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# Paroxysmal dyskinesia in 31 dogs (2020–2025)[1]

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**ABSTRACT.-** Rauber JS, Chaves JNF, Chaves RO, Sekita AMT, Soares TS, Pozzolo AAD, Polidoro DN, Wrzesinski MR, Mielke JFS, Beckmann DV, Mazzanti A. **Paroxysmal dyskinesia in 31 dogs (2020–2025)**. *Pesquisa Veterinária Brasileira 46:e07719, 2026*. Serviço de Neurologia e Neurocirurgia Veterinária, Departamento de Clínica de Pequenos Animais, Universidade Federal de Santa Maria, Av. Roraima 1000, Santa Maria, RS 97105-900, Brazil. E-mail: alexamazza@yahoo.com.br

This retrospective study aimed to identify dogs with a presumptive diagnosis of paroxysmal dyskinesia (PD) and describe its phenotypical characterization, treatment, and outcome. A total of 31 dogs (45,2% females and 54,8% males) examined at two veterinary neurology services from January 2020 to April 2025 were included. The mean age at clinical onset was 52.2 months (range, 2–137 months). Nine dogs were mixed-breed (29%), eight were German Spitz (25.8%), four were Dachshunds (12.9%), two were Chihuahuas (6.4%), two were Shih Tzus (6.4%), and one each were Siberian Husky, Labrador Retriever, Pinscher, and Maltese (3.2% each). A presumptive diagnosis was established based on independent analysis of the video recordings of the episodes. Limb dystonia was present in all dogs, whereas associated dystonic head tremors were present in 61.3% of the cases. Other clinical signs included generalized tremors (38.7%), kyphosis (32.2%), and incoordination (12.9%). The mean episode duration was 6.8 minutes (range, 1–60 minutes). Triggers such as stress and/or excitement were identified in 15 dogs (48.4%). Seventeen dogs (54.8%) were treated for the episodes: 11 with antiseizure drugs (54.5% improvement) and 6 with a gluten-free diet (66.7% improvement). Fourteen dogs (45.2%) did not receive any treatment, and spontaneous remission was seen in six (42.8%). Paroxysmal dyskinesia can affect dogs of various breeds and ages with no sex predilection. The disorder manifests as a range of clinical signs, including limb dystonia, which was present in all dogs in this study. Stress and excitement were identified as the main triggers in nearly half of the cases. Both the treated and untreated dogs showed clinical improvement, highlighting the heterogeneity of PD. Further studies are needed to better understand the disease and its progression.

INDEX TERMS: Movement disorder, dystonia, stress, tremor, dog.

**RESUMO.-** [**Discinesia paroxística em 31 cães (2020–2025).**] O objetivo deste estudo retrospectivo foi identificar cães com diagnóstico presuntivo de discinesia paroxística (DP) e descrever a caracterização fenotípica, o tratamento e os desfechos clínicos. Um total de 31 cães (45,2% fêmeas e 54,8% machos) foram incluídos, atendidos em dois serviços de neurologia veterinária de janeiro de 2020 a abril de 2025. A idade média de início clínico foi de 52,2 meses (variação: 2 a 137 meses). Nove cães eram sem raça definida (29%), oito Spitz Alemão (25,8%), quatro Dachshunds (12,9%), dois Chihuahuas (6,4%), dois Shih Tzus (6,4%) e um de cada das seguintes raças: Husky Siberiano, Labrador Retriever, Pinscher e Maltês (3,2% cada). O diagnóstico presuntivo foi estabelecido com base na análise independente das gravações em vídeo dos episódios. Distonia dos membros esteve presente em 100% dos casos, enquanto tremor distônico da cabeça foi observado em 61,3%. Outros sinais clínicos observados incluíram tremores generalizados (38,7%), cifose (32,2%) e

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incoordenação (12,9%). A duração média dos episódios foi de 6,8 minutos (variação: 1 a 60 minutos). Fatores desencadeantes, como estresse e/ou excitação, foram identificados em 15 cães (48,4%). Dezessete cães (54,8%) foram tratados para os episódios — 11 com medicamentos anticonvulsivantes (dos quais 54,5% apresentaram melhora) e seis com dieta sem glúten (66,7% apresentaram melhora). Quatorze cães (45,2%) não receberam nenhum tratamento e a remissão espontânea foi observada em seis deles (42,8%). A discinesia paroxística pode afetar cães de diversas raças e idades, sem predileção por sexo. Manifesta-se com uma variedade de sinais clínicos, incluindo distonia dos membros, presente em todos os cães deste estudo. Estresse e excitação foram os principais gatilhos, identificados em quase metade dos casos. Tanto os cães tratados quanto os não tratados apresentaram melhora clínica, destacando a heterogeneidade da DP e a necessidade de mais estudos para melhor compreender a doença e sua progressão.

