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THERAPEUTIC APPROACHES TO EPILEPTIC SEIZURES AND STATUS EPILEPTICUS IN DOGS

Abstract

Epilepsy is the most common neurological disorder in dogs. Status epilepticus (SE) is a serious and life-threatening neurological emergency in veterinary medicine, defined as sustained seizure activity or multiple consecutive seizures lasting longer than 30 minutes without complete recovery of consciousness in between. In some cases, SE becomes refractory to conventional treatment protocols and may result in severe outcomes, including death. This review aims to comprehensively evaluate both pharmacological and non-pharmacological treatment approaches used in the management of epileptic seizures and refractory status epilepticus in veterinary and human medicine. The objective is to provide current evidence-based information on effective seizure control, prevention of complications, and improvement of survival rates.

Keywords: status epilepticus, dog, treatment

Introduction. Dogs may experience sudden and short-lasting seizure activity. Status epilepticus is defined as continuous or recurrent seizure activity without full recovery of consciousness between episodes, lasting longer than 30 minutes, according to the International League Against Epilepsy (ILAE) and the American Epilepsy Society (AES) [1]. SE represents a critical clinical condition associated with high morbidity and mortality rates [2], with patients presenting with status epilepticus exhibiting a mortality rate ranging from 25.3% to 38.5% [1]. Regardless of etiology, any patient experiencing seizure activity may develop SE [3].

The frequency and severity of SE-associated complications are directly proportional to the duration of seizure activity; therefore, immediate termination of ongoing seizures is the primary therapeutic goal, followed by management of the underlying cause and associated complications [2, 4]. These complications broadly include primary and secondary permanent brain damage, hyperthermia, acidosis, disseminated intravascular coagulation, renal failure, and cardiovascular dysfunction [5, 6]. Experimental studies in rodents have demonstrated a direct relationship between seizure duration and the degree of neuronal injury, morbidity, and mortality [7].

Successful treatment of SE requires identification and management of the underlying etiology. Treatment protocols are determined according to the stage of the seizure; during ongoing seizure activity, treatment aims to terminate the seizure, whereas after seizure control, therapy is directed toward preventing recurrence [2]. In animals presenting with status epilepticus, therapeutic management is guided by the response to treatment and involves the sequential implementation of first-line, second-line, and third-line interventions, as well as alternative or non-conventional treatment approaches when indicated. (Table 1). In some animals, seizures may become resistant to standard antiepileptic drugs, and antiepileptic drug resistance represents a major obstacle to successful SE management [3]. Electroencephalography (EEG) represents a useful diagnostic and monitoring tool in the evaluation and management of drug-resistant epilepsy (Figure 1) [8].

Impending status epilepticus (stage 1) lasts 5-10 minutes and is generally recommended to be treated with first-line agents.

When seizure activity persists for 10-30 minutes, the patient is considered to be in established status epilepticus (stage 2). In this stage, first-line agents, non-anesthetic antiepileptic drugs, and/or general anesthetics may be used, although responsiveness to first-line therapy is reduced.

If seizures continue for more than 30 minutes the patient is classified as being in refractory status epilepticus (stage 3), and general anesthetic agents are required to suppress seizure activity. At this stage, the patient is resistant to first-line and non-anesthetic antiepileptic therapies [1, 4].

When seizure activity persists for more than 24 hours, the condition is classified as super-refractory status epilepticus (stage 4), and patients are typically resistant to all therapeutic agents [1, 2, 4, 9].

Non-pharmacological approaches include vagus nerve stimulation, deep brain stimulation, and transcranial magnetic stimulation. Modulation of neuronal activity through neurostimulation techniques is a rapidly evolving field, particularly in the treatment of drug-resistant epilepsy [10]. This review aims to examine current therapeutic strategies used to control seizures, prevent complications, and improve survival in epileptic seizures and refractory status epilepticus.

