RESEARCH ARTICLE

Assessing the Efficacy of Progesterone as a Therapeutic Agent for Spinal Myelitis in Dogs: A Comprehensive Study

Ramkumar, P.K.[1], Saravanan, M.[2], Karthika, K.[2]*, Premalatha, N.[3], Velusamy, R.[4], Yogeshpriya, S.[2]

Abstract

This study investigated the treatment of myelitis in dogs, analyzing 198 dogs with neurological disorders leading to ataxia, of which 48 (24.00%) were confirmed as myelitis. Clinical assessments included haemato-biochemical analysis, cerebrospinal fluid (CSF) analysis, and radiography. Significant findings revealed decreased total protein (5.85±0.42 g/dL) and albumin (3.01±0.13 g/dL) in the myelitis group compared to healthy controls (6.22±0.14 g/dL and 3.32±0.08 g/dL, respectively), alongside elevated ALT (78.13±0.85 U/L), AST (53.71±0.64 U/L), ALP (75.81±0.82 U/L), and CPK (96.0±1.45 U/L). CSF protein levels were lower (0.024±0.001 g/L) compared to controls, with no significant differences in total nucleated cell counts. Six dogs each with myelitis were treated with glucocorticoids (Group-I) or progesterone (Group-II) over 14 days, assessing clinical signs, biochemical changes, serum progesterone, and Neuron Specific Enolase levels. The overall incidence of neurological disorders causing ataxia was 2.38% (48/198), with 59% encephalitis, 24% myelitis, and 17% neuromyopathies. The progesterone treatment protocol showed significant therapeutic efficacy for myelitis cases.

Keywords: Glucocorticoid, Neuron specific enolase, Myelitis, Prednisolone, Progesterone.

Ind J Vet Sci and Biotech (2025): 10.48165/ijvsbt.21.1.08

Introduction

The nervous system is vital for an animal's spatial orientation in its environment, encompassing various divisions responsible for posture, mental states, specific senses, and internal regulation. Inflammation in the brain, spinal cord, peripheral nervous system, or muscles can cause ataxia. Progesterone, a well-studied hormone in veterinary practice, historically used for reproductive issues, has shown promise in recent research for its neuroprotective properties. According to Hagg and Oudega (2006), damage to the spinal cord can lead to blood vessel injury, clot formation, reduced blood flow, and impaired autoregulation, causing ischemia. Secondary to the primary mechanical trauma, endothelial cell damage, haemorrhages, and swelling exacerbate tissue damage, triggering inflammatory responses and lesion expansion. This cascade of events, described as primary injury followed by secondary complications, includes impaired blood flow mechanisms, hypoxia, and cell death in both white and grey matter. The delicate nature of the spinal cord makes it vulnerable to various injuries, disrupting neural cell membranes and causing damage to blood vessels and axons. During spinal cord injury, haemorrhage, edema, and subsequent ischemia contribute to tissue swelling (Kobrine *et al.,* 1975). According to Mietto *et al.* (2015) sudden reperfusion post-ischemia worsens inflammation and reactive oxygen species release, exacerbating the injury. This study was aimed to determine the incidence and localization of lesions causing myelitis, assess behavioral, clinical, haemato-biochemical, and cerebrospinal fluid changes, and evaluate the efficacy of progesterone as a therapeutic agent compared to a standard glucocorticoid treatment protocol for spinal myelitis in dogs.

[1]Veterinary Clinical Complex, Veterinary College and Research Institute (VCRI), Orathanadu- 614 625, Thanjavur, TANUVAS, India

[2]Department of Veterinary Medicine, VCRI, Orathanadu-614 325, Thanjavur, TANUVAS, India

[3]Department of Veterinary Preventive Medicine, Madras Veterinary College, TANUVAS, Chennai-600007, India

[4]Department of Veterinary Parasitology, VCRI, Orathanadu-614 625, Thanjavur, TANUVAS, India

Corresponding Author: Dr. Karthika, K., Assistant Professor, Department of Veterinary Medicine, VCRI, Orathanadu, TANUVAS, India. E-mail: karthika1988.6@gmail.com

How to cite this article: Ramkumar, P. K., Saravanan, M., Karthika, K., Premalatha, N., Velusamy, R., & Yogeshpriya, S. (2025). Assessing the Efficacy of Progesterone as a Therapeutic Agent for Spinal Myelitis in Dogs: A Comprehensive Study. Ind J Vet Sci and Biotech. 21(1), 35-41.