TERMOS DE INDEXAÇÃO: Distúrbio de movimento, distonia, estresse, excitação, cão.

## INTRODUCTION

Paroxysmal dyskinesia (PD) is a movement disorder characterized by involuntary, self-limiting, recurrent episodes without loss of consciousness and absence of pre- or postictal phases (Cerda-Gonzalez et al. 2021, Santifort & Mandigers 2022). In dogs, PD has been increasingly recognized in clinical practice, partly because of video recordings made possible by the greater accessibility of smartphones, which help differentiate it from other conditions, particularly epileptic seizures (ES) (Mandigers et al. 2024, Matz et al. 2025).

The pathophysiology of movement disorders involves dysfunction of the basal nuclei, which are important structures that connect the thalamus and cerebral cortex through motor circuits. These circuits are modulated by neurotransmitters, especially dopamine, which stimulates the direct pathway and inhibits the indirect pathway, thereby facilitating movement. In addition, the cerebellum is believed to play a role in the pathophysiology of PD by influencing communication between the thalamus, cortex, and basal nuclei (Kaji et al. 2017, Tewari et al. 2017). Alterations in these communications may impair transmission within motor circuits, resulting in abnormal movement characteristics in movement disorders such as PD (Obeso et al. 2002, Cerda-Gonzalez et al. 2021).

Based on its etiology, PD can be classified as genetic, reactive (intoxications, metabolic diseases, drug-induced, or diet-related), structural (neoplasms, inflammatory diseases, or secondary to other structural causes), or of unknown cause (Mandigers et al. 2024).

The treatment of PD is often empirical and based on trial and error (Mandigers et al. 2024), with variable responses to different therapies, even among dogs of the same breed, such as Soft-Coated Wheaten Terriers (Packer et al. 2021), Norwich Terriers (De Risio et al. 2016), Maltese (Polidoro et al. 2020), and Welsh Terriers (Whittaker et al. 2022). Isolated case reports involving Welsh Terriers (Green & Olby 2021) and German Shorthaired Pointers (Harcourt-Brown 2008) have demonstrated the influence of antiepileptic drugs in reducing episodes.

In addition to pharmacological therapies, some animals with PD may benefit from the introduction of a gluten-free diet, as first observed in Border Terriers (Lowrie et al. 2015, 2016, 2018), or simply from routine changes, as reported in Scottish Terriers (Urkasemsin & Olby 2015). This may be attributed to triggers such as stress, excitement, and physical exercise that precipitate episodes (Urkasemsin & Olby 2014, Cerda-Gonzalez et al. 2021, Mandigers et al. 2024). Nevertheless, the study by Lowrie & Garosi (2016) demonstrated the self-limiting nature of this disease in Jack Russell Terriers and Labrador Retrievers, raising the hypothesis that this characteristic may also be present in dogs of other breeds and reinforcing the importance of monitoring the natural course of the disease in animals diagnosed with PD.

Although studies have contributed to the identification, understanding, and management of PD in dogs, most have been based on samples from a single breed, generally with a suspected or confirmed genetic predisposition (Mandigers et al. 2024). This has advanced knowledge of PD in specific populations but limits understanding of the disease in a broader context, such as in dogs of breeds other than those previously studied or in populations encompassing multiple breeds. To date, no studies have been conducted on dogs with PD in Brazil.

In this context, this retrospective study aimed to identify dogs evaluated from January 2020 to April 2025 at two veterinary neurology services in Brazil with a presumptive diagnosis of PD and to obtain information regarding breed, sex, age, phenotypic characterization of episodes, treatment, and clinical response.

## MATERIALS AND METHODS

**Ethical approval.** Since this is a retrospective study, ethics committee approval was not required.

The medical records of dogs evaluated from January 2020 to April 2025 with suspected PD were reviewed from the "*Serviço de Neurologia e Neurocirurgia Veterinária*" (Veterinary Neurology and Neurosurgery Service – SNNV) of a higher education institution and from a private referral center specializing in veterinary neurology.

Dogs were included if a complete neurological record containing information on breed, age, sex, clinical history, neurological examination, and description of episodes was available. In addition, video recordings of the episodes provided by the owners were required, as well as a minimum follow-up period of 3 months after the first consultation.

Laboratory tests (complete blood count, serum biochemistry, serology and polymerase chain reaction – PCR for major infectious diseases, and cerebrospinal fluid analysis in dogs with suspected PD) were required to be within the reference range for the species. Brain imaging studies (magnetic resonance imaging – MRI or computed tomography – CT) were also required to reveal no abnormalities.