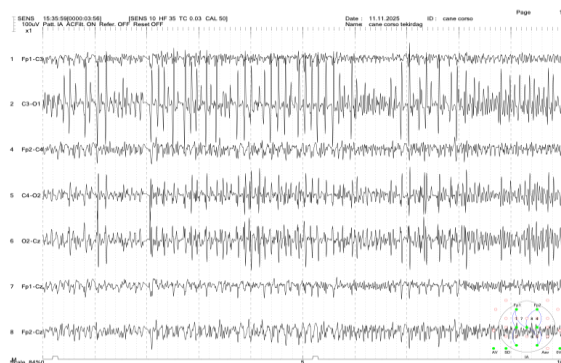
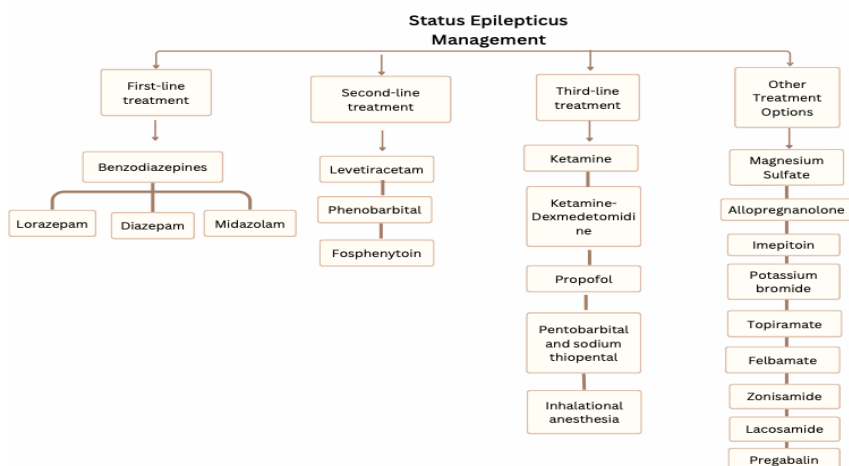


Figure 1. Electroencephalographic image demonstrating generalized seizure activity involving all channels in a 4-year-old Cane Corso dog with drug-resistant epilepsy.

Table 1. Pharmacological management of status epilepticus, including first, second and third-line and non-conventional options.



1.

Pharmacological treatment methods.

1.1. First-line therapy. In the initial management of seizures, bolus administration of benzodiazepines (BZDs) should be performed promptly. If seizures recur, a second bolus is administered, followed by a third bolus if necessary. If seizure activity persists, continuous rate infusion of benzodiazepines should be initiated, and second-line agents should be administered if seizures continue [1]. In veterinary medicine, midazolam, diazepam, and lorazepam are the most commonly used benzodiazepines [4].

1.1.1. Benzodiazepines. Benzodiazepines are widely used as first-line agents in the treatment of status epilepticus in both humans and dogs [10]. Benzodiazepines increase the effects of the neurotransmitter GABA by binding to GABA_A receptors [9, 11].

Midazolam has fewer adverse effects compared to other agents and is considered safe and reliable. It can be administered intranasally (IN), intravenously (IV), or intramuscularly (IM) at a dose of 0.2–0.5 mg/kg). Intranasal administration can be life-saving when IV access is not available. Studies in dogs have demonstrated that the IN

route is as effective and safe as IV administration and may even be superior in certain clinical settings [2, 10]. The drug has a half-life of 1 hour; therefore, frequent dosing or a constant-rate infusion may be required for seizure control [4].

Diazepam is administered as a bolus at a dose of 0.5–2.0 mg/kg IV, IN, or rectally ®. Repeated bolus administration may result in central nervous system depression and cardiorespiratory collapse. Therefore, diazepam boluses should be limited to 2–3 doses, followed by continuous rate infusion if necessary [4].

Lorazepam is more commonly used in human medicine, with limited studies in dogs. In canine studies, a dose of 0.2 mg/kg IV has been used [12]. Lorazepam binds more tightly to GABA receptors than diazepam, resulting in a longer duration of action [13].

1.2. Second-line therapy. Second-line treatment options include levetiracetam, barbiturates, and fosphenytoin [12].

1.2.1. Levetiracetam. Levetiracetam exerts its effect by binding to synaptic vesicle protein 2A (SV2A), modulating neurotransmitter release. In status epilepticus, it can be administered at a dose of 20–60 mg/kg via IV, IM, PO, or R routes. One study demonstrated that rectal administration of levetiracetam at 40 mg/kg achieved minimum therapeutic plasma concentrations [12].

1.2.2. Phenobarbital. Phenobarbital exerts antiepileptic effects by enhancing the function of GABA_A receptors and through its NMDA-antagonistic activity [11]. Distribution to the central nervous system (CNS) may take 20–30 minutes, potentially delaying onset of action. Phenobarbital may be administered IV at a total dose of 16–20 mg/kg, divided into three doses given every 30 minutes. Parenteral administration, particularly in patients previously treated with benzodiazepines, may cause significant hypotension and cardiorespiratory depression. Long-term use may result in sedation, ataxia, polydipsia, polyuria, hepatotoxicity, neutropenia, dyskinesia, thrombocytopenia, decreased T4 and fT4 levels, and superficial necrolytic dermatitis [1, 13].

