Prostatic disorders in the dog

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Abstract

Common canine prostatic disorders include benign prostatic hypertrophy (BPH), prostatitis, prostatic cysts and prostatic adenocarcinoma. BPH is a spontaneous and age-related disorder of intact male dogs, which occurs in more than 80% male dogs over 5 years of age, and which is associated with clinical signs of sanguinous prostatic fluid, constipation and dysuria. BPH signs respond to castration or to finasteride treatment 0.1–0.5 mg/kg per os once daily, as finasteride inhibits conversion of testosterone to dihydrotestosterone, causing prostatic involution via apoptosis. BPH often occurs concurrently with prostatic infection, abscessation, cysts and neoplasia in the intact dog, and finasteride-induced prostatic involution may be beneficial in treatment of all of these conditions except neoplasia. Two studies suggest that risk of prostatic adenocarcinoma is increased in neutered, compared to intact male dogs. Although canine prostatic neoplasia, unlike human prostatic neoplasia, usually does not respond to androgen deprivation, recent reports of prostatic intraepithelial neoplasia (PIN) in a high percentage of older male dogs, with and without prostatic adenocarcinoma, suggests that PIN may be a precursor to adenocarcinoma in the dog as it is believed to be in man. © 2000 Elsevier Science B.V. All rights reserved.

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1. Introduction

The canine prostate is an androgen-dependent, ovoid-shaped, bilobed gland composed of glandular and stromal elements, which encircles the urethra of the male dog caudal to
the neck of the urinary bladder. The prostate contributes fluid to the first and third fractions of the ejaculate (England et al., 1990). Prostatic fluid is secreted continuously in intact male dogs, and flows retrograde into the urinary bladder or antegrade out the external urethral orifice in volumes ranging from a few drops to several milliliters, dependent on prostate size.

Prostatic growth and secretion are mediated by dihydrotestosterone (DHT), a metabolite of testosterone (T) formed in the presence of the enzyme, 5α-reductase. Canine prostatic size decreases by 50% within 3 weeks of castration, and by 70% within 9 weeks of castration (Barsanti and Finco, 1995).

The caudal/dorsal aspect of the prostate in most dogs can be palpated per rectum. The normal gland is not easily seen in survey radiographs, but can be identified using retrograde contrast urethrocytography with bladder distension or by ultrasonography (Johnston et al., 1991). Hyperplastic prostates usually are visible radiographically, and

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**Fig. 1.** Radiographic image of the prostate, lateral projection. The black line represents the distance between the sacral promontory and the pubis (DSPP). The greatest cranio–caudal distance of the prostate is measured parallel to this line. Reprinted with permission from Kamolpatana (1998).
the prostate is considered enlarged when the prostatic diameter, as visualized on the lateral radiographic view, is greater than 70% of the pubic–sacral promontory distance (DSPP) (Fig. 1) (Feeney et al., 1987).

Prostatic volume in cubic centimeters in the dog can be estimated from greatest craniocaudal \( L \), transverse \( W \) and dorsoventral \( D \) diameters detected using ultrasonographic examination, and applied to the formula: volume in cubic centimeters = \((L \times W \times D)/2.6\) + 1.8 (Kamolpatana et al., 1999).

### 2. Benign prostatic hypertrophy in the dog

Benign prostatic hypertrophy (BPH) is a spontaneous and age-related condition in humans and intact male dogs. The pathogenesis of BPH is not completely understood; however, DHT is accepted as a key hormone in stimulating enlargement of the canine prostate by enhancing growth in both stromal and glandular components. A major protein in canine seminal plasma, canine secretory prostatic esterase (CPSE), is produced by prostatic epithelial cells under the influence of T; serum CPSE is elevated in dogs with BPH (mean concentration of 189.7 ng/ml, \( n = 25 \)) compared to normal intact dogs (mean concentration of 41.8 ng/ml, \( n = 20 \)) (Bell et al., 1995).

More than 80% of intact male dogs over 5 years of age exhibit BPH, and prostatic volume in affected dogs is 2 to 6.5 times greater than that of normal dogs of similar weight (Laroque et al., 1994, 1995; Hornbuckle et al., 1978; Kamolpatana, 1998). Prostatic volume in beagles (BW = 7–13 kg) with BPH has been reported to range from 20 to 31 cm\(^3\) (Wheaton et al., 1979), 19 to 21 cm\(^3\) (measured by MRI) (Laroque et al., 1995; Cohen et al., 1995) and 5 to 24 cm\(^3\) (measured by ultrasonography) (Iguer-Ouada and Verstegen, 1997).

