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Case Report

Clinical Management of Epileptic Seizures in a Labrador retriever Dog

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ABSTRACT

This report presents one 4 year male, Labrador retriever, dog which was found to suffer from epileptic seizures without any physical abnormalities. It was previously vaccinated for Leptospira, Canine Herpes, Canine Parvo, Corona viruses and Rabies. Deworming was done regularly at every six months. His blood parameters were found to be in normal range. It had the history of seizures earlier. It is suspected to be a case of symptomatic epilepsy due to intra cranial lesions. It was treated with Phenobarbitone (Phenobarbitone Sodium®, AHPL), Corticosteroids (Solu–Medrol®, Pfizer) slow IV injection, Mannitol (M–20®, Fresenius Kabi) and Diuretics (Lasix®, Sanofi Aventis) for five days. The fluid therapy was continuously administered to avoid the hypoglycemia and dehydration. So it was injected with dextrose 5%®, Fresenius Kabi, IV and electrolyte imbalance was checked by Ringer lactate®, Baxter, IV alternatively. On the basis of the symptoms and the response to the line of treatment, it could be the case of symptomatic epilepsy. Clinicians have reported success in symptomatic epilepsy by using osmo–theraputic agents, diuretis, corticosteroids and phenobarbiturate agents. This dog also recovered successfully from epileptic seizures after treatment with mannitol, methyl prednisolone sodium succinate, frusemide and phenobarbitone sodium.

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Epilepsy is one of the most common chronic neurological disorders in dogs (0.5–1%). In some breeds there is a strong suspicion of an underlying genetic factor as there is an accumulation of epileptic individuals within families with an incidence as high as 20% (Casal et al., 2006). Moreover, the majority of the pedigree studies suggest a polygenic mode of inheritance. Canine epilepsy can be classified either as idiopathic (genetic) or symptomatic (structural/metabolic) according to recent International League against Epilepsy (ILAE) recommendations in humans (Berg et al., 2010).

Epilepsy refers to a group of chronic neurological symptoms characterized by recurrent unprovoked seizures (Blume et al., 2001). These seizures are transient symptoms due to abnormal, excessive or synchronous neuronal activity in the brain. Seizure disorders occur frequently in dogs and cats (Fisher et al., 2005).

Most dogs that were presented with recurrent seizures have idiopathic epilepsy, which is thought to have a genetic basis and has no identifiable underlying cause when a full diagnostic work–up was undertaken. Symptomatic epilepsies, which arise secondary to brain diseases such as intracranial neoplastic lesions or central nervous system inflammatory disorders, are less common (Kate Chandler, 2011). This case report had tried to suggest some practical and effective strategies for managing and monitoring dogs with symptomatic epilepsy and also described the importance of sedation during seizures along with the clinical management of the epileptic seizures in a Labrador retriever dog.

One 3 years old, 40kg body weight, Labrador retriever male dog was presented for treatment of continuous epileptic seizures. The dog was well covered for de–worming and vaccination for Canine Parvo virus, Canine Distemper, Leptospira 1, 2, 3, Hepatitis and Corona virus as well. It had previous history of the same disease symptoms with the temporary blindness, circling, walking aimlessly, Rectal temperature–110^0 F and also vigorous peddling of its limbs. It had severe whining until it was sedated with drug (diazepam) as it seemed like it was in severe pain. History didn't confirm it to be idiopathic epilepsy as none of its family members and relatives ever suffered from epileptic seizures. Its blood profile was found to be normal at that time. The dog was recovered after three days of treatment with injection Analgin (Vetalgin®, MSD 1.0gm, I/V, o.d. Injection Diazepam (Calmpose®, Ranbaxy, 1mg/kg BW, IV, b.i.d.) Inj–Dextrose (Dextrose 5% ®, Fresenius Kabi, 50ml/kg BW/day, IV), Injection Ceftriaxone (Intacef ®, Intas, 25mg/kg BW, IV, o.d.), Injection Mannitol (M–20®, Fresenius Kabi, 2ml/kg BW, IV b.i.d., Injection Meloxicam (Melonex®, Intas, 0.2mg/kg BW, IV, b.i.d.).

