Original Article

Rapid post-castration decline in serum canine prostatic specific esterase (CPSE) and prostatic volume in dogs with benign prostatic hyperplasia

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Abstract

The objectives of this study were to quantify temporal changes in prostatic volume and serum CPSE concentrations in dogs with benign prostatic hyperplasia (BPH) (n = 6) following surgical castration, and to evaluate the potential of CPSE as a biomarker for monitoring prostatic regression. Prostatic volume was determined by transabdominal ultrasonography, and serum CPSE concentrations were measured at baseline (Week 0) and at Weeks 1, 2, and 4 post-castration. The mean (±SE) prostatic volume before castration was 11.4 ± 1.9 cm^3 and decreased significantly to 6.3±1.1 cm^3 by Week 1 ($P < 0.001$), representing a reduction to approximately 56% of the baseline value. Smaller progressive declines were observed at subsequent time points up to Week 4 ($P > 0.05$). In parallel, serum CPSE concentrations dropped sharply from 133.8 ± 19.8 ng/mL at baseline to 16.2 ± 7.6 ng/mL by Week 1 (approximately 10% of the initial value, $P < 0.05$), with minimal additional change thereafter ($P > 0.05$). A strong positive correlation ($\rho = 0.720$, $P < 0.001$) was observed between prostatic volume and serum CPSE levels. These findings indicate that both prostatic volume and serum CPSE concentrations decrease markedly within the first week after castration, highlighting CPSE as a minimally invasive biomarker for monitoring prostatic involution and postoperative recovery in dogs. Persistent elevation of CPSE levels or failure to reduce prostatic volume beyond one week after castration may warrant further evaluation for underlying conditions, such as chronic prostatitis or prostatic neoplasia.

Keywords: CPSE, dog, orchidectomy, prostate

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Introduction

Castration, bilateral orchidectomy, is among the most performed surgical procedures in male dogs, carrying significant implications for both contraception and reproductive health, particularly in the prevention of prostatic disorders. Among prostatic disorders, benign prostatic hyperplasia (BPH) is highly prevalent and androgen-dependent, occurring in a substantial proportion of intact male dogs over 6 years of age, as the prostate's responsiveness to testosterone increases with advancing age (Berry *et al.*, 1986a). Castration remains the preferred treatment for BPH because it permanently removes the source of testosterone from Leydig cells in the testes, thereby inducing substantial prostatic regression and alleviating clinical symptoms (Kawakami *et al.*, 1995; Cazzuli *et al.*, 2022).

Canine prostatic-specific esterase (CPSE) is a serine protease produced almost exclusively by the prostate gland (Chapdelaine *et al.*, 1984), making it a reliable biomarker for assessing prostatic function in dogs. Analogous to prostate-specific antigen (PSA) in human andrology, CPSE and PSA are closely related serine proteases with similar biological roles; however, their terminology, species specificity, and clinical applications differ. PSA is the established biomarker for human prostate health, particularly used for screening and monitoring prostate cancer (Min *et al.*, 2025). In contrast, CPSE is the canine counterpart, used exclusively in dogs, mainly for diagnosing and monitoring BPH (Alonge *et al.*, 2018). Serum CPSE concentrations correlate with prostatic size and androgenic activity (Holst *et al.*, 2021) and are elevated in dogs with BPH progression (Posastiuc *et al.*, 2025) and prostatic carcinoma (Melandri and Alonge, 2021). A threshold concentration of 52.3 ng/mL has been proposed to distinguish healthy intact dogs from those with prostatic disorders (Alonge *et al.*, 2018). Following castration, circulating testosterone levels decline rapidly, resulting in prostatic regression (Kawakami *et al.*, 1995) and a presumed parallel reduction in serum CPSE. However, the time course, magnitude, and pattern of CPSE decline after castration remain incompletely characterized, limiting its clinical interpretation. While PSA has been extensively studied in human andrology and is widely used to monitor both physiological and pathological changes in the prostate, research on CPSE remains relatively limited, despite its commercial availability in veterinary practice for several years. Comprehensive studies characterizing CPSE dynamics under physiological conditions and after androgen withdrawal are scarce, highlighting a critical knowledge gap.