Once dogs with suspected PD were identified, in the first stage, the presumptive diagnosis of PD was determined based solely on independent video evaluations by two veterinarians: one board-certified by the European College of Veterinary Neurology (ECVN) and the other with more than 20 years of experience in the field. The episodes were classified by the evaluators as probable PD, unlikely PD, or inconclusive.

The category "probable PD" was assigned to dogs showing characteristic signs of PD, defined by evident dysregulation of muscle tone (Santifort & Mandigers 2022) involving the pelvic and/or thoracic limbs, without loss of consciousness, without autonomic signs, and

without pre- or postictal phases (Lowrie & Garosi 2017, Polidoro et al. 2020, Mandigers et al. 2024). The classification "unlikely PD" was used when the observed clinical signs were not compatible with PD. The category "inconclusive" was assigned when the videos did not provide sufficient information to establish a presumptive diagnosis of PD.

A presumptive diagnosis of PD was made when both evaluators classified the episodes as probable PD. In cases where one evaluator assigned "probable PD" and the other "inconclusive," or when both defined the episodes as inconclusive, a second stage of evaluation was conducted, which included complementary analysis of the dog's neurological record made available to the evaluators. The information analyzed primarily concerned the episode duration, preservation of consciousness, absence of autonomic signs, and presence of triggers. In addition, owners were asked to provide supplementary videos of the episodes.

Triggers included episodes of excitement and stress. Excitement was defined as a response to positive stimuli that agitated the animal, such as the owner's arrival at home or moments preceding walks. Stress was characterized by negative situations that cause agitation, such as changes in routine, owners leaving home, or the absence of individuals with whom the animal regularly interacted.

The results were analyzed using descriptive statistics and expressed as percentages. The evaluators' video classifications were tabulated, and interobserver agreement was assessed using Cohen's kappa test. The clinical responses in the treated and untreated patients were analyzed using Fisher's exact test, with a significance level of $p < 0.05$.

## RESULTS

Fifty neurological records of dogs suspected of having PD were analyzed. After applying the previously established inclusion criteria, 46 videos were selected for the first-stage evaluation. Of these, 25 (54.3%) received a presumptive diagnosis of PD, as both evaluators classified the episodes as "probable PD." Ten dogs were excluded because their videos were classified as "unlikely PD" by both evaluators or as "inconclusive" by one and "unlikely PD" by the other. The remaining 11 dogs were referred for a second-stage evaluation due to discordant classifications between evaluators ("probable PD" by one and "inconclusive" by the other). In this second stage, following analysis of the neurological records and supplementary videos, six dogs were reclassified as "probable PD" and included, while five were excluded (Fig. 1).

Thus, 31 dogs with a presumptive diagnosis of PD, corresponding to a prevalence of 0.9% among the cases evaluated during the analysis period, were included in the study. Seventeen animals were male (54.8%), and 14 were female (45.2%). Regarding animal breed, nine (29%) were mixed-breed, eight (25.8%) were German Spitz, four (12.9%) were Dachshunds, two each (6.4%) were Chihuahuas, Shih Tzus, and Yorkshire Terriers, and one (3.2%) each was a Siberian Husky, Labrador Retriever, Pinscher, and Maltese (Table 1). The mean age at the onset of clinical signs ranged from 2 to 137 months, with a mean of 52.2 months. The follow-up period ranged from 3 to 45 months