Source of support: Nil

Conflict of interest: None

Submitted 04/09/2024 **Accepted** 11/10/2024 **Published** 10/01/2025

Materials and Methods

The study was conducted at the Department of Veterinary Medicine and the Veterinary Clinical Complex, Veterinary College and Research Institute, Orathanadu, Thanjavur, TANUVAS, India, during the year 2022-2023. A total of 198 dogs of various breed, age, and sex with neurological disorders leading to ataxia were included. Six apparently healthy dogs undergoing routine health check-ups served as the control group. Additionally, dogs presented with spinal myelitis injuries were divided into two treatment groups (n=6 each), receiving standard glucocorticoid and progesterone

protocols, respectively. The haemato-biochemical profile was assessed in these dogs on day 0, 7 and 14 post-treatment and was compared with the control group to evaluate the effectiveness of two treatment.

The spinal cord lesion was successfully identified as myelitis using a series of essential spinal reflex tests, including the patellar reflex, withdrawal reflex, cutaneous trunci reflex, and pain response. These reflex assessments were pivotal in precisely localizing the lesion within the spinal cord and aiding in the conclusive diagnosis of myelitis. Localization of lesion in spinal cord injury (SPI) was categorized according to the region and the specific neurological signs as: C1-C5: Upper Motor Neuron (UMN) signs in thoracic limbs, pelvic limbs, or both; C6-T2: Lower Motor Neuron (LMN) deficits in thoracic limbs and UMN signs in pelvic limbs; T3-L3: UMN signs exclusively in pelvic limb, thoracic limbs unaffected; and L4-S3: LMN signs in pelvic limbs, tail, and perineum, thoracic limbs unaffected.

The clinical examination was used to classify/grade the neurological status on a scale of 0 to 5 (modified from Scott, 1977) as Neurologically normal; Spinal hyperesthesia without neurological deficits; Ataxia, ambulatory para- or tetraparesis; Non-ambulatory para- or tetraparesis; Para- or tetraplegia with or without bladder control, and intact deep pain sensation; and Para- or tetraplegia, urine retention or overflow, and deep pain sensation loss, respectively. Scoring was also done for postural reactions and spinal reflexes and for bladder tone and fecal control (Table 1).

Table 1: Postural reaction and spinal reflexes examinations score, and urinary bladder tone and faecal control score

Test/parameter	Score	Description
Conscious proprioception	1	Absent
	2	Sluggish
	3	Normal
Deep pain sensation and withdrawal reflexes	1	Absent
	2	Mild/only superficial
	3	Strong superficial and deep
Patellar, cranial tibial, Sciatic and gastrocnemius reflexes	0	Absent
	1	Present but reduced
	2	Normal
	3	Exaggerated
	4	Exaggerated clonus
Panniculus reflex	1	Absent
	2	Normal
Bladder tone	1	Increased
	2	Normal
	3	Decreased
Fecal control	1	Absent or incontinence
	2	Normal

Localization of lesion in the spinal cord segment was performed based on involvement of UMN and LMN as described by Denny and Butterworth (2000) (Table 2).

Table 2: Lesion and its reflexes in spinal cord segments

Lesion in Spinal cord segments	Thoracic limb reflexes	Pelvic limb reflexes
C1-C5	UMN	UMN
C6-T2	LMN	UMN
T_3-L_3	Normal	UMN
L_4-Caudal segments	Normal	LMN

In Group II, dogs with myelitis were treated with methyl prednisolone succinate (30 mg/kg IV) within 8-12 h post-injury, followed by 15 mg/kg after 2 h, and continued daily for two weeks. Group III received hydroxyprogesterone caproate (2 mg/kg IM) on day 0, followed by oral micronized progesterone (5 mg/kg SID) for two weeks. The effectiveness of both treatments was compared using Scott's (1977) spinal cord injury grading system and by monitoring relevant reflexes and haemato-biochemical profile.