A predictable reduction in CPSE following castration could serve as a biochemical indicator of effective androgen deprivation, analogous to the decline in PSA observed in men treated with luteinizing hormone-releasing hormone (LHRH) superagonists, even in the absence of malignant prostatic disease (Wenisch *et al.*, 2014). Conversely, persistently elevated CPSE levels after castration may suggest androgen-independent pathology. Despite its diagnostic potential in clinical practice, limited data are available describing post-castration changes in serum CPSE concentrations in dogs. Establishing temporal reference values and characterizing the variability of CPSE following androgen withdrawal through surgical castration would enhance understanding of the relationship between prostatic involution and serum CPSE dynamics, thereby improving its clinical applicability in monitoring prostate pathology.

The objectives of this study were to quantify changes in prostatic volume and serum CPSE concentrations in dogs with BPH following surgical castration. The temporal pattern and extent of CPSE reduction post-castration would help evaluate the potential use of CPSE as a biomarker for monitoring prostatic regression and detecting abnormal post-castration prostatic activity.

Materials and Methods

Animals: The study was conducted at the Small Animal Teaching Hospital, Faculty of Veterinary Science, Chulalongkorn University, and included six client-owned intact male dogs (one Beagle, one Chihuahua, one Chinese Crested, one Poodle, and two Shih Tzus) diagnosed with subclinical BPH. Dogs had an average age of 9.2 years (range: 7 to 12 years) and an average weight of 6.7 kg (range: 4.5 to 16 kg) (Table 1). The diagnosis of BPH was established based on findings from digital rectal examination and ultrasonographic evaluation, according to the criteria described by Laurusevičius *et al.* (2024). On rectal examination, the prostate was typically displaced cranially into the abdominal cavity, with both lobes remaining bilaterally symmetrical and exhibiting a firm, non-painful consistency. Ultrasonographic assessment revealed a symmetrical gland with heterogeneous echotexture, often showing a diffuse, small-cystic pattern (Cunto *et al.*, 2019; Laurusevičius *et al.*, 2024). Several factors may influence serum CPSE concentrations in intact male dogs, including bacterial prostatitis, prostatic neoplasia, and recent ejaculation (Alonge *et al.*, 2020). To minimize these effects, all dogs were required to undergo 3 days of sexual rest before and throughout the study. Dogs exhibiting clinical signs of prostatitis or other prostatic disorders, reproductive abnormalities, or with a prior history of BPH treatment were excluded from participation. Informed consent was obtained from all owners before enrollment. The study was conducted in compliance with institutional and international ethical standards for animal research, and the experimental protocol was approved by the Institutional Animal Care and Use Committee (IACUC) of the Faculty of Veterinary Science, Chulalongkorn University (Protocol No. 2431080).

Experimental design: All enrolled dogs underwent routine surgical castration under general anesthesia. Prostatic volume was assessed by transabdominal ultrasonography, and blood samples were collected for measurement of serum CPSE concentrations at baseline (week 0) and at weeks 1, 2, and 4 following castration.

Table 1 Characteristics and pre-castration prostatic volume (PV) and serum canine prostatic specific esterase (CPSE) concentrations in dogs with subclinical BPH.

Dogs	Breed	Bodyweight (kg)	Age (y)	PV (cm^3)	CPSE (ng/mL)
1	Chihuahua	4.5	9	10.5	158.9
2	Shih Tzu	5.0	10	11.1	212.7
3	Shih Tzu	4.8	12	9.3	85.8
4	Beagle	16.0	8	20.7	147.6
5	Poodle	4.8	9	7.3	98.8
6	Chinese Crested	5.1	7	9.5	99.1

Prostate ultrasonography: Prostatic volume was measured using B-mode transabdominal ultrasonography while dogs were positioned in dorsal recumbency. A Mindray M9 ultrasound machine (Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China) was used. The ultrasound frequency was adjusted to optimize image clarity for each dog's individual characteristics. Prostatic dimensions were measured in sagittal and transverse planes to obtain length (L), width (W), and depth (D) (Ponglowhapan *et al.*, 2024). Then, prostatic volume was calculated using the formula: PV = ((L × W × D) / 2.6) + 1.8 cm^3 (Kamolpatana *et al.*, 2000).