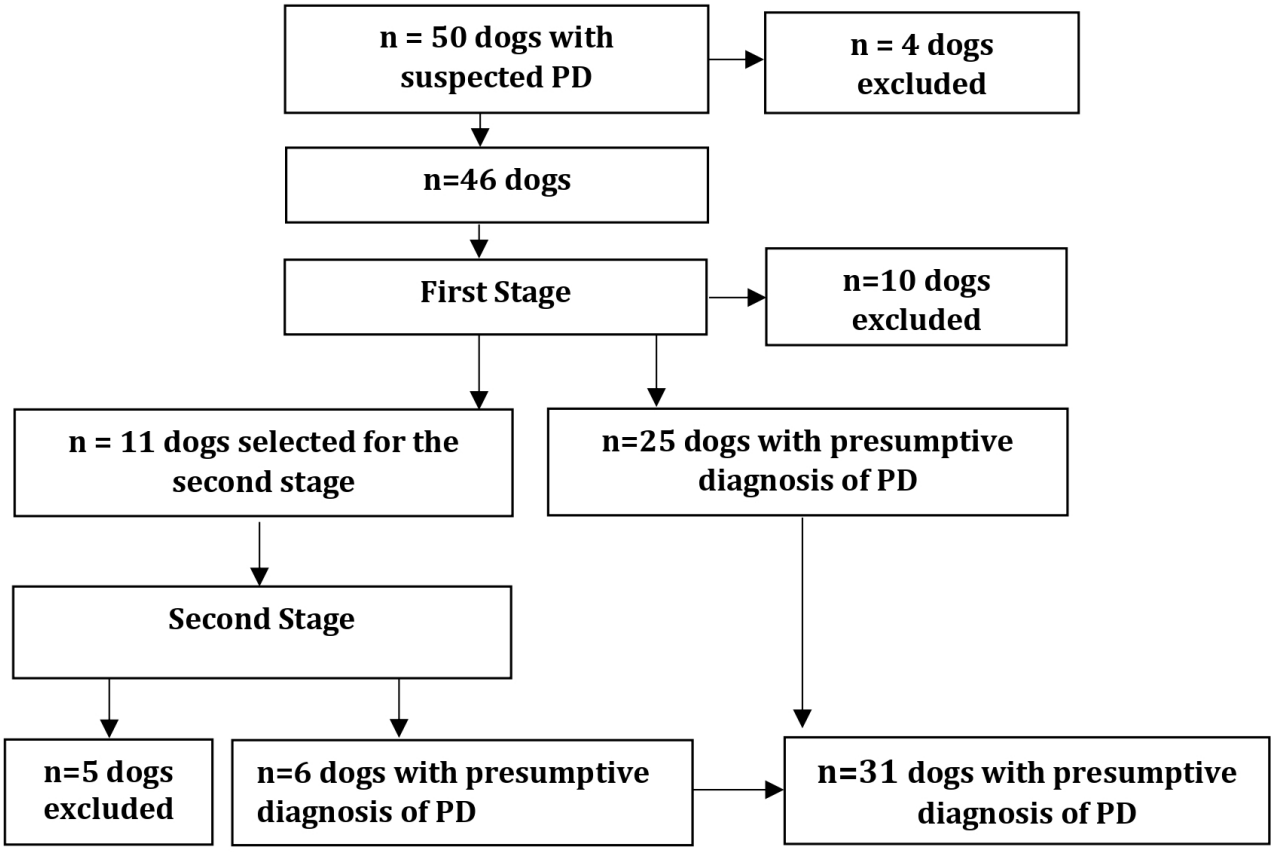


Fig. 1. Flowchart of case selection and diagnostic classification of dogs with a presumptive diagnosis of paroxysmal dyskinesia.

with a mean of 13.4 months. Updated information could not be obtained for five dogs because of their inability to contact their owners. Therefore, the current seizure frequency (April 2025) was available for 26 of 31 (83.9%) animals.

Dystonia involving the thoracic and/or pelvic limbs was present in all cases (100%). Dystonia also affected the cervical and head regions in 19 dogs (61.3 %). Kyphosis was identified in 10 of the 31 dogs (32.2%), generalized tremors in 12 (38.7%), dystonic head tremors in one (3.2%), and incoordination in four (12.9%) (Table 1, Video S1).

Episode duration ranged from 1 to 60 minutes, with a mean of 6.8 minutes. Triggers of episode onset were identified in 15 dogs (48.4%); stress was the sole trigger in four (12.9%) and excitement in five (16.1%). In six dogs (19.3%), both were considered triggers by the owners (Table 1).

Complementary diagnostic tests were performed in five dogs (16.1%): brain CT in two, cerebrospinal fluid analysis in two, and brain MRI in one. No abnormalities were identified in any case.

Of the 31 dogs included in this study, 17 (54.8%) received treatment during the episodes. A gluten-free diet was recommended to six dogs (35.3 %), and antiepileptic drugs were administered to 11 (64.7 %). None of the dogs received any of the treatments simultaneously. In four cases, updated information on episode frequency was unavailable due to a lack of follow-up and an inability to contact the owners.

Among the 11 dogs treated with antiepileptic drugs, 9 (81.8%) received phenobarbital alone, and 2 (18.2%) received a combination of phenobarbital and potassium bromide. Of these, six (54.5%) showed a reduction in episode frequency, two (18.2%) showed no change, and updated information on episode frequency was unavailable for three (27.3%). None of the dogs showed an increase in episode frequency after initiation of antiepileptic therapy.

Among the six dogs managed with a gluten-free diet, four (66.7%) showed a reduction in episode frequency, one (16.6%) experienced an increase, and one (16.6%) maintained the same episode frequency as before treatment.