Postural Reaction and Spinal Reflexes Examinations

Reflexes, including conscious proprioception (CP), deep pain reflexes (DP), myotatic reflexes (PR; patellar, cranial tibial, gastrocnemius), and flexor withdrawal reflexes (SR), were evaluated on the 0^{th}, 7^{th}, and 14^{th} days post-treatment. Scores were classified as outlined in Table 3.

Table 3: Scoring of postural reaction and spinal reflexes

Reflexes	Scores		
	1	2	3
CP	Absent	Sluggish	Normal
DP	Absent	Mild/only superficial	Strong superficial and deep
SR	Absent	Sluggish	Normal
PR	Absent	Present but reduced	Normal
	Exaggerated clonus	Exaggerated	

Urinary Bladder Tone and Faecal Control

Urinary bladder tone (BT) and fecal control (FC) were evaluated on the 0^{th}, 7^{th}, and 14^{th} days post-treatment, with scores for BT (1: abnormal, 2: normal) and FC (1: incontinence, 2: normal).

The data on various parameters evaluated were expressed as mean ± standard errors, and were statistically analysed for comparison of groups and periods effects at $p<0.05$ (Snedecor and Cochran, 1994).

Results and Discussion

The incidence of myelitis in dogs was 24% (18/158), with 64.58% attributed to spinal cord injury and 35.42% to other causes. Neuroanatomic lesion localization was performed to

identify the origin of ataxia. Among the 198 cases examined, 59% (116 cases) were classified as encephalitis, 24% (48 cases) as myelitis, and 17% (34 cases) as neuromyopathy. Clinical signs of myelitis included urinary incontinence in 39.58%, paraparesis/plegia in 37.50%, Horner's syndrome in 35.42%, hind limb long-strided gait in 16.67%, Schiff sherington posture in 10.42%, double engine gait in 6.25%, and tetraparesis/plegia, fore and hindlimbs long strided gait and hindlimb short strided gait each in 4.17 % cases as shown in Table 4.

Table 4: Various clinical signs of myelitis in dogs (n=48)

Clinical signs	No. of cases	Percent
Urinary incontinence	19	39.58
Paraparesis/plegia	18	37.50
Horner's syndrome	17	35.42
Hind limb long strided gait	8	16.67
Schiff sherrington posture	5	10.42
Double engine gait	3	6.25
Tetraparesis/plegia	2	4.17
Fore and hind limbs long strided gait	2	4.17
Hind limb short strided gait	2	4.17

The haemogram of dogs with myelitis showed no significant difference compared to controls (Table 5), which was consistent with findings of Sivanarayanan (2015) and Sudhir (2019). The biochemical analysis (Table 6) revealed significant decrease in total protein and albumin, and highly significant ($p<0.01$) increase in ALT, AST, ALP and CPK levels in myelitis-affected dogs as compared to healthy ones. However, the kidney-liver function tests, blood glucose, and mineral profile were statistically similar in both the groups. Progesterone treatment, according to various studies, does not significantly affect biochemical parameters and is considered safe with no reported side effects (Root Kustritz, 2001; Cahil, 2007; Deeks, 2011).

Table 5: Mean ± SE of haemogram of control dogs and dogs with myelitis

Parameters	Control (n=6)	Myelitis (n=48)	p value
RBC ($x10^6$/µL)	7.75±0.22	7.49±0.06	0.545^{ns}
Hb (g/dL)	14.07±0.50	14.08±0.11	0.964^{ns}
PCV (%)	39.00±0.73	38.94±0.21	0.993^{ns}
WBC ($x10^3$/µL)	9.55±0.30	9.28±0.20	0.720^{ns}
Neutrophil (%)	71.67±0.56	70.42±0.33	0.947^{ns}
Lymphocytes (%)	22.17±0.48	22.13±0.23	0.615^{ns}
Eosinophils (%)	1.67±0.21	1.29±0.07	0.092^{ns}
Monocytes (%)	3.83±0.31	4.83±0.18	0.778^{ns}
Basophils (%)	1.17±0.31	1.33±0.07	0.723^{ns}