Clinical signs in affected dogs include sanguinous prostatic fluid dripping from the tip of the penis, blood in the urine or semen, constipation, and difficult urination (Krawiec and Heflin, 1992). Diagnosis of BPH is based on detection of blood in prostatic fluid of the ejaculate or in prostatic fluid emitted from the end of the penis, and on detecting uniform prostatic enlargement by palpation, radiography and/or ultrasonography.

The treatment objective in dogs with BPH is to decrease prostatic size, which alleviates signs related to BPH; castration is the recommended treatment in most dogs (Basinger, 1987). Diethystilbestrol at the dose of 0.2–1.0 mg, PO, every 2–3 days, for 3–4 weeks was reported, historically, as an effective medical treatment of BPH in dogs, but potential adverse side effects of diethystilbestrol in dogs include bone marrow suppression and pancytopenia, and squamous metaplasia of the prostate, with ductal obstruction and cyst formation. Medroxyprogesterone acetate (3 mg/kg, SQ, given twice at 4-week intervals, and megestrol acetate, 0.55 mg/kg, per os, for 4 weeks) have been used to treat BPH in dogs, and are reported not to adversely affect semen quality or libido (Bamberg-Thalen and Linde-Forsberg, 1993). However, only 53% of BPH dogs treated with medroxyprogesterone acetate showed decreased prostatic size by 6 weeks of treatment, and the potential adverse sequelae of progesterone administration in dogs,
development of diabetes mellitus and mammary nodules, may argue against use of this regime.

In all mammals, T is metabolized to DHT under the influence of the inhibitable enzyme, 5α-reductase (Russell and Wilson, 1994). Two isoenzymes of 5α-reductase, type I and type II, translated from genes on different chromosomes, have been identified in tissues of humans, nonhuman primates, dogs, rats and mice (Liang et al., 1985). In humans, the prostate, epididymis, seminal vesicles and genital skin have type II 5α-reductase, and nongenital skin has type I. The canine prostate is reported to contain both type I and type II 5α-reductase, although there are no data regarding localization or amounts of each isoenzyme in specific lobes of the prostate of this species (Kamolpatana, 1998).

Finasteride (Proscar™ Merck, West Point, PA 19486, USA) is a commercially available synthetic steroid (azasteroid) which inhibits type II 5α-reductase and which, therefore, blocks conversion of T to DHT (Cohen et al., 1995; Span et al., 1998). It is an effective treatment for BPH in humans when given at a dose of 5 mg, PO, every 24 h for 12–24 months. Finasteride decreases serum concentrations of DHT, without affecting serum concentrations of T, cortisol, estradiol, luteinizing hormone, prolactin, or thyroid-stimulating hormone in men with BPH. Experimental beagles over 5 years of age with BPH that were treated with finasteride (1 mg/kg BW, PO, every 24 h, for 16–21 weeks) showed prostatic atrophy, and decrease in prostatic volume by 50–70% (determined by MRI) as compared to placebo-treated controls (Cohen et al., 1995, Laroque et al., 1995; Iguer-Ouada and Verstegen, 1997). This dose of finasteride also decreased serum concentrations of DHT in dogs, with no adverse effect on testis histology or semen quality.

A dose–response study of finasteride at 0.10–0.50 mg/kg, PO, every 24 h for 7 days (5 mg per dog in dogs weighing 10–50 kg), in normal intact male dogs caused significant decrease in serum concentrations of DHT without changing serum concentrations of T, suggesting that the 5 mg per dog dose for dogs ranging from 10 to 50 kg may be as effective as the 1 mg/kg dose reported earlier in research dogs (Kamolpatana et al., 1998). Finasteride at all doses was associated with a mean decrease in concentration of DHT of 55% (decrease from mean of 155 ± 32.3 pg/ml to 70 ± 15 pg/ml), and had no effect on serum T concentrations (pretreatment 2.6 ± 0.38 ng/ml; posttreatment 2.2 ± 0.63 ng/ml).