Serum CPSE analysis: Blood samples were obtained from the cephalic vein and collected into serum tubes without anticoagulant. Samples were allowed to clot at room temperature for 30 minutes and subsequently centrifuged at 2500×*g* at room temperature for 10 minutes to separate the serum fraction. The resulting serum was carefully aliquoted and stored at -80°C until further analysis. All serum samples were analyzed within a single assay run to minimize inter-assay variability. Before analysis, samples were thawed at room temperature and thoroughly homogenized by vortexing. Serum CPSE concentrations were determined using a quantitative enzyme-linked immunosorbent assay (ELISA) with a commercially available kit (Odelis® CPSE, Virbac, Carros, France), according to the manufacturer's protocol. The assay demonstrated high analytical precision, with intra-assay and inter-assay coefficients of variation of 2.04% and 1.71%, respectively. All samples were analyzed in duplicate after a 10-fold dilution with the provided dilution buffer. Calibration curves were established using standards at concentrations of 20, 10, 5, and 2.5 ng/mL. Optical density was measured at 450 nm, and CPSE concentrations were calculated from the standard curve and corrected for the applied dilution factor.

Statistical analysis: Changes in prostatic volume and serum CPSE concentrations following castration were expressed as percentage changes from baseline (%PV from initial and %CPSE from initial, respectively). Due to non-normal data distribution, temporal variations across weeks 0, 1, 2, and 4 were analyzed using the Friedman test for both parameters. Post hoc pairwise comparisons between time points were conducted using the Wilcoxon signed-rank test, with statistical significance set at $P < 0.05$. Statistical differences in prostatic volume (PV) and serum CPSE concentrations at Weeks 1, 2, and 4 post-castration were compared with baseline values measured prior to castration (Week 0). To determine if a relationship existed between the percentage of initial prostatic volume and serum CPSE levels, we applied Spearman's rank correlation analysis (ρ). A two-tailed *P*-value < 0.05 was considered statistically significant. All statistical analyses were conducted using IBM SPSS Statistics software, version 29.0.2.0 (IBM Corp., Armonk, NY, USA).

Results

Changes in prostatic volume: The mean (±SE) prostatic volume of dogs prior to castration (Week 0) was 11.4 ± 1.9 cm^3, which markedly decreased to 6.3 ± 1.1 cm^3 by Week 1 post-castration ($P < 0.01$). No significant differences in prostatic volume were detected among Weeks 1, 2, and 4 ($P > 0.05$) (Table 2), indicating that the major reduction occurred within the first week after castration. When expressed as a percentage of the initial baseline volume (%PV), the prostatic volume at Week 0 was 100%, which declined significantly to 55.7 ± 2.2% at Week 1 ($P < 0.001$). Further reductions were observed at Week 2 (47.8 ± 2.9%, $P < 0.001$) and Week 4 (37.9 ± 2.2%, $P < 0.001$) (Fig. 1). Overall, prostatic volume at all post-castration time points was significantly reduced compared with baseline values ($P < 0.01$).

Changes in serum CPSE concentrations: The mean (±SE) serum CPSE concentration of dogs before castration was 133.8 ± 19.8 ng/mL, which showed a marked reduction to 16.2 ± 7.6 ng/mL by Week 1 post-castration. No significant differences in serum CPSE concentrations were observed among Weeks 1, 2, and 4 ($P > 0.05$) (Table 2). When expressed as the percentage of the baseline (Week 0) value, the relative (%CPSE from initial) concentration was set at 100%. By Week 1, %CPSE from initial declined sharply to 9.9 ± 3.2% ($P = 0.028$), followed by a further decrease to 7.1 ± 3.2% ($P = 0.028$) at Week 2. At Week 4, a slight increase to 9.4 ± 6.1% ($P = 0.026$) was detected; however, this change was not statistically different from Week 2 ($P > 0.05$) (Fig. 2). At all post-castration time points, serum CPSE concentrations were significantly lower than those measured at baseline ($P < 0.05$).