**Table 1. Distribution according to breed, sex, age, clinical signs, duration of episodes, presence of trigger, and response to treatment in 31 dogs with a presumptive diagnosis of paroxysmal dyskinesia**

| Dog | Breed, sex, age (month) | Clinical signs | Duration (minute) | Trigger | Treatment | Response |
|---|---|---|---|---|---|---|
| 1 | German Spitz, M, 48 | K, DT (PL), AI | 5 | S | NT | CR |
| 2 | Pinscher, F, 36 | TR, DT (H, TL, PL) | 3 | S/Ex | AE | CR |
| 3 | Mixed Breed, M, 96 | K, DT (H, TL), AI | 2 | NO | GFD | CR |
| 4 | Mixed Breed, M, 4 | DT (H, TL, PL) | 3 | Ex | GFD | IC |
| 5 | Mixed Breed, F, 60 | DT (H, TL, PL) | 5 | S/Ex | GFD | CR |
| 6 | Shih Tzu, M, 48 | K, DT (H, PL) | 2 | Ex | GFD | CR |
| 7 | Dachshund, M, 2 | TR, DT (H, TL, PL) | 60 | NO | NT | NC |
| 8 | Mixed Breed, M, 36 | DT (TL) | 10 | S/Ex | NT | IC |
| 9 | Dachshund, F, 36 | DT (H, TL), TR | 60 | NO | NT | NI |
| 10 | Dachshund, M, 5 | K, TR, DT (H, TL) | 3 | S/Ex | NT | CR |
| 11 | Maltese, M, 116 | K, DT (TL, PL) | 2 | S/Ex | AE | CR |
| 12 | German Spitz, F, 44 | K, TR, DT (TL) | 5 | NO | NT | CR |
| 13 | German Spitz, F, 31 | TR, DT (H, TL) | 3 | S/Ex | NT | IC |
| 14 | Mixed Breed, M, 115 | DT (TL, PL) | 2 | NO | NT | NC |
| 15 | Chihuahua, M, 45 | DT (TL, H), AI | 5 | Ex | ST | CR |
| 16 | Dachshund, F, 137 | K, DT (TL), AI | 8 | S | GFD | CR |
| 17 | Chihuahua, F, 24 | TR, DT (H, TL) | 1 | NO | AE | CR |
| 18 | German Spitz, F, 5 | TR, DT (H, PL) | 2 | NO | AE | NI |
| 19 | Mixed Breed, F, 96 | TR, DT (H, TL) | 1 | NO | AE | NI |
| 20 | Mixed Breed, M, 24 | DT (H, TL, PL) | 1 | S/Ex | AE | CR |
| 21 | Yorkshire, M, 48 | K, TR, DT (PL) | 30 | Ex | NT | NI |
| 22 | Labrador, F, 60 | DT (H, TL) | 10 | NO | NT | CR |
| 23 | German Spitz, F, 21 | DT (H, TL, PL) | 3 | NO | AE | NC |
| 24 | German Spitz, F, 72 | DT (TL, PL) | 1 | NO | NT | NC |
| 25 | Yorkshire, M, 96 | DT (PL) | 5 | NO | GFD | NC |
| 26 | German Spitz, M, 96 | K, DT (TL) | 2 | S | AE | CR |
| 27 | Siberian Husky, M, 32 | DT (H, TL) | 1 | S | NT | NC |
| 28 | German Spitz, M, 60 | TR, DT (TL) | 1 | NO | AE | NC |
| 29 | Shih Tzu, M, 6 | DT (PL) | 1 | NO | NT | CR |
| 30 | Mixed Breed, F, 60 | TR, DT (H, TL) | 4 | NO | AE | NI |
| 31 | Mixed Breed, F, 60 | DT (H, TL, PL) | 2 | S | AE | CR |

M = male, F = female, K = kyphosis, DT = dystonia, PL = pelvic limbs, AI = ataxia/incoordination, TR = tremors, H = head, TL = thoracic limbs, S = stress, Ex = excitement, NO = not observed, NT = no treatment, AE = antiepileptic, GFD = gluten-free diet, CR = crisis reduction, IC = increase crisis, NC = no change, NI = no information.

Of the 14 dogs (45.2%) that did not receive treatment, six (42.8%) showed spontaneous improvement, four (28.5%) maintained the same episode frequency, two (14.3%) experienced an increase, and in two (14.3%), updated information regarding episode frequency was unavailable.

## DISCUSSION

The total number of dogs with neurological disorders evaluated at the two neurology centers from January 2020 to April 2025 was 4,218, resulting in a PD prevalence of 0.9% (31/4,218). To date, no study has determined the prevalence of PD in heterogeneous canine populations from multiple breeds.

Previous studies have predominantly focused on specific breeds with suspected or confirmed genetic predisposition, such as Norwich Terriers (De Risio et al. 2016), Welsh Terriers (Whittaker et al. 2022), Cavalier King Charles Spaniels (Gill et al. 2012), Soft-Coated Wheaten Terriers (Kolicheski et al. 2017), Shetland Sheepdogs (Nessler et al. 2020), Markiesjes (Mandigers et al. 2021), and Weimaraners (Christen et al. 2023). Although this breed-focused approach contributes to the understanding, characterization, and treatment decisions within specific populations, it raises questions. It limits the comprehension of the broader clinical and etiological spectrum of PD in other breeds and mixed-breed dogs.