In a double blind, placebo-controlled clinical study to determine the effect of finasteride at 0.10–0.50 mg/kg of body weight, PO, every 24 h, on prostatic diameter and volume in dogs with spontaneous BPH, finasteride treatment decreased prostatic diameter by an average of 20%, prostatic volume by an average of 43%, and serum concentrations of DHT by an average of 58% after 16 weeks of treatment (Kamolpatana, 1998). The nine experimental dogs were client-owned animals with spontaneous BPH; they ranged in age from 2.7 to 11 years, in weight from 10.3 to 49 kg, and represented seven breeds. Pretreatment prostatic diameters ranged from 52.8 to 109 (mean ± SEM = 70.6 ± 6.3)% DSPP, and prostatic volume ranged from 10.4 to 82 (mean ± SEM = 35.5 ± 9.1) cm³. Posttreatment, the average prostatic diameter in treatment dogs ranged from 46.6 to 61 (mean ± SEM = 53.8 ± 2.5)% DSPP, and mean prostatic volume in treatment dogs ranged from 7.72 to 36.8 (mean ± SEM = 30 ± 7.9) cm³. Prostatic volume
decreased by 43%, suggesting that ultrasonography is comparable to radiography (prostatic diameter decreased by 20%) in evaluating change in prostatic size during medical treatment in BPH dogs, and may suggest uniform distribution of type II 5α-reductase in the dog prostate. Constipation and presence of blood in the seminal fluid decreased continuously after 1 week of treatment. By 4 weeks of treatment there was no blood in seminal fluid of the treatment dogs, except one; in that dog, blood persisted during the 16 weeks of treatment, even though his prostatic volume decreased by 53%. These results are similar to those in a study in which finasteride administration (1 mg/kg, PO, every 24 h, for 16 weeks, in BPH dogs) resulted in decreased prostatic volume (by 50–52%) measured by MRI (Cohen et al., 1995; Laroque et al., 1995).

Finasteride treatment in dogs causes decrease in semen volume, but has no adverse effect on semen quality and no effect on serum concentration of T. The average seminal volume of nine BPH dogs before treatment was 17 ± 11 (range = 1–70, n = 6), and seminal volume declined to 14 ± 12 (n = 5), 9 ± 6 (n = 7), 7.9 ± 7 (n = 5), 7.6 ± 4.8 (n = 6), 5.9 ± 3 (n = 7), and 7.8 ± 4 ml (n = 6) at 1, 2, 3, 4, 8, and 16 weeks of finasteride treatment, respectively (Kamolpatana, 1998). There was not a significant change in seminal volume in control dogs over 16 weeks of treatment. There was no significant difference in total number of sperm per ejaculation or in the spermiogram in treatment and control dogs before or during 16 weeks of treatment (P > 0.1) (Kamolpatana, 1998). Five treatment dogs that, during and after finasteride treatment, had normal libido during copulation, were successfully bred to bitches, and the bitches became pregnant with normal pregnancy, gestation length and litter size.

One study has reported that, after cessation of finasteride treatment, prostatic volume in research beagles with induced BPH slowly increased, and, by 12 weeks following cessation of treatment, prostatic volume was similar to pretreatment volume (Iguer-Ouada and Verstegen, 1997). In that study, however, pretreatment prostatic volume was as low as 5 cm³ (measured by ultrasonography), and clinical signs related to BPH were not described, so whether this time of BPH recurrence is the same in dogs with spontaneous BPH is unknown.

To determine whether finasteride-induced prostatic involution in dogs with spontaneous BPH occurs by programmed cell death (apoptosis) of prostatic cells, nine BPH dogs were randomly assigned to treatment or control groups and treated with finasteride (n = 5) at the dose of 0.10–0.50 mg/kg BW, PO, every 24 h, or placebo (n = 4) for 16 weeks. Prostatic cells from the third fraction of the ejaculate were concentrated by cytopsin centrifugation and examined for apoptosis using the terminal deoxyribonucleotidyl transferase (TdT)-mediated dUTP nick end labeling (TUNEL) method for detecting apoptotic cells (Oncor Apop Tag Plus In Situ Apoptosis Detection Kit, Oncor, Gaithersburg, MD 20877, USA) (Mesner and Kaufmann., 1997). When the four control BPH dogs finished the 16 weeks of placebo, they were then treated with finasteride (0.10–0.50 mg/kg BW, PO, every 24 h, for 16 weeks), and followed for another 16 weeks. These results were not significantly different than those from the first 5 treatment dogs, so treatment dogs results were pooled (n = 9). Percentage apoptotic prostatic cells in the prostatic fluid of treatment dogs increased significantly, from 9% before treatment to 33%, 31%, 26%, 24%, 27%, and 18% after 1, 2, 3, 4, 8 and 16 weeks treatment, respectively (Kamolpatana, 1998). There was no significant change in percentage of...
apoptotic prostatic cells in the prostatic fluid ejaculate of control BPH dogs. Finasteride-induced prostatic involution, therefore, appears to occur via apoptosis, not necrosis, in BPH dogs.

