Transarterial Prostatic Embolization: Initial Experience in a Canine Model

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OBJECTIVE. The purpose of this study was to prospectively evaluate pathologic responses to transarterial prostatic embolization and its technical safety in a canine model.

MATERIALS AND METHODS. Ten adult male beagle dogs were surgically castrated and given hormonal therapy for 4 months to induce prostatic hyperplasia. After three months of hormonal therapy, the dogs were randomly assigned to a transarterial prostatic embolization group ($n = 7$) or a control group ($n = 3$). Dogs in the transarterial prostatic embolization group were subjected to embolization with microspheres 300–500 μm in diameter. Four months after the study was begun, all dogs were sacrificed for pathologic study. Transrectal ultrasound and MRI were performed to evaluate pathologic responses. The data on prostate size acquired with transrectal ultrasound were processed for statistical analysis by paired Student *t* test.

RESULTS. The canine prostatic hyperplasia model was successfully established in 10 dogs. The increase in mean prostate size being as great as 572% after 3 months of hormonal therapy. An intraprostatic cavity was detected 1 month after transarterial prostatic embolization in all seven dogs. Four dogs had significant shrinkage of the prostate, and the other three had an increase in prostate size. Imaging examinations and necropsy revealed a huge cavity occupying almost the entire prostate in the three dogs with increased prostate size. No complications associated with transarterial prostatic embolization were encountered.

CONCLUSION. Transarterial prostatic embolization is a safe procedure that can induce prostatic infarction and ablate the prostate. The findings suggest the procedure has potential clinical applications in the care of patients with benign prostatic hyperplasia.

Keywords: benign prostatic hyperplasia, canine model, embolization

DOI:10.2214/AJR.10.5947

Received October 14, 2010; accepted after revision January 3, 2011.

Supported in part by grant 3PR05A098 (F. Sun) from Plan Regional de Investigación, Consejería de Educación, Ciencia y Tecnología, Junta de Extremadura (PRI).

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AJR 2011; 197:495–501

0361–803X/11/1972–495

Benign prostatic hyperplasia (BPH) is common among elderly men in Western countries and poses increasing public health concerns as the population ages. Approximately 50% of men older than 40 years will have histologic hyperplasia or BPH, and 30–50% of those men will have lower urinary tract symptoms [1]. The term lower urinary tract symptoms has been applied to any one or more of nocturia, urgency, frequency, a sensation of not completely emptying the bladder, stop-start urination, straining to urinate, a need to urinate soon after voiding, and weak urinary stream [2]. It is generally believed that lower urinary tract symptoms are a complex condition with static and dynamic components, including a mass-related increase in urethral resistance, age-related detrusor dysfunction, and abnormal neuromuscular tone at the bladder neck [3].

The various therapeutic options for relieving the lower urinary tract symptoms associated with BPH include medical, minimally invasive, and surgical therapies. Among those, transurethral resection of the prostate (TURP) has been the benchmark therapy that results in a rate of alleviation of lower urinary tract symptoms that exceeds 70% [4]. However, this mainstay therapy for BPH is associated with frequent adverse consequences, which are critical considerations in selecting treatment. During TURP for BPH, the major complication is hemorrhage, the mean transfusion rate being 8.6% [4]. Long-term complications associated with TURP include incontinence in 2.2% of patients, bladder neck contracture and urethral stricture in 3.8%, retrograde ejaculation in 65–70%, and erectile dysfunction in 6.5% [4, 5].

Development of alternatives to TURP is of great clinical importance. A new alternative should be of comparable efficacy, more cost-effective, and most important, less invasive with fewer complications affecting the quality of

life of patients. In healthy pigs, transarterial prostatic embolization has proved technically feasible, effective in destroying the prostate, and safe without major complications or adverse effects on libido or erectile function [6]. The safety and pathologic responses remain to be addressed in dogs with a pathologically tumorlike prostate. The purpose of our study was to prospectively test in a canine model the hypothesis that transarterial prostatic embolization can effectively and safely destroy a hyperplastic prostate and be an alternative to surgical therapy for BPH.

Materials and Methods

Animals

Protocols were approved by the institutional ethics committee for animal research. Ten adult male beagle dogs (weight, 8.8–14.7 kg) were assigned to a transarterial prostatic embolization group (n = 7) or a control group (n = 3). All dogs were subjected to surgical castration and subsequent hormonal therapy for establishment of a prostatic hyperplasia model. In the dogs in the transarterial prostatic embolization group, selective angiography plus embolization was performed. Only selective angiography was performed on the dogs in the control group.