Among the purebred dogs included in this study, the German Spitz was the most common breed, representing 25.8% (8/31). Evidence suggests that PD in this breed may be associated with gluten sensitivity, as proposed by Rogers et al. (2023), who identified positive serology in two dogs. Case reports by Baptista da Silva et al. (2023) and Kim et al. (2024) further supported this hypothesis by documenting clinical improvements after the introduction of a gluten-free diet. In the present study, none of the German Spitz dogs received a gluten-free diet; however, three showed a reduction in episode frequency: two without any therapeutic intervention and one treated with phenobarbital. In the latter case, even three months after complete discontinuation of the drug, the dog remained episode-free.

The favorable response to phenobarbital in this dog initially raised questions regarding the diagnosis of PD, particularly considering focal motor epileptic seizures as the main differential diagnosis. This uncertainty is reinforced by the findings of Yu et al. (2022), who predominantly described focal motor episodes in German Spitz dogs with epilepsy, wherein limb dystonia was the most frequent clinical sign. The authors referred to these events as "dystonic epileptic seizures," a term originally used in human medicine to describe focal seizures originating in the frontal lobes and characterized by dystonic motor manifestations (Bonelli et al. 2007).

However, in the present study, the diagnosis of PD in dogs that responded to phenobarbital was based on the clinical features observed during the episodes, which aligned with the definition proposed by Berendt et al. (2015) in the International Veterinary Epilepsy Task Force Consensus Statement. This definition describes focal motor epileptic seizures as "episodic focal motor phenomena, such as facial spasms, repetitive head-shaking movements, rhythmic blinking, facial muscle contractions, or repeated rhythmic jerking of one limb." In the present study, the episodes observed in German Spitz dogs, characterized mainly by evident limb dystonia, did not exhibit the typical focal motor pattern described for epileptic seizures and were considered more compatible with the hyperkinetic manifestations of PD.

Although some dogs in the study by Yu et al. (2022) underwent electroencephalography (EEG), not all were evaluated using this modality, limiting definitive confirmation of the epileptic nature of the observed events. In humans, interictal EEG recordings have demonstrated electrical discharges originating from the motor cortex in patients diagnosed with PD (Ohmori et al. 2002, Van Strien et al. 2012). Functional communication between the cerebral cortex and basal nuclei, as well as their potential pathologies and clinical manifestations, remains incompletely elucidated in human medicine (Gusmão et al. 2021), and even less so in veterinary medicine. Therefore, the clinical presentation of PD in dogs, which is distinct from the focal motor seizure pattern described by Berendt et al. (2015), remains fundamental for defining the disease. Even in cases where EEG findings suggest epileptic activity, the possibility that some clinical manifestations observed in dogs may represent a seizure type that has not yet been fully characterized cannot be excluded, reinforcing the need for further investigation into the underlying neurophysiological mechanisms.

The presumptive diagnosis of PD was established based on video analysis of episodes by two evaluators. Inter-observer agreement yielded a kappa value of 0.8884, indicating almost perfect agreement (Landis & Koch 1977). This high level of concordance was likely attributable to the evaluators' experience, as one was a board-certified neurologist (at ECVN), and the other had 20 years of experience in neurology. In contrast, another study comparing experienced and inexperienced evaluators reported a low agreement (Packer et al. 2021). Establishing a presumptive diagnosis of PD based on video analysis and clinical history is widely accepted in the literature (Matz et al. 2025). This approach has been adopted in several studies, emphasizing preservation of consciousness and a normal neurological examination as key criteria for differentiating PD from other episodic disorders, particularly epileptic seizures (De Risio et al. 2016, Lowrie & Garosi 2017, Lowrie et al. 2018, Polidoro et al. 2020, Rogers et al. 2023).

The second stage of evaluation was necessary for 11 dogs, and allowed for inclusion and presumptive diagnosis in 54.5% (6/11). At this stage, complementary videos and full-episode recordings were reviewed, and neurological records were made available. The combination of these data facilitated the inclusion of additional dogs, with episode duration being one of the decisive elements, as 50% (3/6) included in the second stage had episodes lasting 5 minutes or longer.