3. Infection/inflammation of the canine prostate gland

Both acute and chronic infections occur in the canine prostate gland, usually as a result of ascent of normal aerobic urethral bacteria (including mycoplasma) into a gland with benign prostatic hypertrophy. *Escherichia coli* is the most common bacterial organism identified in dogs with bacterial prostatitis, followed by *Staphylococcus aureus*, *Klebsiella* spp., *Proteus mirabilis*, *Mycoplasma canis*, *Pseudomonas aeruginosa*, *Enterobacter* spp., *Streptococcus* spp., *Pasteurella* spp., and *Haemophilus* spp. *Brucella canis* may infect the canine prostate, but is more commonly associated with epididymal and testicular infection and clinical signs referable to those organs. Infections with anaerobic bacteria also have been observed, as have infections with fungal agents (*Blastomyces dermatitidis*, *Cryptococcus neoformans*, or *Coccidioides immitis*) via hematogenous spread, urethral ascent, or penetration through the scrotum with descending prostate infection from a testicular source (Klausner et al., 1995).

Clinical signs in dogs with acute prostatitis include depression, pain on rectal palpation of the prostate, fever, straining to urinate or defecate, a “stiff-legged” gait, hematuria, edema of the scrotum, prepuce or hindlimb, and pollakiuria. One, some or all of these signs may be present. In addition, dogs with prostatic abscessation, peritonitis or septicemia may show signs of septic shock. Laboratory findings in most affected dogs include regenerative leukocytosis, although occasional animals are leukopenic. Urine collected by cystocentesis may contain blood, bacteria, and leukocytes, because prostatic fluid constantly drips retrograde from the prostatic urethra into the urinary bladder in the intact male dog.

Clinical signs in dogs with chronic prostatitis may be absent, or may consist of poor semen quality (decreased percentage of motile or morphologically normal sperm in the ejaculate) with infertility or sometimes decreased libido if prostatic contraction is painful. Urine may contain blood, bacteria as with acute prostatitis, and dogs with chronic prostatitis may be admitted to a veterinarian for suspected lower urinary tract disease.

Presumptive diagnosis of acute prostatitis is based on presence of clinical signs in an intact or recently castrated male dog. Presumptive diagnosis of chronic prostatitis is based on signs of infertility or decreased libido. Definitive diagnosis is made by detecting inflammatory exudate in prostatic fluid collected by ejaculation or prostatic massage. In general, needle aspiration of an infected prostate or prostatic abscess should be avoided when possible, so as to prevent seeding of the needle track with bacteria. The exudate should be characterized cytologically and microbiologically, which permits diagnosis of bacterial and fungal disease as well as determination of bacterial sensitivity to antibiotics. Normal canine semen and/or prostatic fluid should contain less than 10,000 bacteria per milliliter, and sediment should not contain significant numbers of inflammatory cells. Quantitative culture of prostatic fluid collected by ejaculate or...
prostatic massage has been demonstrated to have an 80% to 100% correlation with culture of prostatic tissue (Barsanti et al., 1983; Ling et al., 1983, 1990; Cowan et al., 1991). Prostatic ultrasonography is recommended for all dogs with prostatic disease because, diagnostically, this procedure can detect more than one type of prostatic disease in a single prostate, and because, therapeutically, presence of cysts or abscesses may indicate need for surgical drainage or medical therapy for BPH to shrink the prostate. B. canis serology is indicated in male dogs with prostatitis in order to rule out canine brucellosis.