Prostatic Hyperplasia Model

After a 24-hour fast, 3 mg/kg IV propofol (Diprivan, AstraZeneca) was administered to each dog for induction of anesthesia. The dog was then endotracheally intubated and connected to an anesthesia system and mechanical ventilator (Leon Plus, Heinen and Löwenstein). Anesthesia was maintained with inhaled concentrations of 3.3–3.6% sevoflurane (Sevorane, Abbott Laboratories) in oxygen during the procedure.

Castration was performed under sterile conditions. A midline incision approximately 2 cm long was made slightly cranial to the scrotum. Either testicle was exteriorized. After the spermatic cord was ligated and cut, the testicle was removed. The other testicle was removed in the same manner. After complete hemostasis was obtained, the dogs were allowed to recover from anesthesia. The dogs were given amoxicillin trihydrate plus potassium clavulanate (Synulox, Pfizer) intramuscularly 20 mg/kg daily for 5 days and carprofen (Rimadyl, Pfizer) orally 4 mg/kg twice a day for 3 days.

One month after castration, all dogs were given hormonal therapy. Twenty-five milligrams of 3α,17β-dihydroxy-5α-androstane and 0.25 mg of 17β-estradiol were dissolved in 1 mL of glycerol trioleate and then injected intramuscularly 3 times per week. The hormonal therapy lasted 4 months until the end of the study.

Angiography and Embolization

Angiography and embolization were performed after 3 months of hormonal therapy. Under general anesthesia and sterile conditions, femoral arterial access was established percutaneously. A 5-French selective catheter (Sos Omni, AngioDynamics) was placed into the right and left internal iliac arteries to obtain selective angiograms (BV Pulsera, Philips Healthcare). The dogs in the control group were then allowed to recover from anesthesia.

After selective angiography of the internal iliac artery, transarterial prostatic embolization was performed on the dogs in that group. After systemic heparinization (100 IU/kg body weight, heparin, Heparina Rovi 0.5%, Rovi), a 3-French infusion catheter (MicroFerret 18, William Cook Europe) was inserted coaxially through the angiographic catheter and selectively placed into the prostatic branch of the inferior vesical artery. Microspheres (Embosphere, Biosphere Medical) calibrated to 300–500 μm in diameter were used for embolization. Each vial of microspheres, containing 2.0 mL of particles, was diluted in a mixture of 20 mL of 50% contrast medium and 50% normal saline solution. Under fluoroscopic control, the mixture was slowly injected. Embolization was immediately terminated when complete stasis was achieved or when reflux of contrast medium toward the internal pudendal artery was observed. Embolization on the other side was performed with the same technique. After transarterial prostatic embolization, all dogs were examined twice a day for 3 days and then once a day for 4 days for possible complications associated with embolization.

Sonography and MRI

Transrectal sonography of the prostate was performed on all dogs immediately before surgical castration, hormonal therapy, angiography, or transarterial prostatic embolization and 1 month after angiography or embolization. After general anesthesia was induced, the dogs were placed in the left lateral position. The prostate was scanned with a transrectal probe (HDI 5000, Philips Healthcare). The prostate volume was assessed by the three distances method defined in the axial and sagittal planes.

MRI of four dogs was performed with a 1.5-T system (Intera, Philips Healthcare) before and 1 month after embolization. The dogs were placed in the supine position with a sensitivity-encoding coil (Flex-M, Philips Healthcare) around the lower abdomen for image acquisition. A typical MRI examination consisted of axial T1-weighted turbo spin-echo and T2-weighted turbo spin-echo images with an FOV of 14 × 14 cm and 5-mm section thickness in a 232 × 232 matrix. Additional contrast-enhanced T1-weighted images also were obtained, for which a bolus of 0.1 mmol/kg gadopentetate dimeglumine (Magnevist, Bayer Schering Pharma) was administered by manual injection into an antecubital vein.

Histopathologic Study

One month after angiography or transarterial prostatic embolization, all dogs were sacrificed by IV injection of potassium chloride solution while under anesthesia. Necropsy and gross pathologic study were immediately performed. The prostate and its surrounding structures in the pelvis were carefully inspected for pathologic changes. The prostate was removed and fixed with 10% buffered formalin solution for microscopic study. Microscopic study was conducted by two pathologists.