The mean episode duration in this study was 6.8 minutes. Whittaker et al. (2022) reported a mean of 4 minutes in Welsh Terriers, and Polidoro et al. (2020) reported a mean of 4.5 minutes in Maltese dogs. Episode duration contributed to inclusion or exclusion decisions in 63.6% (7/11) of cases, particularly in differentiating PD from epileptic seizures, which typically last up to 2 minutes (Kähn et al. 2024). Episodes lasting 5 minutes or longer with immediate or near-immediate recovery (absence of a postictal phase) were considered suggestive of PD. Conversely, short episodes (1–3 minutes) supported the exclusion of doubtful cases where the differentiation between PD and epilepsy was uncertain. In epileptic seizures, prolonged episodes are expected to result in more pronounced postictal signs, such as confusion, lethargy, blindness, and ataxia (Hülsmeyer et al. 2015, Nagendran et al. 2025), given the sustained convulsive activity. Although episode duration was not the sole diagnostic criterion, it served as a supportive factor when analyzed alongside other elements

such as preserved consciousness, absence of autonomic signs, and specific clinical characteristics.

Although complementary diagnostic tests were not performed in all animals, the presence of normal neurological examinations, complete blood counts, and serum biochemistry results in all patients, along with normal cerebrospinal fluid analysis and imaging (CT and MRI) in 16.1% (5/31), made structural, toxic, metabolic, and drug-induced etiologies unlikely. These findings suggest that the included cases likely represent genetic PD or PD secondary to dietary factors. However, genetic or serological testing to confirm the etiology was not performed in any animal, as such tests are only available internationally, which increases costs and leads many owners to decline.

Stress and/or excitement were identified in 48.4% (15/31) of the cases. These findings are consistent with those of previous studies, which have frequently reported stress and excitement as possible triggers of PD in dogs (Herrtage & Palmer 1983, Urkasemsin & Olby 2014, De Risio et al. 2016, Lowrie et al. 2018, Green & Olby 2021, Nessler et al. 2020, Polidoro et al. 2020). Trigger identification did not assist in differentiating PD from epilepsy because triggers are also common in idiopathic epilepsy (Forsgård et al. 2019), with stress being the most frequently reported (Shell et al. 2017, Forsgård et al. 2019). Nonetheless, recognizing triggers may aid clinical management by guiding owners to minimize such factors or, when avoidance is not feasible, consider anxiolytic medications that may help control PD episodes (Mandigers et al. 2024).

In humans, the dominant features of PD include chorea, athetosis, ballism, and dystonia; however, these manifestations are not easily distinguished in dogs. In veterinary medicine, the term "dyskinesia" is broadly used to describe movement disorders (Cerda-Gonzalez et al. 2021). Dystonia is the predominant clinical sign of PD in dogs (Santifort & Mandigers 2022). In the present study, dystonia was observed in all cases involving the pelvic and/or thoracic limbs. These findings are consistent with those of previous studies, such as that of Polidoro et al. (2020), who reported a high prevalence of limb dystonia in Maltese dogs with PD. Additionally, head dystonia, observed in 61.3% (19/31) of dogs, corroborates the recent findings of Liatis & De Decker (2023), who highlighted this sign as a predominant feature in certain types of PD.

The presence of generalized tremors, kyphosis, and incoordination, observed in 38.7%, 32.2%, and 12.9% of the cases, respectively, indicates that although dystonia is the predominant sign, it may not be the only manifestation. The identification of multiple clinical signs underscores the complexity of PD diagnosis, which may be confused with other neurological conditions, such as epilepsy, vestibular disorders (Lowrie & Garosi 2017), cataplexy, narcolepsy, syncope, neuromuscular weakness, and behavioral disorders (Richter et al. 2015, Cerda-Gonzalez et al. 2021).

In all included cases, the primary complaint was suspected epileptic seizures; 35.5% (11/31) of owners sought a second opinion, and 64.5% (20/31) of owners themselves suspected epilepsy. However, among the 353 dogs evaluated for suspected epilepsy at one participating service during the study period, 16 (4.5%) presented with episodes compatible with PD. This highlights the importance of recording episodes and reviewing videos to ensure accurate differentiation.

Although PD and epilepsy appear clinically similar, they occur in distinct regions of the brain (Obeso et al. 2002). However, they may share pathophysiological mechanisms related to altered neurotransmitter release in the synaptic cleft. In humans, cases of patients presenting with both conditions have been reported (Guerrini et al. 2001), possibly due to genetic defects, such as channelopathies, synaptopathies, and/or transportopathies, that affect synaptic function (Ahn & Ko 2020). Although this possibility exists in animals, it has not been confirmed.

The potential pathophysiological overlap between epilepsy and PD raises the hypothesis that antiepileptic drugs may also exert therapeutic effects on PD (Green & Olby 2021). In the present study, 54.5% of the dogs treated with antiepileptic drugs showed reduced episode frequency, and none exhibited increased frequency, unlike some dogs in the dietary or untreated groups.