Treatment strategies for dogs with prostatitis include specific antimicrobial therapy, and consideration of castration or antiandrogen therapy to cause decrease in prostatic size. Antibiotic therapy should be selected based on sensitivity of bacteria cultured from inflammatory exudates and on the ability of the antibiotic to diffuse into prostatic fluid in therapeutic concentrations. The blood–prostate barrier in the normal dog prevents diffusion of drugs with low lipid solubility or those that are highly protein bound in plasma from entering the prostatic fluid in therapeutic concentrations. In addition, pH gradients between blood and prostatic fluid may influence ionized drug trapping in prostatic fluid. In general, antibiotics known to diffuse into the prostatic fluid of the normal dog in therapeutic concentrations include trimethoprim-sulfa, chloramphenicol, and enrofloxacin, all of which are effective in treating most aerobic bacterial infections of the canine prostate (Dorfman et al., 1995). Enrofloxacin also is effective against mycoplasma infections. Trimethoprim-sulfa and enrofloxacin are not effective against anaerobic infections; chloramphenicol is indicated in these cases. Inflammatory compromise of the blood–prostate barrier in acute and chronic prostatitis, which might permit therapeutic concentrations of other antibiotics to reach the site of infection, has not been well studied. Fungal prostatitis usually is a part of systemic fungal infection in the dog, which should be treated with systemic antifungal regimens.

Surgical drainage has been recommended for prostatic abcessation, as antibiotic therapy alone will not result in cure (Mullen et al., 1990; Glennon and Flanders, 1993). Drainage is described using needle aspiration or surgical placement of multiple Penrose drains drawn through and fixed to the ventral lateral abdominal wall. Partial prostaectomy and marsupialization also are described for prostatic abscess drainage. Complications may occur with all methods. In one study of 92 dogs treated with multiple Penrose drain applications, three dogs died during surgery, and 19 died or were euthanized in the immediate postoperative period because of sepsis, shock and peritonitis (Mullen et al., 1990). Long-term follow-up of 57 dogs was associated with good to excellent results in 33, fair results in 14, and poor results in 9. Postoperative complications included painful abdomen, scrotal/preputial/hindlimb edema, hypoproteinemia, hypoglycemia, anemia, sepsis/shock, hypokalemia, and urine leakage from Penrose drains. Poor results in surgical drainage of abscessed prostates in the dog have prompted some clinicians to substitute aggressive antibiotic and antiandrogen (finasteride) therapy for this procedure.

Castration should be considered in dogs with prostatitis if the disorder is a recurrent one, if infection is associated with a hyperplastic or abscessed gland, or if reproductive potential is unimportant to the client. Castration results in prostatic atrophy, and has been shown to reduce duration of chronic bacterial prostatitis and number of bacterial colony-forming units per milliliter of urine in experimentally infected dogs (Cowan et
al., 1991) Castration is not recommended in the presence of acute infection, as such surgery may result in presence of scirrhous spermatic cords. Therapy with megestrol acetate (0.11 mg/kg per os once daily) or finasteride (5 mg per os once daily) may be used to accomplish decrease in prostate size temporarily, until infection is controlled with antibiotics and castration, or until breeding, if desired, can be accomplished.

4. Prostatic cysts

Prostatic cysts in the dog include diffuse cystic change associated with androgen-dependent BPH, as well as retention or paraprostatic cysts, which are cavitating lesions with a distinct wall, containing clear to turbid fluid, either within (retention) or outside (paraprostatic) the prostatic parenchyma (White et al., 1987). Pathogenesis is unknown, but the observation of retention cysts occurring concurrently with estrogen-secreting Sertoli cell tumors has prompted speculation that they may occur as dilation of prostatic acini secondary to estrogen-induced squamous metaplasia. Paraprostatic cysts usually arise cranialateral to the prostate, displacing the bladder cranially and ventrally, or caudal to the gland within the pelvis, as possible dilated embryonal remnants of Wolffian ducts. Mean age at time of diagnosis is reported as 8.0 years (Stowater and Lamb, 1989). Affected dogs may be asymptomatic, or signs referable to concurrent BPH or to physical displacement of abdominal viscera may be present. Diagnosis is confirmed by radiography and ultrasonography, and surgical resection, with or without concurrent castration, is recommended treatment.