1.2.3. Fosphenytoin. Fosphenytoin may be administered as an IV bolus in addition to other agents during status epilepticus [1]. Fosphenytoin exerts its effect by blocking voltage-gated sodium channels and suppressing high-frequency action potential discharges. Limited studies in dogs have reported successful treatment of SE with IV administration at a dose of 15 mg/kg. Adverse effects include cardiac arrhythmias and hypotension [14, 15].

1.3. Third-line therapy. Anesthetic agents are used in cases of status epilepticus refractory to other treatments. Commonly used agents in veterinary medicine include ketamine, propofol, pentobarbital, sodium thiopental, and inhalational anesthetics [1].

1.3.1. Ketamine. Ketamine acts as an NMDA receptor antagonist [6, 11]. It may be administered as a 5 mg/kg IV bolus, followed by a continuous infusion at 5 mg/kg/hour. Adverse effects include nystagmus, increased muscle tone, tachycardia, and hypersalivation [1, 16].

1.3.2. Ketamine–dexmedetomidine. When ketamine alone is insufficient to suppress seizures, dexmedetomidine may be added [1]. Dexmedetomidine reduces

sympathetic activity and norepinephrine release, thereby decreasing excitatory neurotransmitter levels. Additionally, its cerebral vasoconstrictive effects may help prevent cerebral edema. In three dogs diagnosed with idiopathic epilepsy, treatment with benzodiazepines and propofol administered for 12 hours was insufficient to terminate the seizures. In these cases, which progressed to refractory status epilepticus, ketamine was administered intravenously at a dose of 1 mg/kg over 5 minutes, followed by an IV bolus of dexmedetomidine at 3 µg/kg. Thereafter, continuous rate infusions were maintained for 12 hours with ketamine at 1 mg/kg/hour and dexmedetomidine at 3–7 µg/kg/hour. During treatment, body temperature was maintained at a mild hypothermic range (36.7–37.7 °C). No seizures were observed during the 48-hour follow-up period after discontinuation of therapy [1, 17].

1.3.3. Propofol. Propofol enhances GABA_A receptor activity and modulates NMDA receptors via sodium and calcium channels [1]. In dogs, it may be administered as a 1–2 mg/kg IV bolus, followed by continuous infusion up to 6 mg/kg/hour [1, 16]. Propofol infusion syndrome (PRIS) is a serious complication associated with prolonged high-dose infusions and may result in metabolic acidosis, hyperkalemia, bradyarrhythmias, myocardial depression and rhabdomyolysis [11, 18].

1.3.4. Pentobarbital and sodium thiopental. Anesthetic barbiturates act on GABA_A receptors and provide neuroprotective effects by reducing intracellular sodium and calcium, glutamate release, and cerebral oxygen consumption, while scavenging free oxygen radical [10]. They are administered at doses of 3–15 mg/kg IV, with caution to avoid overdose. Adverse effects include CNS and cardiorespiratory depression [1, 13].

1.3.5. Inhalational anesthesia. Inhalational anesthetics act on GABA_A receptors, reduce thalamic neuronal excitability, neurotransmitter release, and cerebral oxygen consumption, while increasing cerebral blood flow [1, 6].

1.4. Other treatment options.

1.4.1. Magnesium sulfate. Magnesium inhibits NMDA receptors and calcium channels and increases cerebral blood flow through vasodilation. It has been shown to be effective in hypomagnesemia-associated seizures and eclampsia in humans (Shorvon and Ferlisi, 2011). A canine case report suggests potential benefits of IV magnesium in SE, although adverse effects include CNS depression, cardiorespiratory depression, and hypotension [1, 19].

1.4.2. Allopregnanolone. Allopregnanolone is a neurosteroid acting on synaptic and extrasynaptic GABA receptors. It has been shown to terminate SE in rodent models and may represent an effective treatment for benzodiazepine-resistant SE in dogs [1, 20, 21].

1.4.3. Imepitoin. Imepitoin enhances GABA activities and weakly blocks calcium channels. It is a potent anticonvulsant and anxiolytic for dogs. Although recommended for dogs with recurrent generalized seizures, its efficacy in canine SE has not yet been established. The recommended dosage is 10–30 mg/kg twice daily [22, 23].

1.4.4. Potassium bromide. Potassium bromide has a synergistic effect with phenobarbital and is not metabolized by the liver, making it suitable for dogs with hepatic disease. It is administered at 20 mg/kg BID as monotherapy or 15 mg/kg BID when used in combination therapy [23].

