CASE REPORT
JIVA 23 (2) August 2025
https://doi.org/10.55296/JIVA/23.2.2025.71-76

JIVA
OFFICIAL JOURNAL OF INDIAN VETERINARY ASSOCIATION, KERALA

Received on 17.06.2025
Accepted on 18.08.2025

FINASTERIDE THERAPY AS A NON-SURGICAL TREATMENT FOR BENIGN PROSTATIC HYPERPLASIA IN AN 11-YEAR-OLD INTACT DOBERMAN: A CASE REPORT

[1]Abhijith S.P., [2]Chandrakumar D, and [3]Abdul Kalam A

[1,3]Emergency Veterinarian, RMV Multispecialty Veterinary Clinic, Bengaluru,
[2]Consultant Veterinarian, Bengaluru.

Corresponding author: abhijithsp560@gmail.com

ABSTRACT

Benign prostatic hyperplasia (BPH) is a non-cancerous enlargement of the prostate gland and is the most common prostatic disorder in intact male dogs. It is a hormonally mediated condition primarily driven by androgens, particularly dihydrotestosterone (DHT), a potent metabolite of testosterone. BPH in dogs closely parallels the condition seen in aging human males and is regarded as a natural, age-related change in the canine prostate. An 11-year-old intact male Doberman was presented to the outpatient department with a history of hematuria, tenesmus, dyschezia, abnormal urine stream, and stranguria. Based on physical examination, hematobiochemistry, and ultrasonographic findings, the case was diagnosed as benign prostatic hyperplasia. In light of the owner's refusal of castration, a conservative medical treatment plan with Finasteride (5 alpha reductase inhibitor) was pursued. After four weeks, follow-up ultrasonography revealed a significant reduction in prostate size, accompanied by resolution of clinical signs. Treatment was continued for several months without any adverse effects. This case demonstrates the effectiveness of finasteride as a non-surgical alternative for managing BPH in breeding dogs, offering favourable clinical outcomes with minimal side effects.

Keywords: prostatic hyperplasia, finasteride, 5 alpha reductase

INTRODUCTION

Benign prostatic hyperplasia is the most common prostatic disorder in intact male dogs, with prevalence increasing significantly with age (Smith 2008). Benign prostatic hyperplasia (BPH) is prostate gland enlargement in which squamous metaplasia or glandular hyperplasia, or together, result from hormone imbalance in intact male dogs (Ladds, 1993). The development of BPH in dogs is androgen-dependent, primarily driven by dihydrotestosterone (DHT), a potent metabolite of testosterone. (Leav *et*

al., 1968). The prevalence of BPH increases with age. Histopathological studies reveal that more than 80% of intact male dogs over six years of age exhibit some degree of prostatic hyperplasia (Johnston et al., 2000).

By the age of nine, over 95% of intact males show evidence of BPH (Smith, 2008). Up to 95% of intact male dogs over the age of 9 years are affected by histological evidence of BPH. (Johnston et al., 2000). Breed predisposition has not been clearly established, although larger breeds may present with more noticeable clinical signs due to anatomical factors. Many cases of BPH are subclinical; however, when clinical signs occur, they may include tenesmus, hematuria, or a ribbon-like appearance of feces due to compression of the colon (Watts et al., 2017).

Age-related hormonal changes result in prostatic glandular hyperplasia and stromal proliferation, contributing to prostate enlargement (Christensen, 2018). Hematospermia and infertility can also be indicative of underlying prostatic enlargement in breeding males (Root Kustritz, 2006). Diagnosis is based on a combination of clinical signs, digital rectal examination, ultrasonographic imaging, and cytologic or histopathologic evaluation of prostatic fluid or tissue (Nyland *et al.,* 2015). Ultrasonography is a valuable, non-invasive tool in detecting symmetrical prostatic enlargement and echotextural changes associated with BPH (Mattoon *et al.,* 2002). Treatment options include androgen suppression through castration or medical therapy with drugs such as finasteride, which inhibits 5α-reductase activity (Sirinarumitr *et al.*, 2001). Castration remains the most effective and definitive treatment for BPH, leading to a rapid and sustained reduction in prostate size (Smith, 2008).