5. Prostatic neoplasia in dogs

Prostatic adenocarcinoma is an uncommon, highly invasive malignant tumor of intact and castrated male dogs. Prevalence from necropsy studies is reported at 0.2–0.6% (Bell et al., 1991), and age at diagnosis has been reported to range from 5 to 17 (median 10) years (Cornell et al., 1997). Two studies suggest increased risk of prostatic adenocarcinoma in castrated male dogs compared to intact male dogs (Obradovich et al., 1987; Bell et al., 1991); in one of these, the risk of a castrated dog developing the disease was 2.38 times greater than that of an intact dog (Bell et al., 1991). Unlike prostatic adenocarcinoma in humans, this tumor in dogs does not appear to respond to androgen deprivation (antiandrogens or castration).

Environmental chemicals that have hormonal activity (environmental estrogens) or act as endocrine disruptors are theorized to cause preneoplastic or overly neoplastic changes in many tissues, including reproductive tissues. Significance of these chemicals in the pathogenesis of spontaneous prostatic adenocarcinoma in the dog is unknown.

In humans, prostatic intraepithelial neoplasia (PIN) is considered a precursor of prostate cancer (Waters, 1999; Waters and Bostwick, 1997; Waters et al., 1997b). PIN includes cytologic features of cell crowding, loss of polarity, and nuclear and nucleolar enlargement. PIN has been identified in 6 of 11 (55%) of sexually intact dogs 7–17 years old with no other evidence of prostatic disease, and in 19 of 29 (66%) of dogs with prostatic adenocarcinoma (Waters and Bostwick, 1997), suggesting that PIN may be a
precursor to adenocarcinoma in the dog as well. Foci of high-grade PIN were detected in only 1 of 11 (9%) elderly castrated dogs from 7 to 17 years of age. Canine and human PIN are similar in basal cell disruption, proliferative index, and microvessel density, suggesting that the canine prostate may be a useful model for studying carcinogenesis and prostate cancer progression in humans (Waters et al., 1997b).

Clinicopathologic findings reported in 168 dogs with prostatic adenocarcinoma include stranguria (45%), tenesmus (44%), hematuria (29%) anorexia (23%) and weight loss (15%) (Cornell et al., 1997). Radiographic findings from 185 affected dogs included: prostatic enlargement (82%), prostatic mineralization (32%), sublumbar lymphadenopathy (24%), axial skeletal metastasis (16%), lung metastasis (15%) and appendicular skeletal metastasis (8%). Ultrasonographic appearance of the neoplastic canine prostate includes prostatomegaly, mineralization of the parenchyma, presence of focal to diffuse hyperechoic areas, and irregular/discontinuous prostatic contour (Bell et al., 1991).

Prevalence of metastasis of canine prostatic adenocarcinoma, which may be as high as 80%, was reported as higher in neutered affected dogs in one study (n = 31; Bell et al., 1991) and as higher in intact affected dogs in another (n = 168; Cornell et al., 1997). Bell et al. (1991) reported that pulmonary metastases were significantly more common in neutered than intact dogs with prostatic adenocarcinoma, while Cornell et al. (1997) reported that intact dogs were 9.5 times more likely than neutered dogs to have metastatic disease to other organs. Reported sites of metastasis, from most to least common, are lungs, regional lymph nodes, liver, urethra, spleen, colon and rectum, urinary bladder, bone, heart, kidney, distant lymph nodes, and adrenal glands. Skeletal metastasis reported in 29 of 127 (23%) dogs with spontaneous prostate cancer included 59 lesions; the lumbar vertebrae and pelvis were most frequently affected, and only 7% of the lesions occurred distal to the elbow or stifle. Bone metastasis leading to myelopathy or lameness was the initial clinical manifestation of malignancy. Dogs with skeletal metastases were younger at prostate cancer diagnosis than dogs without skeletal metastases, and skeletal metastases were high-grade carcinomas (Waters et al., 1997a).

Collection of prostatic cells by transrectal or transabdominal fine needle aspiration, with or without ultrasound guided placement, is reported to diagnose prostatic carcinoma correctly in about 80% of affected dogs.

Treatment of canine prostate cancer generally is unrewarding. Castration results in involution of the non-neoplastic portion of the prostate, but does not affect progression of the neoplastic disease. Surgical resection usually is not recommended, because the disease is not usually diagnosed at an early stage, and prostatic surgery often results in urinary incontinence. External beam radiation therapy is reported to shrink some canine prostatic tumors with relief of urinary outflow obstruction and obstipation (Klausner et al., 1995). Maximum survival following radiation therapy of affected dogs at the University of Minnesota College of Veterinary Medicine has been 5 months.

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References