Statistical Analysis

A statistical software package (SPSS version 15.0, SPSS) was used for analysis. Data were expressed as mean ± SD. To evaluate the dog model of prostatic hyperplasia, the sizes of the prostate in all 10 dogs were compared between the baseline before surgical castration and the data collected 3 months after hormonal therapy. In seven dogs in the transarterial prostatic embolization group, prostate sizes immediately before embolization and 1 month after embolization also were compared. All analyses were conducted for statistically significant differences with the Student t test for paired samples; $p < 0.05$ was considered to indicate a statistically significant difference.

Results

Prostatic Hyperplasia Model

Surgical castration of all dogs was completed uneventfully. No complication was observed after surgery or during hormonal therapy. The prostatic hyperplasia model was established in all dogs.

Angiography and Embolization

Selective catheterization and angiography of both the contralateral and ipsilateral internal iliac arteries or the anterior division were successfully performed with a 5-French angiographic catheter in each procedure. Technical success of superselective catheterization to the bilateral prostatic branches with a 3-French microinfusion catheter was obtained in all cases in the transarterial prostatic embolization group. Subsequent embolization was performed with microspheres. The mean dosage of microspheres used on both sides was 1.1 ± 0.3 mL. The mean procedure duration was 112.9 ± 19.5 minutes. No complication occurred during the procedures.

Selective and superselective angiography showed the prostate as a hypervascular gland

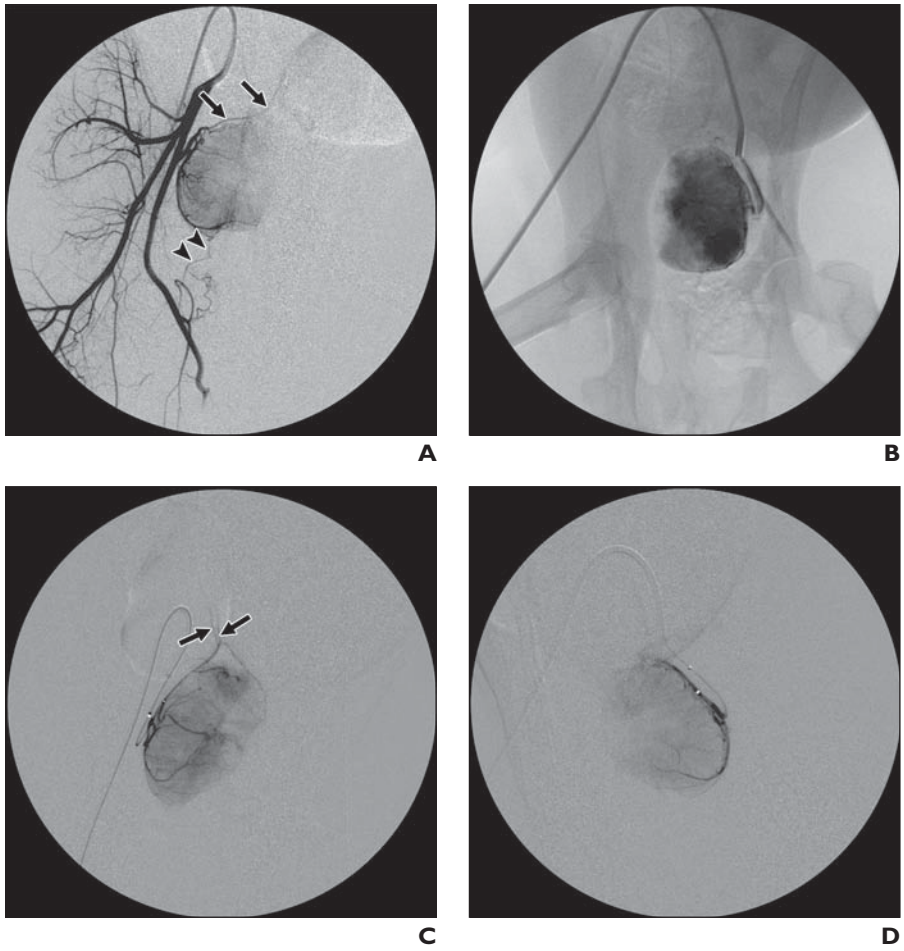


Fig. 1—Adult male dog in third month of hormonal therapy to induce prostatic hyperplasia.
A, Right anterior 20° oblique selective angiogram of right internal iliac artery shows inferior vesical branches (*arrows*) and fine branches originating from distal part of internal pudendal artery (*arrowheads*).
B, Left anterior 30° oblique late arterial phase angiogram of contralateral internal iliac artery shows uneven distribution of prostatic blush, suggesting presence of hypervascular lesion.
C and **D,** Right anterior 45° oblique (**C**) and left anterior 45° oblique (**D**) superselective angiograms obtained with microcatheter show inferior vesical branches (*arrows,* **C**).