1.4.5. Topiramate. Topiramate is a second-generation, broad-spectrum antiepileptic drug that inhibits voltage-gated sodium channels and enhances GABAA activity. Topiramate is used in humans for the treatment of both focal and generalized seizures, either as monotherapy or in combination with other antiepileptic drugs. Studies in dogs have demonstrated that IV doses of 10 and 20 mg/kg are safe and well tolerated [23, 24].

1.4.6. Felbamate. Felbamate inhibits NMDA receptor activity, enhances GABA activity, and has neuroprotective properties. In one study, felbamate treatment was administered to six dogs with a history of seizures lasting longer than six months and cluster seizures, at a dose of 15–20 mg/kg: two dogs received the drug every 8 hours and four dogs every 12 hours. Seizure frequency was evaluated using a short-term follow-up of 3 months and a long-term follow-up of 6 months. Although conducted in a small number of animals, this study demonstrated that felbamate may be used as an adjunctive therapy in patients with idiopathic epilepsy [25]. During felbamate treatment, routine complete blood count and biochemical analyses should be performed. As the drug is metabolized by the liver, it should not be used in patients with hepatic insufficiency [23, 26]. Felbamate may cause mild blood dyscrasias and keratoconjunctivitis sicca [27].

1.4.7. Zonisamide. Zonisamide is a sulfonamide-based drug that blocks voltage-dependent sodium channels and inhibits T-type calcium channels [28, 29, 30]. In dogs, it is administered orally at 5–10 mg/kg every 12 hours [22]. In a study conducted on dogs with idiopathic epilepsy, the addition of zonisamide to phenobarbital and potassium bromide therapy resulted in a 58% reduction in seizure frequency [31].

1.4.8. Etomidate. Etomidate is a GABA_A agonist anesthetic agent used in refractory SE in humans. It reduces cerebral oxygen consumption and intracranial pressure and may be administered at 1–2 mg/kg IV followed by continuous infusion at 0.5–2 mg/kg/hour. It has less respiratory and central nervous system depression and fewer cardiovascular side effects compared to propofol and barbiturates [13, 32].

1.4.9. Lacosamide. Lacosamide exerts its effect by selectively enhancing the slow inactivation of voltage-gated sodium channels, thereby stabilizing hyperexcitable neuronal membranes and inhibiting neuronal firing [33]. It is also a drug that can be used in the treatment of neuropathic pain and chronic inflammatory pain. An initial dose of 50–100 mg every 12 hours is recommended. Lacosamide may be administered as an adjunctive therapy when other antiseizure medications have failed [28].

1.4.10. Pregabalin. It is a second-generation GABA analogue, similar to gabapentin, with antiepileptic, analgesic, and anxiolytic effects. Because its half-life and bioavailability are higher than those of gabapentin, its antiseizure efficacy is greater [28]. In a study conducted in 11 dogs, combination therapy with phenobarbital, potassium bromide, and pregabalin resulted in a reduction in seizure frequency in 9

dogs. Reported adverse effects in dogs included sedation, ataxia, and dizziness [34]. In dogs, a dose of 4 mg/kg PO every 8 hours is recommended [28].

2. Non-pharmacological treatment methods.

2.1. Neurostimulation techniques. Vagus nerve stimulation, deep brain stimulation, and transcranial magnetic stimulation are emerging therapeutic modalities in veterinary medicine. While pharmacological approaches remain the cornerstone of treatment, neurostimulation techniques represent promising alternatives for drug-resistant epilepsy, despite challenges related to cost, technical complexity, and expertise requirements [19].

2.1.1. *Vagus nerve stimulation.* Vagus nerve stimulation (VNS) is a technique that can be applied either by exerting pressure on the orbit or by implanting a device that delivers electrical stimulation [28]. VNS stimulates the release of neurotransmitters, which in turn promotes the release of GABA, thereby increasing the seizure threshold of neurons [19]. In humans treated with VNS, reductions in seizure frequency, seizure duration, and ictal period have been reported [35]. VNS has also been shown to reduce seizure frequency in some dogs with idiopathic drug-resistant epilepsy [36]. In dogs, the electrodes are wrapped around the left vagosympathetic trunk [19]. Reported post-implantation complications associated with VNS include seroma formation at the implantation site, coughing, and muscle twitching [37, 38].

In dogs, ocular compression performed by applying pulsatile compression to the orbit for 10–60 seconds every 5 minutes has been shown to reduce seizure severity and duration as a non-invasive VNS technique [39].