While many cases of BPH are subclinical and discovered incidentally, clinical signs may include tenesmus (straining to defecate), ribbon-like feces due to rectal compression, hematuria (blood in urine), or hematospermia (blood in semen) in breeding males. The prostate may be palpably enlarged and symmetrical on digital rectal examination. In some cases, enlargement can lead to urinary tract obstruction or infections if left unmanaged (Root 2006).

Diagnosis of BPH involves a combination of clinical history, physical examination, and diagnostic imaging. Digital rectal examination typically reveals a symmetrically enlarged, non-painful prostate. Ultrasonography is the most valuable imaging tool, revealing homogeneous or mildly heterogeneous

enlargement. Cytologic or histologic evaluation through prostatic fluid or biopsy can help differentiate BPH from other prostatic diseases such as prostatitis or neoplasia (Nyland *et al.,* 2015).

Castration is the most effective and definitive treatment for BPH. Removal of the testes results in a rapid decline in circulating testosterone and DHT levels, leading to significant reduction in prostate size within a few weeks (Smith, 2008). For breeding animals where castration is not desirable, medical management using 5α-reductase inhibitors such as finasteride has shown efficacy in reducing prostate size without affecting testosterone production or fertility (Sirinarumitr *et al.,* 2001). Other hormonal therapies include deslorelin implants, which temporarily suppress testosterone production.

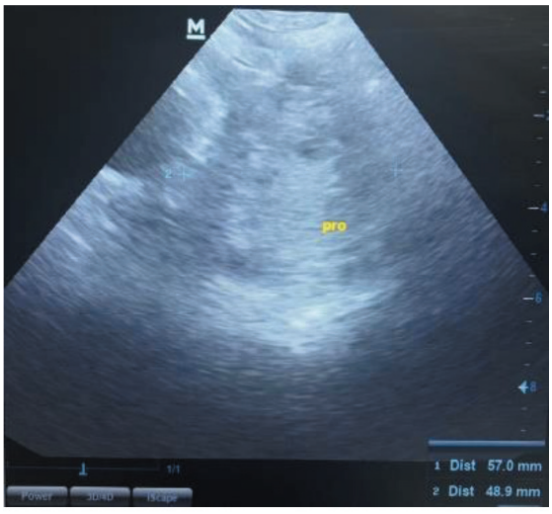

Fig1.1. Ultrasound scan image of prostate on day of presentation

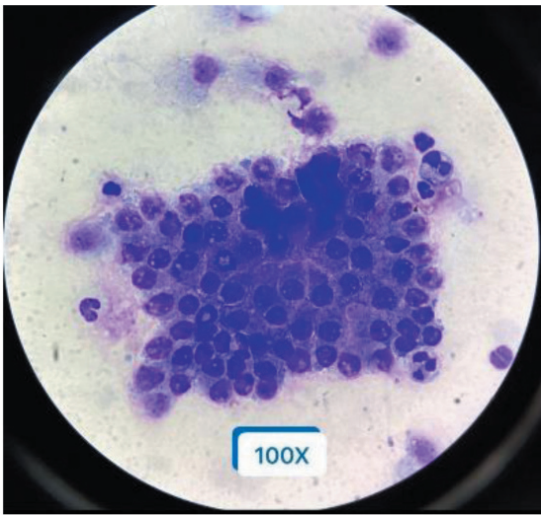

Fig 1.2. FNAC of prostate; Smears show sheets and clusters of benign acinar cells with small, uniform nuclei and abundant cytoplasm. Occasional stromal fragments and inflammatory cells. No nuclear pleomorphism, mitoses, or necrosis.