receiving blood primarily from bilateral inferior vesical arteries. Some fine branches from the distal part of the internal pudendal artery also were found to supply the apex of the prostate. The inferior vesical artery appeared to have an enlarged diameter and was tortuous in some cases. Its upper fine branch was distributed to the fundus of the bladder. In the middle or late arterial phase, a large uneven distribution of prostatic blush was found. This finding is consistent with the angiographic features of BPH in humans [7] (Fig. 1).

No acute urinary retention, urinary incontinence, signs of peritonitis, skin or muscle ischemic necrosis in the perianal region or the buttock, or hind limb lameness were observed during the first-week veterinary observation after the procedures.

Sonography and MRI

The volumes of the prostate before surgical castration (baseline), immediately before hormonal therapy (1 month after castration), and immediately before and 1 month after embolization or angiography (third and fourth months of hormonal therapy) were measured with transrectal sonography. Compared with the baseline volume, the mean volume of the prostate 1 month after castration was significantly

TABLE 1: Body Weight and Prostate Volume During Induction of Benign Prostatic Hyperplasia

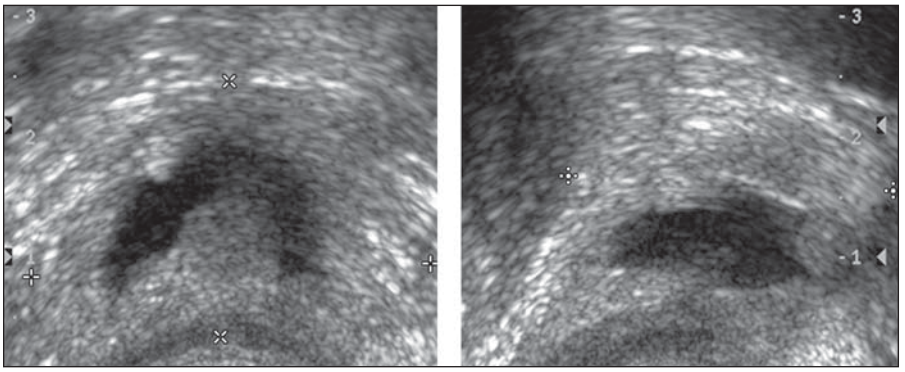
Dog No.	Body Weight (kg)	Prostate Volume (mL)		
		Before Castration	Before Hormonal Therapy	Before Intervention
Transarterial prostatic embolization				
1	11.3	9.28	0.94 (10.1)	29.16 (314.2)
2	12.5	4.21	1.36 (32.3)	29.09 (691.0)
3	12.0	6.72	2.15 (32.0)	36.11 (537.4)
4	14.2	3.07	1.06 (34.5)	29.07 (946.9)
5	14.7	5.05	1.00 (19.8)	36.88 (730.3)
6	13.5	5.19	1.34 (25.8)	29.10 (560.7)
7	13.6	6.00	1.63 (27.2)	39.96 (666.0)
Control				
8	12.1	3.18	1.09 (34.3)	18.44 (579.9)
9	8.8	7.50	0.84 (11.2)	27.96 (372.8)
10	12.4	4.87	1.54 (31.6)	38.52 (791.0)
Mean ± SD	12.8 ± 1.8	5.50 ± 1.93	1.30 ± 0.40 (23.6)	31.43 ± 6.47 (571.5)

Note—Values in parentheses are percentage change in volume compared with volume before castration.

