause was noted in S/40 dogs. The dogs recovered after deworming. Gastrointestinal parasitic load estimation for differential diagnosis of acute neurological signs in puppies was suggested [7]. Hypoglycemia was observed in 2/40 dogs and 1 / 40 dog developed tetanus. Seizure episodes in human have been reported [8]. This might be observed in dogs also and detailed study should be done for this attribute.

Energy metabolism can also be changed by metabolic diseases leading to modified osmolality or production of endogenous toxin [9]. Clinical onset in metabolic and toxic diseases is often acute, progressive and accompanied by symmetrical signs [9]. In this current study total number of dogs with reactive seizures was 40(22.3%) as against 5% reported by Podell 1996 and Brauer et al. 2011. This might be due to non - reporting of all affected cases to the institute where the study was undertaken.

To distinguish idiopathic, structural and reactive seizures, a thorough history taking and neurological examinations, CBC and blood chemistry are mandate. Reactive epileptic seizures have a 1.57 times higher odds of being status epilepticus compared to idiopathic epilepsy [10]. So, metabolic and toxic disturbances must be considered as a differential diagnosis in
cases of single generalised seizures. Our study indicates that metabolic disorder needs to be eliminated in dogs with seizure disorders. Exclusion of metabolic criteria is vital for differential diagnosis and thus therapeutic planning done accordingly for permanent dosing of antiepileptic drugs. A complete history, clinical and neurological examination and blood investigations (CBC, glucose, liver enzymes, urea, creatinine, electrolytes, total protein, albumin, globulin, bile acids) should be a part of investigations along with imaging (radiography, ultrasonography, computed tomography and magnetic resonance imaging if warranted) are mandate for therapeutic planning of epilepsy.

References