In human medicine, antiepileptic drugs are commonly used to treat certain forms of PD (Bruno et al. 2004, Bhatia 2011, Erro et al. 2014), owing to their ability to reduce excitatory neurotransmitters in the synaptic cleft (Erro et al. 2014). However, in veterinary medicine, reports of favorable responses are limited to isolated cases: levetiracetam in a Welsh Terrier (Green & Olby 2021), phenobarbital in a German Shorthaired Pointer (Harcourt-Brown 2008), clonazepam in Labrador Retrievers (Lowrie & Garosi 2016) and Cavalier King Charles Spaniels (Gill et al. 2012). Despite favorable responses in some cases, efficacy may vary, and antiepileptic drugs may not be effective in other cases (Vanhaesebrouck et al. 2011, De Risio et al. 2016, Nessler et al. 2020, Mandigers et al. 2021, Whittaker et al. 2022, Liatis & De Decker 2023).

In some breeds, PD may have a self-limiting course (Lowrie & Garosi 2016), raising the possibility that the improvement observed in some dogs treated with phenobarbital in this study may have occurred independently of the medication.

In Jack Russell Terriers and Labrador Retrievers clinically diagnosed with PD and followed up for more than three years without treatment, 32% achieved complete remission, and among those without complete remission, 75% showed reduced episode frequency. These findings raise questions about when and whether treatment should be initiated in dogs with PD. In the present study, no significant differences in episode frequency evolution were observed between treated and untreated animals ($p = 0.611$), reinforcing the need for further research to clarify the role of different therapeutic approaches.

The use of a gluten-free diet in 35.3% (6/17) of treated dogs, with clinical improvement in four (66.7%), suggests that dietary interventions may play a relevant role in PD management, particularly in dogs with gluten sensitivity, as previously described in Border Terriers (Lowrie et al. 2018). Rogers et al. (2023) reported PD cases with positive gluten serology in different breeds, with 35.7% achieving complete remission and 21.4% showing greater than 50% reduction in episode frequency after initiating a gluten-free diet.

A potential mechanism for PD in gluten-sensitive dogs may involve immune-mediated processes within the gut-brain axis, in which immune activation and inflammatory responses triggered by gluten ingestion could interfere with neuronal excitability and central motor regulation, leading to paroxysmal episodes (Ambrosini et al. 2019, Yue et al. 2021). In the study by Rogers et al. (2023), one dog with negative gluten serology also improved on a gluten-free diet, suggesting that other unidentified immunological mechanisms or metabolites may be involved or that gluten sensitivity may not be fully detectable by currently available serological tests. Therefore,

a placebo effect associated with dietary modifications cannot be excluded (Muñana et al. 2010).

The study limitations include the absence of MRI and cerebrospinal fluid analysis in all animals to further exclude structural PD. However, normal neurological examinations during follow-up and the absence of progressive deficits suggested that structural brain lesions were unlikely, although definitive confirmation was not obtained.

Another limitation is the unavailability of an EEG. Similar to humans, PD episodes are characterized by the absence of autonomic signs, preserved consciousness, and normal (EEG) findings (Urkasemsin & Olby 2014). Although EEG is ideal for distinguishing PD from epileptic seizures, its absence does not preclude the establishment of a presumptive diagnosis when supported by video documentation, compatible history, and a normal interictal neurological examination. This methodology has been adopted in several studies (De Risio et al. 2016, Lowrie et al. 2018, Polidoro et al. 2020, Rogers et al. 2023). Lowrie & Garosi (2017) emphasized that diagnoses based on the observation and recording of episodes are widely used in veterinary practice. Even in studies with access to advanced diagnostics, such as that of Whittaker et al. (2022), most cases were diagnosed without EEG, reinforcing the feasibility of this approach.

This study highlights the importance of prospective research with larger sample sizes and the inclusion of multiple breeds, as well as controlled studies that systematically evaluate pharmacological, dietary, or conservative (natural course monitoring) interventions. Advancing our understanding of PD in dogs, particularly in underrepresented breeds, is essential for developing more effective management strategies.

## CONCLUSION

This study identified dogs with a presumptive diagnosis of paroxysmal dyskinesia (PD) at two veterinary neurology referral services in Brazil from 2020 to 2025, with no sex or breed predisposition. The mean age of onset was 58.1 months. Limb dystonia was present in 100% of the cases and was frequently accompanied by dystonic head tremors, generalized tremors, incoordination, and/or kyphosis. The mean episode duration was 6.8 minutes, and in approximately 50% of cases, triggers such as stress and/or excitement were associated with episode occurrence. Approximately half of the animals received treatment; however, no significant difference in clinical response was observed between treated and untreated dogs, highlighting the heterogeneity of paroxysmal dyskinesia. Further studies are needed to better understand the disease and its progression.

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