TABLE 2: Results of Transarterial Prostatic Embolization

Dog No.	Prostate Volume (mL)		Dose of Microspheres (mL)
	Before Intervention	1 Month After Intervention	
1	29.16	41.51 (142.4)	1.2
2	29.09	19.87 (68.3)	0.7
3	36.11	54.67 (151.4)	0.9
4	29.07	16.86 (58.0)	1.5
5	36.88	65.39 (177.3)	1.3
6	29.10	9.95 (34.2)	1.4
7	39.96	13.48 (33.7)	1.0
Mean ± SD	32.77 ± 4.72	31.68 ± 22.08	1.1 ± 0.3

Note—Values in parentheses are percentage change in volume compared with volume before intervention.



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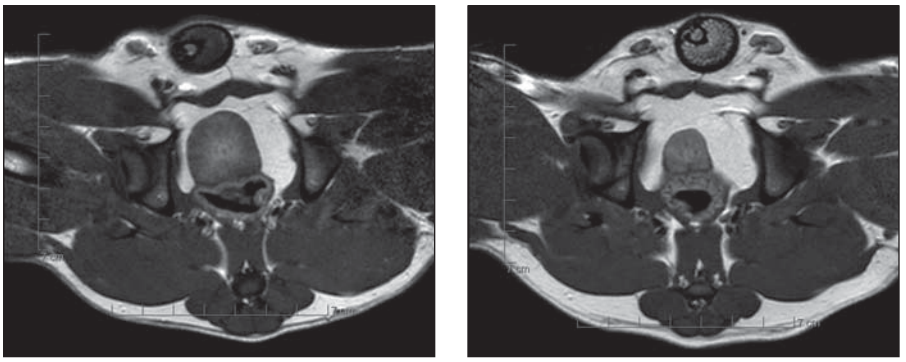

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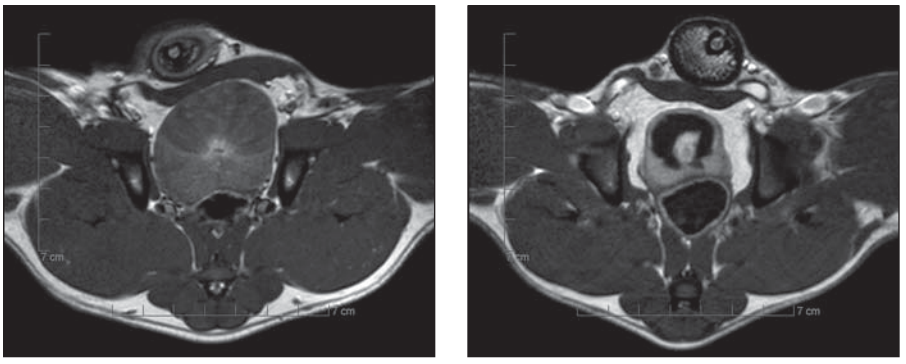

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decreased ($p < 0.001$). The mean volume after 3 months of hormonal therapy was increased as much as 572% (Table 1). In four of the seven dogs subjected to transarterial prostatic embolization, prostate volume decreased to 33.7–68.3%. In the other three, the volume increased to 142.4–177.3% 1 month after embolization (Table 2). The results of the Student *t* test for paired samples suggested no overall significant changes ($p = 0.894$) in mean volume from before to 1 month after embolization. Sonography (Fig. 2A) and MRI (Figs. 2B–2E) 1 month after transarterial prostatic embolization showed various sizes of cavitary necrosis in all dogs.

Histopathologic Study

In all dogs, the surrounding structures, including the ureters, seminal vesicles, deferent ducts on both sides, urinary bladder, urethra, and sigmoid colon, were intact at gross visual inspection. No necrosis or damage in either the internal or the external urethral sphincter was found at necropsy. A slight adhesion between the posterior surface of the prostate and the anterior wall of the rectum was found in two dogs subjected to embolization. Intraprostatic cavities had formed in all the dogs subjected to embolization (Fig. 3). At gross visual inspection, one of the three prostates that became enlarged after embolization had complete cavity formation without residual gland along the inner cystic wall (Fig. 4A). This finding was consistent with the imaging findings (Fig. 4B). The other two dogs had a large cavity with less than 10% residual gland tissue.