ns – Non-significant

Table 6: Mean ± SE of biochemical values of control dogs and dogs with myelitis

Parameters	Control (n=6)	Myelitis (n=48)	P value
Glucose (mg/dL)	97.17±3.07	101.42±0.75	0.249^{ns}
TP (g/dL)	$6.22±0.14^a$	$5.85±0.42^b$	0.00**
Albumin (g/dL)	$3.32±0.08^a$	$3.01±0.13^b$	0.014*
Globulin (g/dL)	2.90±0.08	2.84±0.04	0.755^{ns}
ALT (U/L)	$67.33±3.18^a$	$78.13±0.85^{ab}$	0.00**
AST (U/L)	$52.17±1.87^a$	$153.71±0.64^{ab}$	0.00**
ALP (U/L)	$73.50±1.48^a$	$175.81±0.82^{ab}$	0.00**
BUN (mg/dL)	16.58±0.48	16.01±0.17	0.115^{ns}
Creatinine (mg/dL)	0.92±0.12	0.99±0.04	0.888^{ns}
Direct bilirubin (mg/dL)	0.06±0.01	0.05±0.0	0.985^{ns}
Indirect bilirubin (mg/dL)	0.06±0.01	0.05±0.0	0.895^{ns}
GGT (U/L)	3.73±0.13	3.75±0.04	0.338^{ns}
CPK (U/L)	$84.0±3.11^a$	$96.0±1.45^{ab}$	0.000**
LDH (U/L)	135.94±1.05	136.05±2.20	0.552^{ns}
Ca (mg/dL)	9.95±0.17	9.88±0.07	0.054^{ns}
P (mg/dL)	3.87±0.26	3.86±0.06	0.927^{ns}
K (mg/dL)	4.62±0.21	4.53±0.05	0.662^{ns}
Mg (mg/dL)	2.12±0.07	2.14±0.02	0.848^{ns}

ns-Non-significant, **Highly significant ($p<0.005$), *Significant level ($p<0.05$), Means bearing same superscript do not differ significantly.

Radiography

Radiographic examinations were conducted for spinal injury cases, to exclude osseous causes. No abnormality was detected.

CSF Analysis of Dog with Myelitis Disorders

Statistically highly significant ($p<0.001$) difference was noticed in the values of CSF protein among the groups of myelitis (0.024±0.001 g/L), encephalitis (0.028±0.001 g/L) and neuromyopathy (0.029±0.001 g/L) dogs. No significant differences were observed statistically in the total nucleated cell counts among dogs with encephalitis, myelitis, and neuromyopathy ($p>0.05$), consistent with the findings of Cornelis *et al*. (2017).

Treatment Strategies for Myelitis with Glucocorticoids and Progesterone

The comparison of various haemato-biochemical, serum NSE, serum progesterone, were performed on pretreatment (day 0) and on 7^{th} and 14^{th} day post-treatment, and pre and post- treatment CSF progesterone for Group I (n=6) and Group II (n=6) dogs with healthy control Group III (n=6) and between treatment groups. Modified Scott's recovery grading and postural reaction and Spinal reflexes were compared between treatment groups.

Pre and Post-Treatment Haemogram of Myelitis Groups

A significant reduction (p≤0.01) in WBC levels was observed on day 14 in the prednisolone group, and on day 7 in the progesterone group along with reduced neutophils and increased lymphocytes counts compared to control group. Methyl prednisolone caused a marginal WBC reduction, likely due to its myelosuppressive effects, consistent with the findings of Rezk and Ibrahim (2013), particularly impacting neutrophil counts (Table 7).