After four months of recovery of earlier epilepsy the same dog was presented with Heart Rate (HR)–62, Respiration Rate (RR)–24, Pulse Rate (PR)–76 and Rectal Temperature (RT)–102^{0}F and continuously occurring epileptic seizures. According to the owner the animal had involuntary urination and rubbed its head against the ground before the convulsion episode. Before coming down to lateral recumbence the animal went blind just like previous episode. Other symptoms included peddling of its limbs. The physical examination revealed no abnormalities. From the very first day onwards HR, RR and PR were regularly monitored for any change but it was found to be consistent around the normal values throughout its therapy.

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It was hospitalized until its recovery. Very first day, an 18G intravenous cannula was placed in the left cephalic vein. It was sedated with IV Inj Diazepam. The urinary catheter was fixed for emptying of bladder. Blood sample was collected and sent to CBC, Na^+, K^+, Liver function test and Kidney function test. Its blood profile was Hb: 10.1gm%, TLC: 9000/Cu mm, Neutrophils: 68%, Lymphocytes: 26%, Eosinophills: 02, Monocytes: 04, Basophills: 00, Platelet count: 170 thousand/Cu mm, Total RBC count: 3.8million/Cu mm, PCV: 31.1%, MCV: 81.84fl, MCH: 26.58pg MCHC: 32.48g/dl, Biochemistry: Blood urea: 31.6 mg/dl, Serum creatinine: 1.2 mg/dl, S.G.P.T: 82.6 U/L, SGOT: 65.8 U/L, Serum Sodium: 152 mEq/L, Serum Potassium: 4.5nmEq/L.

Its medication was started with Inj Diazepam (Calmpose®, Ranbaxy, 1mg/kg BW, IV, b.i.d.), Inj Mannitol (M–20®, Fresenius Kabi, 2ml/kg BW, IV b.i.d.), fluids {Inj–Dextrose (Dextrose 5% ®, Fresenius Kabi, 50ml/kg BW/day, IV) and inj Ringers lactate (Ringer lactate®, Baxter,40ml/kg BW/day, IV) alternatively}, Inj Ceftriaxone (Intacef ®, Intas, 25mg/kg BW, IV, o.d.), Inj vitamin B Complex (Neurobione Forte®, Merck, 2ml, IV, b.i.d.), Inj Frusemide (Lasix®, Sanofi Aventis, 3mg/kg BW b.i.d.) and Inj Dexamethasone (Dexona®, Zydus Alidac, 2mg o.d. IV). It was observed for any improvement for 24hrs but it didn't show any improvement. Inj Phenobarbital (Phenobarbitone Sodium®, AHPL, 2.5mg/kg BW, IV, b.i.d.) was administered due its action of longer duration of sedation.

After 72 hrs, the intravenous cannula was placed into right cephalic vein and the same medication was continued for complete five days (120hrs). Inj Methyl Prednisolone Sodium Succinate (Solu–Medrol®, Pfizer, slow IV injection was administered @ of 30mg/kg/BW followed by 15mg/kg/BW IV after 2hrs and from there on at a dose rate of 15mg/kg BW, IV every 6hrs for 24hrs. All the medications were stopped by methyl prednisolone sodium succinate being the last one. The animal was recovered completely after 72 hours of stopping all the medication.

Symptomatic epilepsy was mostly caused by intracranial tumours (16%) and encephalitis (10%). It is not possible to differentiate between idiopathic and symptomatic epilepsy based on clinical signs alone. Indications such as status epilepsy cluster or partial seizures, vocalization during seizures, and altered interictal (interval between seizures/convulsions) neurological status were more common predictors of symptomatic epilepsy. (Pakozdy et al., 2012).