Correlation between %PV and CPSE concentrations: A positive correlation was observed between the percentage of prostatic volume (%PV) from initial and serum CPSE concentrations following castration ($\rho = 0.720$, $r^2 = 0.667$, $P < 0.001$), indicating that the percentage of initial prostatic volume is a strong predictor of the level of serum CPSE concentration after castration (Fig. 3).

Table 2 Prostatic volume (cm^3) and serum CPSE concentration (ng/mL) in dogs with BPH before (Week 0) and after castration (Weeks 1, 2, and 4). Values are expressed as mean ± standard error (SE). Different superscripts (a, b) within a column indicate significant differences ($P < 0.05$).

Weeks	Prostatic volume (cm^3)	CPSE (ng/mL)
0	11.4 ± 1.9[a]	133.8 ± 19.8[a]
1	6.3 ± 1.1[b]	16.2 ± 7.6[b]
2	5.4 ± 0.9[b]	11.1 ± 6.8[b]
4	4.3 ± 0.8[b]	16.2 ± 12.6[b]

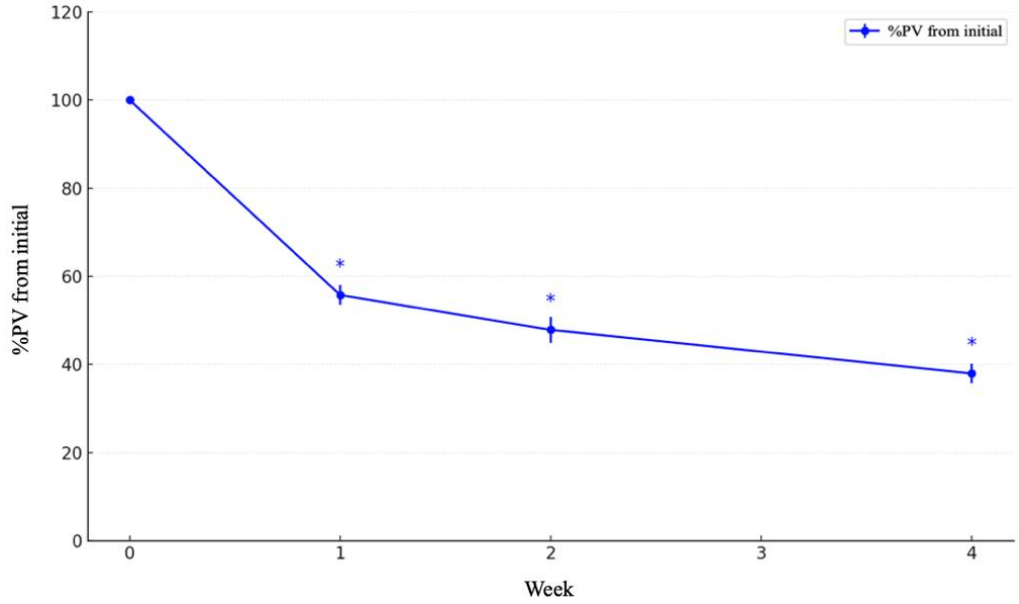

Figure 1 Temporal changes in prostatic volume relative to baseline (%PV from initial) over time before (Week 0) and after castration (Weeks 1, 2, and 4) in dogs with BPH (n = 6). Data are presented as mean ± standard error (SE). Significant differences ($P < 0.05$) from baseline are denoted by an asterisk (*).