Pre and Post-Treatment Blood Biochemistry in Myelitis Groups

A significant increase (p≤0.01) in total protein and globulin was observed on day 7 in the prednisolone group compared to control. Similarly, progesterone treatment led to a significant rise (p≤0.01) in albumin levels on day 7 and day 14 compared to control. Prednisolone significantly increased ALT on day 7, but reduced it by day 14, while progesterone caused a decrease in ALT by day 7, followed by a slight elevation. ALP, CPK and creatinine were also found increased on day 7 and 14 in prednisolone treated group compared to progesterone treated group. Overall, progesterone treatment improved biochemical parameters more effectively than prednisolone (Table 8).

Postural and Spinal Reflexes Scores in Myelitis Treatment Groups

Significant improvements (p≤0.01) in conscious proprioception (CP), deep pain (DP), spinal reflex (SR), and myotatic reflex (PR) were observed on days 7 and 14 in the progesterone group compared to prednisolone. By day 14, the progesterone group achieved the highest score of 3.0±0.0. Progesterone has also been associated with neuroprotection, neurogenesis, and myelin repair, as documented by González-orozco and Camacho-arroyo (2019) and Guennoun (2020). Furthermore,

Table 7: Mean ± SE pre and post treatment hemogram of Myelitis groups (n = 6)

S. No	Parameters	Units	Group III Control (n=6)	Group I: Prednisolone (n=6)			Group II: Progesterone (n=6)			p value
				Day 0	Day 7	Day 14	Day 0	Day 7	Day 14	
1.	RBC	$x10^6/\mu L$	7.72±0.27^a	6.59±0.27^b	6.73±0.30^b	6.83±0.21^b	6.55±0.10^b	6.85±0.31^b	6.70±0.15^b	0.043*
2.	Hb	g/dL	15.33±0.80	15.21±0.80	14.73±0.85	14.67±0.80	15.35±0.25	15.33±0.56	15.92±0.29	0.86^{ns}
3.	PCV	%	37.50±0.62^a	37.50±0.62^a	36.17±0.48^a	34.67±0.56^a	37.63±0.5^{6}a	38.55±1.1^{2}b	38.43±0.68^b	0.04*
4.	WBC	$x10^3/\mu L$	10.73±0.42^b	9.29±0.44^b	9.07±0.28^b	8.23±0.14^a	9.19±0.45^b	8.64±0.38^a	9.04±0.48^b	0.000**
5.	Neutrophils	%	71.83±0.79^b	71.67±0.67^b	64.33±1.36^a	62.33±0.92^a	70.83±1.22^b	70.50±1.12^b	68.67±0.88^b	0.000**
6.	Lymphocytes	%	19.17±0.40^a	21.00±1.15^a	23.33±1.74^b	26.00±0.58^b	17.5±1.22^a	21.5±0.5^{6}a	23.83±0.31^b	0.000**
7.	Eosinophils	%	3.50±0.4^{3b}c	2.17±0.31^a	5.00±0.6^{8}b	4.17±0.4^b	4.50±0.22^b	2.33±0.3^{3}c	2.50±0.34^a	0.000**
8.	Monocytes	%	4.17±0.48	4.67±0.76	6.60±0.61	6.00±0.52	4.33±0.33	4.67±0.8	4.50±0.85	0.09^{ns}
9.	Basophils	%	2.17±0.48	0.50±0.22	0.67±0.33	1.50±0.22	1.00±0.77	1.00±0.45	0.50±0.22	0.120^{ns}

Means bearing same superscript do not differ significantly between groups; NS- Not significant (p≥0.05), *Significant at 5 % level (p≤0.05), **Significant at 1 % level (p≤0.01).

Table 8: Mean (±SE) pre- and post- treatment values of blood biochemistry in Myelitis affected groups of dogs treated with prednisolone and progesterone