2.1.2. *Deep brain stimulation.* Deep brain stimulation (DBS) is a neurostimulation technique in which a device is implanted bilaterally into the centromedian nucleus of the thalamus [40]. In a study conducted at the University of Melbourne, deep brain stimulation devices were successfully implanted in dogs [41]. In another study, DBS therapy was applied to a 4-year-old mixed-breed dog with drug-resistant epilepsy, and a reduction in seizure severity was observed following stimulation; however, to adequately assess its outcomes in veterinary medicine, DBS needs to be evaluated in a larger number of animals [40].

2.1.3. *Transcranial magnetic stimulation.* Repetitive transcranial magnetic stimulation (rTMS) is a noninvasive, extracranial technique that modulates cortical excitability by using sequences of magnetic stimuli generated by a coil placed over the cranial region [37]. A rapidly changing magnetic field is used to induce weak electrical currents in the brain, leading to neuronal depolarization and the generation of action potentials. rTMS can be used to measure cortical excitability and to evaluate the efficacy of antiepileptic drugs [42]. The frequency and stimulation parameters of rTMS are key determinants of its effects. Low-frequency stimulation has anticonvulsant effects, whereas high-frequency stimulation may be proconvulsant [38, 43].

In dogs, TMS is primarily used as a diagnostic tool to assess the integrity of the motor cortex and spinal cord. In a study involving nine dogs with refractory epilepsy, a short-term improvement in interictal interval was observed following TMS compared

with the pre-treatment period. Overall, six of the nine epileptic dogs exhibited a reduction in seizure frequency after TMS, and an increase in the number of seizure-free days was reported [37, 42].

2.2. Hypothermia. Hypothermia reduces brain activity and ATP consumption, and decreases glutamate release, free radical production, oxidative stress, mitochondrial dysfunction, and calcium overload. It inhibits cellular apoptosis by promoting the release of neurotrophic factors. Hypothermia also reduces inflammation and decreases the permeability of the blood–brain barrier [2, 44, 45]. During status SE, the application of hypothermia prevents neuronal loss and reduces both the duration and frequency of seizures [46]. Studies support the anticonvulsant and neuroprotective effects of hypothermia [44, 47].

2.3. Electroconvulsive therapy. Electroconvulsive therapy (ECT) operates via transcutaneous electrical stimulation of the cerebral cortex and is used in the treatment of refractory psychiatric disorders by inducing a seizure-like episode. The exact mechanism of action of ECT, including its effectiveness in terminating SE, is not fully understood. ECT is thought to induce post-treatment alterations in neurotransmitter levels, with increased GABA activity mediating inhibition of action potential propagation. In human studies, ECT has been reported to achieve a success rate of 59% in 27 patients with SE [48, 49, 50].

Conclusion. SE is a common emergency in dogs with complex pathophysiology and is difficult to manage. Traditionally, SE is controlled using first-line therapy, second-line therapy, and anesthetic agents. However, the management of seizures that develop resistance to current treatments has become a challenge for veterinarians. Therefore, it is necessary to investigate new treatment modalities in terms of their efficacy, adverse effects, and their ability to suppress or reduce seizure frequency. Medical management can be particularly difficult to implement for animals with persistent and drug-resistant epilepsy and for their owners. Studies have shown that non-medical treatment options may also be feasible and effective. Nevertheless, there is a need to evaluate treatment efficacy in larger patient populations.

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**ТЕРАПЕВТИЧНІ ПІДХОДИ ДО ЛІКУВАННЯ ЕПІЛЕПТИЧНИХ НАПАДІВ ТА
ЕПІЛЕПТИЧНОГО СТАТУСУ У СОБАК**

Анотація

Епілепсія є найпоширенішим неврологічним захворюванням у собак. Епілептичний статус (*status epilepticus*, SE) – це тяжкий, потенційно загрозливий для життя невідкладний неврологічний стан у ветеринарній медицині, що характеризується безперервною судомною активністю або серією послідовних епілептичних нападів тривалістю понад 30 хвилин без повного відновлення свідомості між ними. У частини пацієнтів епілептичний статус набуває рефрактерного перебігу, не відповідає на стандартні протисудомні схеми лікування та може призводити до розвитку тяжких ускладнень, включаючи летальний наслідок. Метою цього

огляду є комплексний аналіз сучасних фармакологічних і нефармакологічних підходів до лікування епілептичних нападів та рефрактерного епілептичного статусу, що застосовуються у ветеринарній і гуманній медицині. Особливу увагу приділено сучасним доказовим даним щодо ефективного контролю судомної активності, профілактики ускладнень, підвищення виживаності пацієнтів та покращення результатів лікування.

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