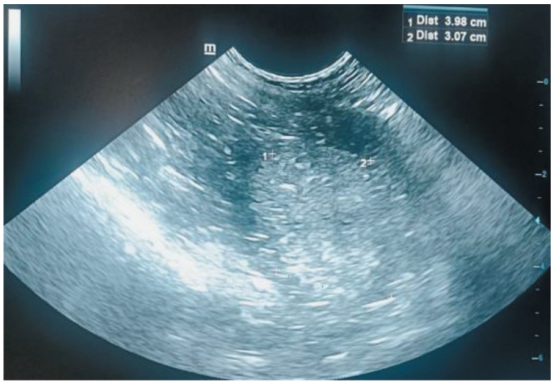

Fig 1.3. Ultrasound scan image of the prostate after therapeutic management

CASE HISTORY & OBSERVATIONS

A 11-year-old intact male Doberman was presented to the outpatient department with a history of hematuria, tenesmus, dyschezia, abnormal urine stream and stranguria. Ribbon like flat feces were one of the special complaints mentioned by the owner. On rectal examination, a round moderately hard structure could be

appreciated causing rectal compression. On ultrasonographic examination, the prostate gland is enlarged, measuring approximately 57 mm x 48 mm (Fig 1.1), echotexture appears heterogeneous, no focal lesion identified within prostate. The enlargement was symmetrical and intact capsule. No evidence of enlarged sub lumbar lymph node. Mild post-void residual urine suggestive of possible bladder outlet obstruction. Transabdominal ultrasound guided prostatic biopsy confirmed the diagnosis of BPH (Fig 1.2).

TREATMENT & DISCUSSIONS

Although castration is the best method to correct prostate enlargement in dogs, the pet parent denied it. Thus, medical management using Finasteride @ 0.2mg/kg OD (Finast, Dr, Reddy's, India) was adopted. After two weeks of oral treatment, clinical symptoms such as hematuria and stranguria had resolved. By the fourth week, dyschezia had also significantly improved. At the two-month follow-up, a reduction in prostate enlargement was noted.

The canine prostate gland, an integral component of the male reproductive system. In dogs, the prostate gland serves as the sole male accessory gland, playing a crucial role in the secretion of components of seminal plasma (Smith, 2008).

The size of the canine prostate gland can vary due to factors such as the natural aging process, breed, body weight, sexual maturity and disease (Gadelha *et al.,* 2009).

The normal prostate gland is typically not well-defined on radiographic imaging. However, when enlarged, it alters the anatomical position of the rectum and urinary bladder, making it more distinguishable. While radiography cannot differentiate parenchymal abnormalities of the prostate, ultrasonography can identify those abnormalities (Singh *et al.,* 2021).

BPH results from an imbalance in the hormonal regulation of the prostate. As dogs age, there is continued exposure of prostatic tissue to circulating androgens, particularly DHT, which is synthesized from testosterone by the enzyme 5α-reductase. DHT binds to androgen receptors in prostatic cells, stimulating both glandular and stromal proliferation, leading to prostatic enlargement (Leav *et al.,* 1968). Unlike neoplastic processes, this hyperplasia is not malignant but can still cause discomfort and complications. Location, size and appearance of the prostate vary with previous disease, age and status of animal (Feeney *et al.* 1989). Leroy *et al.* (2013) observed that transabdominal ultrasonography has become the preferred imaging method for evaluating the prostate gland in dogs. In intact dogs, the prostate

was of fine to medium coarse echotexture with smooth margins, homogeneous and medium echogenicity. (Mattoon *et al.,* 2002).