S. No	Parameters	Units	Group III Control (n=6)	Group I: Prednisolone (n=6)			Group II: Progesterone (n=6)			P value
				Day 0	Day 7	Day 14	Day 0	Day 7	Day 14	
1.	Glucose	mg/dL	76.67±3.32^a	76.67±3.3^{2}a	102.17±3.1^{2}b	77.67±2.91^a	79.93±5.4^{6}a	97.95±2.42^b	90.62±2.61^a	0.000**
2.	TP	g/dL	6.32±0.18^a	6.32±0.1^{8}a	6.95±0.08^b	6.10±0.15^a	6.27±0.06^a	6.28±0.05^a	6.20±0.04^a	0.000**
3.	Albumin	g/dL	2.72±0.10^a	2.72±0.1^{0}a	2.52±0.11^a	2.52±0.11^a	2.81±0.0^{3}a	3.18±0.0^{3}b	3.25±0.01^b	0.000**
4.	Globulin	g/dL	3.60±0.21^a	3.61±0.2^{3}a	4.43±0.13^b	3.58±0.23^a	3.05±0.08^a	3.11±0.05^a	2.95±0.03^a	0.000**
5.	ALT	U/L	75.83±0.91^a	75.83±0.9^{1}a	79.33±2.0^{3}b	78.50±1.48^a	72.95±1.90^a	72.52±1.35^a	73.14±1.20^a	0.007**
6.	AST	U/L	55.33±2.25^a	55.33±2.25^a	54.50±1.6^{1}a	57.17±2.12^b	50.43±2.20^a	46.44±2.3^{1}a	48.56±2.56^a	0.009**
7.	ALP	U/L	71.83±1.3^{5}a	114.28±1.5^{3}c	136.87±2.0^{1}c	172.32±2.3^{3}c	113.62±2.5^{3}b	119.05±3.3^{0}b	116.82±3.41^b	0.000**

8.	BUN	mg/dL	17.07±0.73	17.48±0.61	17.23±0.86	17.53±0.87	18.30±0.78	17.45 ±0.48	17.13±0.59	0.898^{ns}
9.	Creatinine	mg/dL	0.80±0.05^{a}	0.80±0.05^{a}	1.03±0.0^{7}a	1.27±0.10^{b}	0.75±0.07^{a}	0.89±0.06^{a}	0.82±0.08^{a}	0.000**
10.	D Bilirubin	mg/dL	0.04±0.01	0.04±0.01	0.06±0.01	0.07±0.01	0.04±0.02	0.07±0.01	0.06±0.01	0.739^{ns}
11.	ID Bilirubin	mg/dL	0.06±0.01	0.06±0.01	0.07±0.0	0.07±0.01	0.05±0.01	0.05±0.02	0.06±0.01	0.297^{ns}
12.	GGT	U/L	4.65±0.23	4.65±0.23	5.0±0.18	4.92±0.19	4.37±0.34	4.29±0.35	4.40±0.20	0.326^{ns}
13.	CPK	U/L	88.33±11.71	88.33±11.71	122.0±12.57	130.67±8.62	106.33±17.44	90.33±11.59	89.00±7.24	0.061^{ns}
14.	LDH	U/L	145.17±5.71^{b}	145.17±5.7^{bc}	141.28±6.35^{bc}	149.46±3.5^{8b}c	120.38±3.7^{2}a	126.46±2.7^{1}a	127.62±2.41^{a}	0.000**
15.	Ca	g/dL	10.40±0.16	10.3 ±0.19	11.17±0.12	11.33±0.12	10.40±0.21	10.29±0.17	10.32±0.10	0.704^{ns}
16.	P	mg/dL	4.75±0.12	4.75±0.12	4.87±0.12	4.63±0.15	4.16±0.22	4.03±0.37	3.99±0.31	0.052^{ns}
17.	K	mg/dL	4.70±0.09	4.70±0.09	4.84±0.17	4.70±0.09	4.54±0.12	4.51±0.08	4.53±0.10	0.319^{ns}
18.	Mg	mg/dL	2.07±0.08	2.07±0.08	2.13±0.08	2.17±0.03	2.04±0.03	2.13±0.07	2.11±0.05	0.799^{ns}

Means bearing same superscript do not differ significantly between groups; $^{NS-}$ Not significant (p≥0.05), *Significant at 5 % level (p≤0.05), **Significant at 1 % level (p≤0.01).