In the three control dogs, the findings at microscopic examination suggested massive prostatic hyperplasia with extensive acinar dilatation lined by prominent hypertrophied

Fig. 2—Adult male dog subjected to transarterial prostatic embolization (dog 6).
A, Transrectal ultrasound image 1 month after procedure shows prostate in axial (left) and sagittal (right) planes. Intraprostatic cavity and residual gland in prostate are evident.
B, Transverse T1-weighted contrast-enhanced turbo spin-echo MR image shows conditions before surgical castration.
C, Transverse T1-weighted contrast-enhanced turbo spin-echo MR image shows conditions immediately before hormonal therapy.
D, Transverse T1-weighted contrast-enhanced turbo spin-echo MR image shows conditions in third month of hormonal therapy, immediately before transarterial prostatic embolization.
E, Transverse T1-weighted contrast-enhanced turbo spin-echo MR shows conditions 1 month after transarterial prostatic embolization.

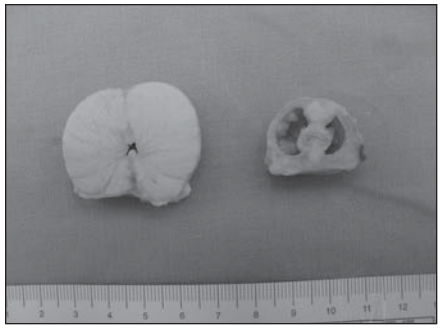

Fig. 3—Adult male control dog (dog 9) and dog subjected to transarterial prostatic embolization (dog 6). Photographs of gross specimens show volume of control prostate is 28.22 mL at end of study and embolized prostate is 9.95 mL 1 month after procedure. Prostate size immediately before embolization was 29.10 mL, similar to size of control. Prostate size, intraprostatic cavity formation, and residual gland have decreased markedly 1 month after transarterial prostatic embolization.

epithelial cells. The interstitium was inapparent, and inflammation was absent. The major microscopic findings in the embolized prostates included cavitary necrosis in the central areas of both lobes, which were lined with atrophied glands, inflammatory cell infiltration, and fibrosis. Some residual islands of normal glandular hyperplasia were also seen in the periphery of the gland. In the case of complete cavity formation without residual gland at gross visual inspection, only very little residual gland was observed microscopically (Fig. 4C).

Discussion

Dogs are the only large mammals other than humans that experience spontaneous prostatic hyperplasia and prostate cancer. Spontaneous BPH in dogs begins as glandular hyperplasia when the dog is as young as 3 years. Almost all intact male dogs have BPH, more than 95% by the age of 9 years [8, 9]. Prostate cancer is rare in dogs. Adenocarcinoma is the most common prostatic malignancy, occurring in 8- to 10-year-old dogs [9]. Canine models of spontaneously occurring prostate cancer, although optimal for preliminary studies, are difficult to use because of a lack of commercially available old male dogs and old dogs with prostate cancer.

Hormone-induced canine prostatic hyperplasia has been a well-established model since the first report by Walsh and Wilson [10]. A number of investigators have used this model to study the cause of BPH, test new drugs for medical therapy, and evaluate minimally invasive therapeutic options [11–13]. In this study, we used hormone-induced canine prostatic hyperplasia to evaluate transarterial prostatic embolization with a focus on technical safety and pathologic response. The dog model was successfully established in all 10 beagles after 3 months of hormonal therapy with a 572% increase in mean prostate size compared with the size before surgical castration. Microscopic study of the three control dogs revealed glandular hyperplasia and hypertrophied epithelial cells, which were consistent with previous evidence [10, 14] that suggested the experimentally induced glandular hyperplasia was histologically indistinguishable from the spontaneous disease. Other advantages of the model are its similarities to human anatomy and physiology, particularly with respect to the urogenital and vascular systems. It therefore may well meet the needs for preclinical studies of transarterial prostatic embolization.

The canine model has limitations, however. Because the dogs are castrated before hormone therapy, sexual function—libido, erectile function, and ejaculation—cannot be evaluated after transarterial prostatic embolization. Unlike men, dogs with BPH rarely have lower urinary tract symptoms because the canine prostate lacks a capsule [15]. In dogs, hyperplastic growth expands outwardly in all directions, usually producing rectal compression and constipation [16]. Thus it is not feasible to assess urodynamic changes associated with transarterial prostatic embolization either in dogs with spontaneous BPH or in this canine model of hormone-induced hyperplasia. In addition, the prostate-specific antigen assay commonly used to predict therapeutic effects in men cannot be used in the canine model because prostatic-specific antigen is not detected in the serum of healthy dogs or dogs with BPH [16, 17].