Diazepam, phenobarbital and pentobarbital have all been suggested for the treatment if severe signs of epilepsy develop in dogs (Smedile et al., 1996). The effect of diazepam in our case remained only for 3hrs which was reduced to half an hour with the same dose rate after four time of administration. Phenobarbital has a long terminal half life in dogs (Fukunaga et al., 2008). Considering the previous reports and our results of long terminal half–life of phenobarbitone, we were right in selection of drugs to control the seizures. Cyclosporine/prednisolone combination therapy increases survival time in dogs with granulomatous meningoencephalitis compared to prednisolone alone, and even long–term remission can occur (Pakozdy et al., 2013).

The animal was completely anorectic and unable to take water as well which surely would have disturbed his fluid and electrolyte balance in the body. Lasix and fluid restriction produces dehydration, fall of blood pressure, low cerebral perfusion pressure and increased risk of cerebral thrombosis. After considering all the situation and animal condition it was decided to infuse the fluids and electrolytes at the dose rates mentioned in the treatment part. Dextrose was administered for maintenance of the energy and fluid balance in the animal body in the form of 5% solution IV. Ringers lactate was given for electrolyte balance in the body fluids. According to the Burdett and Stephens (2006), the key principles are to replace losses or deficits 'like for like', continue maintenance and to anticipate additional on–going losses. Here we tried to maintain the fluid and electrolyte balance by giving fluids like Dextrose Normal Saline, Dextrose–5% and Ringers lactate at regular intervals by calculating the dose rate @ 40ml/kg BW/day which didn't let the animal to go into hypoglycemia and dehydration state.

Osmotherapy found to be most rapid and effective way to decrease tissue water and brain bulk (Pollay, 1996). Mannitol is the most popular osmotic agent. Osmotic therapy using mannitol reduces intracranial pressure by mechanisms that remain unclear. Mannitol may also improve cerebral perfusion by decreasing viscosity or altering red blood cell rheology. Lastly, Mannitol may exert a protective effect against biochemical injury. IV Mannitol is given in the dosage of 1.0 g/kg, then 50 g every 2–3 hours (Davis and Lucatorto, 1994). The osmotic effect can be prolonged by the use of loop diuretics (Furosemide) after the osmotic agent infusion. Loop diuretics (Furosemide) can be used as an adjunct. Furosemide (0.7 mg/kg) has been shown to prolong the reversal of blood brain osmotic gradient established with the osmotic agents by preferentially excreting water over solute (Pollay, 1996). In fact, systemic complications of steroids can worsen the patient's condition (Rosenberg , 2000). Corticosteroids have not proven effective in stroke unless stroke is caused by documented cerebral vasculitis. Inj Dexamethasone 4–6 mg IM every 4–6 hours may be useful in these cases. They have also been used in chronic meningitis and in acute bacterial meningitis under cover of antibiotics. Edema surrounding brain tumours particularly metastatic brain tumours responds dramatically to treatment with high doses of Dexamethasone (Rosenberg, 2000). Glucocorticoids are believed to exert their influence on brain tumours mainly by reducing tumor–associated vasogenic edema, probably by decreasing the increased capillary permeability of blood brain barrier (BBB). The role of corticosteroids in head trauma is uncertain (Rosenberg, 2000). Other agents – Barbiturates, Procaine derivatives, Indomethacin, Propofol and THAM (Thrometamine) are some other agents which have been tried and used in the past but are not being used routinely in present practice (Richling, 1987). Barbiturates produce a marked decrease in metabolic rate and it seems likely that the fall in cerebral blood flow and intracranial pressure (ICP) is secondary. Heller et al., 1995 have shown that most newly diagnosed epileptic patients can be satisfactorily treated with a single antiepileptic drug that's nothing but the phenobarbitone. Recently, Satishchandra and Madhu Nagappa (2012) also mentioned the potential role of Phenobarbitone in clinical management of epileptic seizures.

The presented case could be a case of symptomatic epilepsy of any origin of cerebral edema, intracranial tumor or encephalitis. This case–report describes convulsive activity and successful recovery after combined therapy with Barbiturates, corticosteroids, diuretics and Mannitol. It is difficult to point out one single effective curable line of treatment in such cases. But they can be treated by using the same line of treatment. One has to monitor the fluid therapy carefully as the animal may go for dehydration due to use of diuretics

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