Table 9: Mean ± SE of recovery grades and pre- and post-treatment serum progesterone (P4) and NSE in myelitis affected dogs of two treatment groups (n=6 each)

Serum parameters	Control (n=6)	Group I: Prednisolone (n=6)			Group II: Progesterone (n=6)			P value
		0th Day	7th Day	14th Day	0th Day	7th Day	14th Day	
Mean recovery grade		3.50±0.34^{c}	2.00±0.45^{b}	0.50±0.34^{ab}	3.50±0.34^{c}	1.83±0.48^{bc}	0.17±0.17^{a}	0.000**
P4 (ng/mL)	0.78±0.1^{1}a	0.50±0.11^{a}	0.54±0.12^{a}	0.44±0.08^{a}	0.59±0.15^{a}	2.18±0.27^{b}	3.31±0.01^{c}	0.00**
NSE (ng/mL)	7.08±0.97^{a}	40.83±1.85^{c}	23.33±1.74^{b}	10.0±0.5^{8}a	45.17±1.40^{c}	34.0±3.28^{bc}	22.50±5.71^{b}	0.00**

Means bearing same superscript do not differ significantly otherwise significant between groups; $^{NS-}$ Not significant (p≥0.05), ** Significant at 1 % level (p≤0.01), NSE- Neuron Specific Enolase.

transient receptor potential (TRP) channels play a role in inflammation regulation, making them potential mediators of progesterone's therapeutic effects (Silverman *et al*., 2020).

Modified Scott Recovery Grading in Myelitis Treatment Groups

Pre- and post-treatment clinical evaluation of dogs with myelitis following treatment with prednisolone and progesterone as per modified Scott recovery grading is depicted in Table 9, and Fig. 1 & 2, respectively. The recovery grade for progesterone treated group was highly significant compared to prednisolone treated group (Table 9).

Serum Progesterone and NSE and CSF Progesterone in Myelitis Treatment Groups

The mean ± SE values of pre and post-treatment serum progesterone (P4) and NSE in myelitis treatment groups are shown in Table 9. Critical analysis revealed an overall improvement in the serum progesterone and NSE in progesterone than prednisolone treatment group. The mean ± SE values of pre and post-treatment CSF progesterone in myelitis treatment groups revealed a highly significant (p≤0.01) increase on the day 14 of progesterone therapy (1.27±0.09 vs. 0.18±0.01 ng/mL), but no change was noticed with prednisolone therapy (0.16±0.01 vs. 0.17±0.01 ng/mL).

Conclusion

In the present study, myelitis accounted for 24% of neurological disorders among 198 dogs, with spinal cord injury being the predominant cause, representing 64.58% of cases. Progesterone and methylprednisolone demonstrated equal potency in treating spinal myelitis in dogs. However, progesterone offers several advantages, including cost-effectiveness, safety during pregnancy, and fewer side effects. Administered once every three days due to its sustained release in an oil-based adjuvant, progesterone is a more convenient treatment option. In contrast, methylprednisolone, though effective, is more expensive, requires daily administration, increases the risk of ulcers, and is not recommended during pregnancy. Therefore, progesterone emerges as a preferable alternative in the management of canine spinal myelitis.

Acknowledgement

We sincerely thank the Dean of the Veterinary College and Research Institute, Orathanadu, and the Professor and Head of the Veterinary Clinical Complex, Veterinary College and Research Institute, Orathanadu, for their unwavering support in the successful conduct of this research.

Pre treatment	Post treatment
	
Fig 1a: Flaccid Paralysis of Hindlimbs	**Fig 1b:** Post treatment recovery– Animal standing on all limbs
	
Fig 1c: Absence of deep pain reflex in Hindlimbs	**Fig 1d:** Post Treatment recovery
	
Fig 1e: Spastic paralysis of hindlimbs	**Fig 1f:** Post Treatment recovery

Fig. 1: Pre and post-treatment clinical evaluation of myelitis affected dogs - Prednisolone treatment group