CONCLUSION

An 11-year-old intact male Doberman presented with a history of tenesmus, hematuria, and ribbon-shaped feces, suggestive of prostatic enlargement. Digital rectal examination revealed a bilaterally symmetrical, non-painful, and enlarged prostate. Transabdominal ultrasonography confirmed uniform prostatic enlargement consistent with benign prostatic hyperplasia (BPH). As the owner denied, surgical castration was not pursued. Instead, medical management with finasteride at a dosage of 0.2 mg/kg once daily was initiated. Within eight weeks, a marked reduction in prostatic size was noted on follow-up ultrasonography (Fig 1.3), accompanied by resolution of clinical signs. The treatment was continued for several months, with no adverse effects observed. This case highlights the effectiveness of finasteride therapy as a non-surgical alternative for managing BPH in breeding dogs, with good clinical outcome and minimal side effects.

REFERENCES

R Christensen, B. W. (2018). Prostatic disease in dogs. *Veterinary Clinics of North America: Small Animal Practice, 48*(4), 701–719. https://doi.org/10.1016/j.cvsm.2018.03.004

Feeney, D. A., Johnston, G. R., Klausner, J. S., & Bell, F. J. (1989). Canine prostatic ultrasonography. *Seminars in Veterinary Medicine and Surgery (Small Animal), 4*(1), 44–57.

Gadelha, C. R. F., Vicente, W. R. R., Ribeiro, A. P. C., Apparício, M. F., Covizzi, G. J., & Machado, L. D. S. (2009). Age-related ultrasonography, cytology and microbiologic exam of canine prostate. *Arquivo Brasileiro de Medicina Veterinária e Zootecnia, 61*(6), 1261–1267. https://doi.org/10.1590/S0102-09352009000600002

Johnston, S. D., Root Kustritz, M. V., & Olson, P. N. S. (2000). *Canine and feline theriogenology*. W. B. Saunders.

Ladds, P. W. (1993). The male genital system. In K. V. F. Jubb, P. C. Kennedy, & N. Palmer (Eds.), *Pathology of domestic animals* (4th ed., pp. 471–529). Academic Press.

Leav, I., & Ling, G. V. (1968). Prostatic hyperplasia in the dog: A spontaneous model for benign human prostatic hyperplasia.

Journal of Urology, 100(4), 607–612. https://doi.org/10.1016/S0022-5347(17)62520-7

Leroy, C., Conchou, F., Layssol Lamour, C., Deviers, A., Sautet, J., Concordet, D., & Mogicato, G. (2013). Normal canine prostate gland: Repeatability, reproducibility, observer dependent variability of ultrasonographic measurements of the prostate in healthy intact beagles. *Anatomia, Histologia, Embryologia, 42*(5), 355–361. https://doi.org/10.1111/ahe.12017

Mattoon, J. S., & Nyland, T. G. (2002). Prostate. In T. G. Nyland & J. S. Mattoon (Eds.), *Small animal diagnostic ultrasound* (2nd ed.). Elsevier.

Nyland, T. G., & Mattoon, J. S. (2015). *Small animal diagnostic ultrasound* (3rd ed.). Elsevier Health Sciences.

Root Kustritz, M. V. (2006). *Clinical canine and feline reproduction: Evidence-based answers*. Wiley-Blackwell.

Singh, R., Sangwan, V., Devi, N. U., Mohindroo, J., & Pathak, D. (2021). Ultrasonographic and radiographic assessment of prostate gland in perineal hernia dogs. *Indian Journal of Animal Research, 55*(5), 568–574. https://doi.org/10.18805/IJAR.B-3774

Sirinarumitr, T., Johnston, S. D., et al. (2001). Effects of finasteride on size of the normal canine prostate gland. *Theriogenology, 56*(3), 457–465. https://doi.org/10.1016/S0093-691X(01)00575-3

Smith, J. (2008). Canine prostatic disease. *Veterinary Clinics of North America: Small Animal Practice, 38*(6), 1263–1279. https://doi.org/10.1016/j.cvsm.2008.07.004

Watts, A. E., et al. (2017). Prostatic disorders in dogs. *Theriogenology, 95*, 124–134. https://doi.org/10.1016/j.theriogenology.2016.08.008