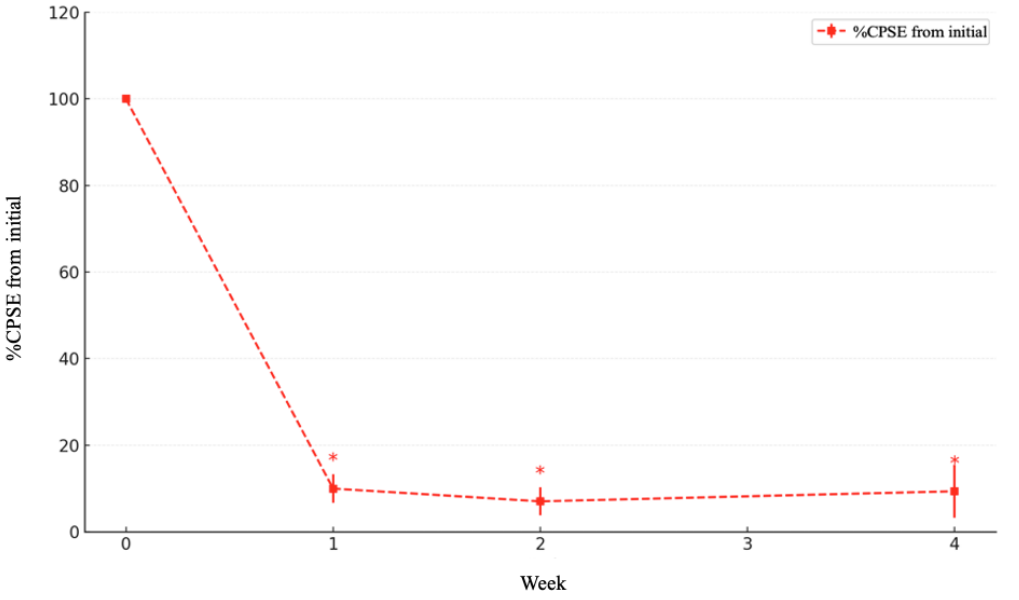

Figure 2 Temporal changes in serum CPSE concentrations relative to baseline (%CPSE from initial) over time before (Week 0) and after castration (Weeks 1, 2, and 4) in dogs with BPH (n = 6). Data are presented as mean ± standard error (SE). Significant differences ($P < 0.05$) from baseline are denoted by an asterisk (*).

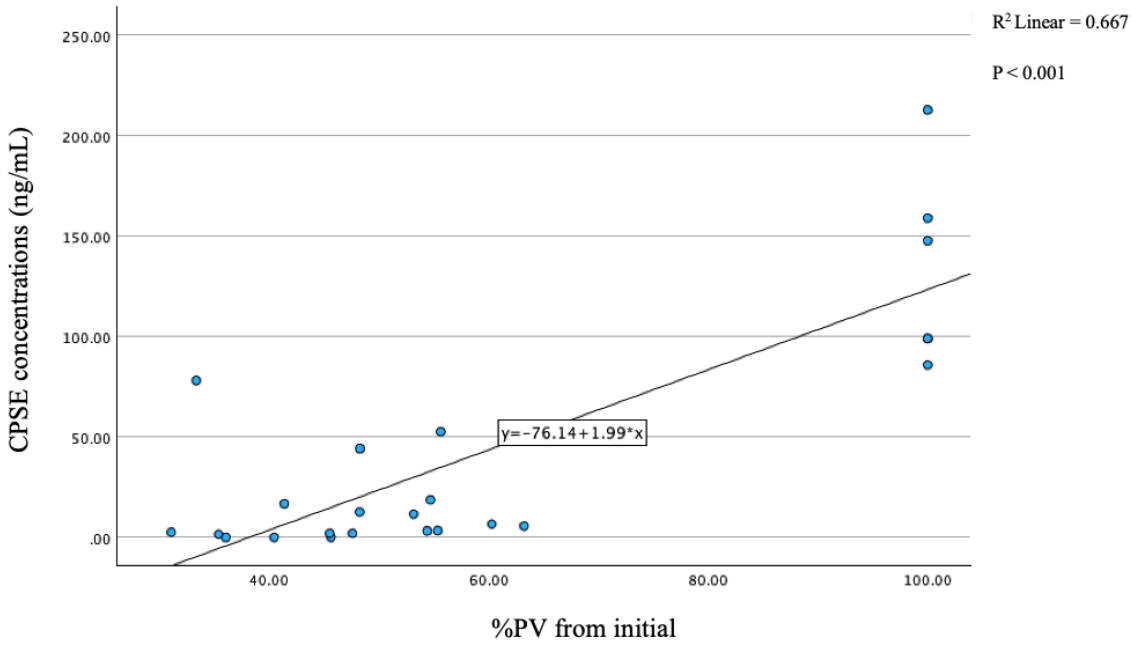

Figure 3 Scatter plot showing the relationship between percent change in prostatic volume (%PV from initial) and serum CPSE concentrations in dogs with BPH (n = 6) before and after castration.