Pre treatment	Post treatment
	

Fig 2a: Spastic paralysis of hindlimbs	**Fig 2b:** Post treatment recovery
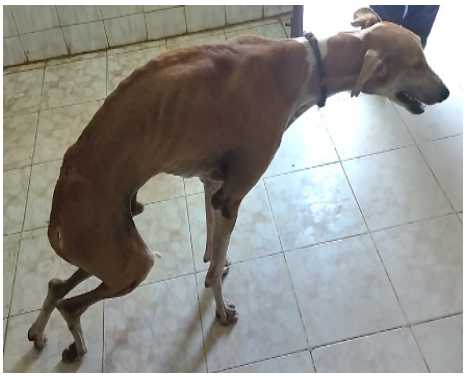	
Fig 2c: Absence of Conscious proprioception in hindlimbs	**Fig 2d:** Post treatment recovery
	
Fig 2e: Spastic paralysis of hindlimbs	**Fig 2f:** Post treatment recovery

Fig. 2: Pre and post-treatment clinical evaluation of myelitis affected dogs - Progesterone treatment group

References

Cahill, M.A. (2007). Progesterone receptor membrane component 1: An integrative view. *Journal of Steroid Biochemistry and Molecular Biology*, *105*, 16-36.

Cornelis, I.N.E. (2017). Meningoencephalomyelitis of unknown aetiology in dogs: A diagnostic, therapeutic and prognostic challenge. *Thesis,* Ghent University. Faculty of Veterinary Medicine.

Deeks, E.D. (2011). 17 α-Hydroxyprogesterone Caproate (Makena™) in the prevention of preterm birth. *Paediatrics Drugs, 13*(5), 337-345.

Denny, H.R., & Butterworth, S J. (2000). A guide to canine and feline orthopaedic surgery. 4th edition. Blackwell Sciences Ltd., p. 175-182.

González-Orozco, J.C., & Camacho-Arroyo, I. (2019). Progesterone actions during central nervous system development. *Frontiers in Neuroscience, 17*(13), 503.

Guennoun, R. (2020). Progesterone in the brain: Hormone, neurosteroid and neuroprotectant. *International Journal of Molecular Science, 21*, 5271.

Hagg, T., & Oudega, M. (2006). Degenerative and spontaneous regenerative processes after spinal cord injury. *Journal of Neurotrauma, 23*(3-4), 264-280.

Kobrine, A.I., Doyle, T.F., & Martins, A.N. (1975). Autoregulation of spinal cord blood flow. *Clinical Neurosurgery, 22,* 573-581.

Mietto, B.S., Mostacada, K., & Martinez, A.M. (2015). Neurotrauma and inflammation: CNS and PNS responses. *Mediators in Inflammation, 2015,* 251204.

Rezk, N.A., & Ibrahim, A.M. (2013). Effects of methyl prednisolone in early ARDS. *Egyptian Journal of Chest Diseases & Tuberculosis*, *62*(1), 167-172.

Root Kustritz, M.V. (2001). Use of supplemental progesterone in management of canine pregnancy. In: *Recent Advances in Small Animal Reproduction*. Concannon P.W., England G., Verstegen J. III, Linde Forsberg C., editors. IVIS, Ithaca, NY, USA.

Scott, H.W. (1997). Hemilaminectomy for the treatment of thoracolumbar disc disease in the dog: A follow-up study of 40 cases. *Journal of Small Animal Practice*, *38*(11), 488-494.

Silverman, H.A., Chen, A., Kravatz, N.L., Chavan, S.S., & Chang, E.H. (2020). Involvement of Neural Transient Receptor Potential Channels in Peripheral Inflammation. *Frontiers in Immunology*, *11*, 1-18.

Sivanarayanan, T.B. (2015). Evaluation of bone marrow derived mesenchymal stem cells and stem cell conditioned media for the repair of acute and subacute nerve injuries. *Ph.D. Thesis* Indian Veterinary Research Institute, Izatnagar, India

Snedecor, G.W. and Cochran, W.G. (1994). *Statistical Methods*. 8th edn., Iowa State University Press, Ames, Iowa, USA.

Sudhir, K.G. (2019). Evaluation of surgicotherapeutic approach in spinal disorders in dogs with posterior paresis and hindquarter weakness. *Thesis.* Maharashtra Animal and Fishery Sciences University, Nagpur, India