Discussion

In this study, we quantified the changes in prostatic volume and serum CPSE concentrations in dogs undergoing surgical castration for BPH. The principal findings were (i) a rapid and substantial reduction in prostatic volume within the first week post-castration, with minimal additional decline thereafter, (ii) a parallel marked decline in serum CPSE concentration during the same period, and (iii) a strong positive correlation between percentage prostatic volume (%PV from initial) and serum CPSE levels post-castration. Collectively, these observations enhance our understanding of the immediate effects of androgen withdrawal on the prostate and emphasize the value of CPSE as a reliable biomarker for monitoring prostatic involution.

A substantial and rapid decrease in prostatic volume was observed following castration, dropping from 100% at baseline to 55.7 ± 2.2% by Week 1 and further to 37.9 ± 2.2% by Week 4. Comparable findings were reported by Kawakami *et al.* (1995), who noted that prostatic volume declined to 41% of the initial size within 4 weeks after castration in dogs affected by BPH. This sharp decline during the first week indicates that the majority of prostatic involution occurs very shortly after androgen withdrawal, whereas the plateau observed between Weeks 2 and 4 suggests that subsequent changes are of lesser magnitude and likely reflect gradual tissue remodeling rather than active regression. A rapid decrease in prostatic volume to approximately 50% of its original size may explain the noticeable alleviation or resolution of clinical signs in dogs with BPH within 1–2 weeks following castration, providing veterinarians with a physiological basis for the early clinical improvement observed after surgery.

It should be noted that the dogs enrolled in our study exhibited early-stage (subclinical) BPH. Subclinical BPH is generally asymptomatic and often remains unrecognized until prostatic enlargement becomes sufficient to induce clinical signs such as constipation, hematuria, dysuria, reduced fertility, or hind-limb lameness (Laurusevičius *et al.*, 2024). Elevated CPSE concentrations have been associated with increased prostatic volume, greater clinical complexity, and progression of BPH (Posastiuc *et al.*, 2025). Future studies should therefore investigate CPSE dynamics and prostatic regression in dogs presenting clinically evident BPH.

The mechanism underlying this rapid reduction in prostatic volume is attributed to the abrupt cessation of testosterone production following removal of the testes. Castration disrupts Leydig cell function, leading to a steep decline in circulating testosterone levels, typically within 7 days (Cazzuli *et al.*, 2022). This androgen deprivation triggers pronounced structural and functional changes within the prostate, including epithelial atrophy, stromal reduction, and decreased secretory activity (Kawakami *et al.*, 1995). Histologically, the canine prostate is a bilobed, compound tubuloalveolar gland composed predominantly of glandular epithelium supported by a fibromuscular stroma. The glandular acini, which form the major component of the parenchyma, are highly responsive to androgens. Under normal conditions, the acini are lined by a single layer of cuboidal to columnar epithelial cells that exhibit secretory hypertrophy under the influence of testosterone and dihydrotestosterone (DHT) (Barsanti and Finco, 1986; Kawakami *et al.*, 1995). Four weeks after orchidectomy, the prostatic epithelium exhibited a marked reduction in epithelial cell height, accompanied by the complete absence of morphological features indicative of secretory activity (Kawakami *et al.*, 1995). These cellular changes lead to decreased acinar diameter and luminal collapse, which altogether contribute to the substantial reduction in total prostatic volume observed during the first week post-castration in our study. The stromal component,

although also affected, tends to regress more gradually and to a lesser extent (Leav & Ling, 1968; Berry *et al.*, 1986b). Overall, the early prostatic shrinkage following castration primarily reflects acute epithelial atrophy rather than stromal remodeling.

The morphological regression of the prostate after castration was paralleled by a substantial decline in serum CPSE concentrations, suggesting the close relationship between androgen-dependent prostatic activity and CPSE secretion. Additionally, the prostate is the only organ producing CPSE (Chapdelaine *et al.*, 1984). In this study, serum CPSE levels in dogs with BPH decreased from 133.8 ± 19.8 ng/mL before castration to 16.2 ± 7.6 ng/mL at Week 1, representing approximately a 90% reduction during the same period, during which prostatic volume decreased by nearly half. The positive correlation between the percentage of prostatic volume and serum CPSE concentrations ($\rho = 0.720$, $P < 0.001$) further reinforces the notion that CPSE secretion reflects the functional and morphological integrity of the prostatic epithelium. Since CPSE is synthesized almost exclusively by prostatic epithelial cells (Chapdelaine *et al.*, 1984), the observed decrease in its circulating concentration is likely to correspond to the rapid epithelial atrophy and loss of secretory function that occur following androgen withdrawal. This temporal relationship suggests that serum CPSE may serve as a sensitive biochemical indicator of early prostatic regression after castration, analogous to the behavior of PSA in men following androgen deprivation therapy (Wenisch *et al.*, 2014). Moreover, the stabilization of CPSE concentrations after Week 1, with no significant changes detected at Weeks 2 and 4, indicates that the major endocrine and cellular responses to castration occur rapidly within a week and plateau thereafter. This pattern mirrors the plateau phase of prostatic volume reduction, further supporting the tight coupling between prostatic function and CPSE output. Conversely, persistent elevations in CPSE beyond this expected post-castration decline may therefore warrant further investigation, as they could reflect androgen-independent prostatic disorders such as chronic prostatitis or neoplastic transformation (Alonge *et al.*, 2018). Therefore, serum CPSE could offer potential clinical utility for postoperative monitoring and differential diagnosis of residual or recurrent prostatic disease in neutered dogs.

The strong correlation between percentage of residual prostatic volume (%PV from initial) and serum CPSE concentrations ($\rho = 0.72$, $P < 0.001$) provides further evidence that CPSE reflects both morphological and functional aspects of the prostate. It supports the concept that CPSE may serve as a non-invasive surrogate marker for prostatic size and secretory activity that is analogous to PSA in human andrology. In men, PSA declines significantly following androgen deprivation or prostatectomy, which helps monitor treatment efficacy and residual disease.

In the present study, the fact that the major declines in both prostatic volume and CPSE occur early has important clinical implications. For veterinarians monitoring treatment of BPH via castration, measurement of CPSE on Day 7 after castration may provide a useful early indication of prostatic regression. Given the marked reduction in prostatic volume within the first week post-castration, establishing reference timelines for CPSE decline would provide valuable clinical benchmarks. This finding may help veterinarians determine the optimal timing for postoperative re-evaluation and interpretation of treatment efficacy. Deviations from the expected decline in CPSE could suggest incomplete prostatic involution or the development of androgen-independent pathology. Indeed, previous work has shown that while CPSE levels are elevated in BPH, they may not reliably distinguish carcinoma, likely because neoplastic prostatic tissue may secrete less CPSE (Gobello *et al.*, 2002). Based on our observations during Weeks 1–4, CPSE concentrations remained consistently below 20 ng/mL. In neutered dogs, CPSE values exceeding this threshold may indicate androgen-independent prostatic disorders, such as prostatitis, carcinoma, or residual glandular tissue, and should prompt further diagnostic assessment. Similarly, persistently elevated CPSE levels or stabilization of prostatic volume beyond one week after castration warrant investigation.

Despite its strengths, this study has certain limitations. First, while our findings showed that CPSE concentrations had already fallen below 20 ng/mL by Day 7 post-castration, the exact timing of this decline remains undetermined. Future studies evaluating earlier postoperative intervals could clarify how rapidly CPSE decreases to this level. Second, the follow-up period was limited to 4 weeks; extending the observation period would provide deeper insight into the long-term dynamics of prostatic regression and stabilization of serum CPSE levels. Additionally, although the sample size was modest, the consistent trends observed across individuals underscore the robustness of this study. Future studies involving larger and more diverse canine populations would further enhance the generalizability and clinical applicability of these results.

In conclusion, our findings demonstrate that castration induces a rapid and marked reduction in prostatic volume and serum CPSE concentrations in dogs, with most of the changes occurring within the first week. The strong correlation between morphometric and biochemical alterations supports the clinical utility of CPSE for both the diagnosis and monitoring of androgen-dependent prostatic regression in veterinary andrology. Furthermore, measurement of CPSE at early follow-up may allow for the timely detection of abnormal post-castration prostatic activity and potentially distinguish normal involution from pathological persistence or recurrence.

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