Technical safety is a major concern before transarterial prostatic embolization is used as a primary treatment in clinical trials. In our study, seven dogs were subjected to bilateral transarterial prostatic embolization without complications either during the procedure or in the 1-month follow-up period. At necropsy, apart from slight adhesion between the prostate and rectum in two dogs subjected to embolization, all surrounding structures were intact at gross visual inspection. The foregoing findings suggest that transarterial prostatic embolization is technically safe. Untoward embolization may be a potential complication,

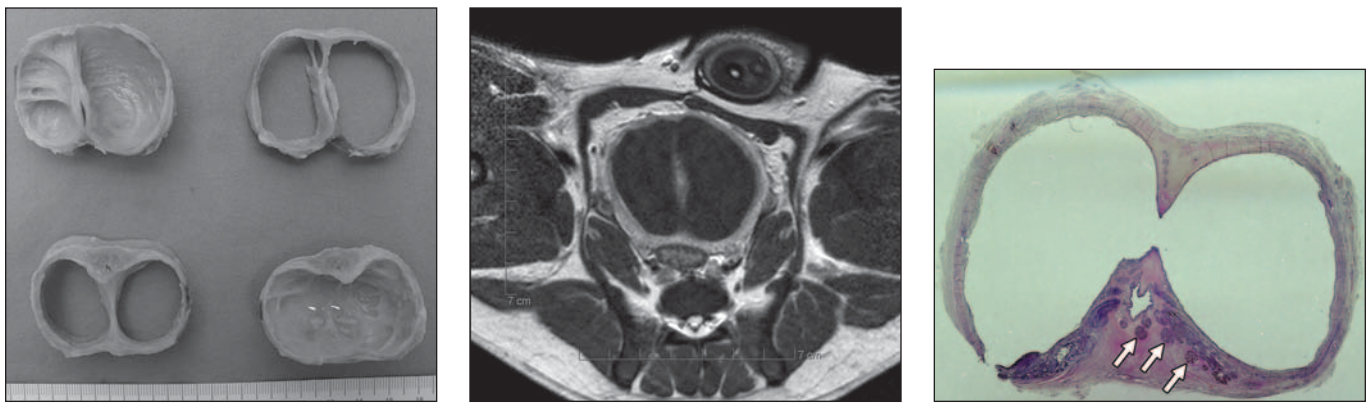

Fig. 4—Adult male dog subjected to transarterial prostatic embolization (dog 5).
A, Photographs of gross specimens show evidence of complete intraprostatic cyst formation without residual gland.
B, Transverse T1-weighted contrast-enhanced turbo spin-echo MR image shows conditions immediately before sacrifice.
C, Photomicrograph (H and E, ×1.1) of histologic section shows partial residual gland (*arrows*) in prostate.

most frequently due to reflux of the embolic agent during injection to the nontarget vessels. Jeon et al. [18] performed transarterial prostatic embolization on five beagles and after the procedure found focal bladder hemorrhage in one dog, but the bleeding did not involve the entire thickness of the bladder wall. In that case, no symptoms or signs were observed after embolization. In a report on transarterial prostatic embolization in six dogs [19], urinary retention occurred in one dog on the second day after embolization, and bladder catheterization was required.

Cavitary necrosis as a result of transarterial prostatic embolization is common. In our study, lesions of cavitary necrosis of various sizes were found in all dogs at ultrasound and MRI examinations 1 month after transarterial prostatic embolization. Cavitary necrosis and large cyst formation also have been described in reports of other experimental studies performed to test transarterial prostatic embolization [18, 19]. The prostate contains a massive amount of glandular tissue that produces and stores prostatic fluid. When a large amount of necrosis occurs in the prostate, the area of necrosis gradually sloughs, and an intraprostatic cavity forms. Results of a previous histopathologic study [20] suggested that sloughing in the prostate occurs by surface liquefaction, cavitation of necrosis, and to a lesser degree, formation of granulation tissue at the margins. Intraprostatic cavities in dogs and men after imaging-guided ablation therapy have been widely described [20–25]. In dogs, intraprostatic cavities appeared 1 week after ethanol injection and 2–3 weeks after laser therapy [20, 21]. In patients, intraprostatic cavities were found 6–12 weeks after laser or high-intensity focused ultrasound ablation [20, 25]. The cavities induced by high-intensity focused ultrasound ablation were reported to be present 12 months after treatment [25]. The difference between dogs and men with respect to intraprostatic cavity was attributed to the dominantly glandular nature of the canine prostate and the dense fibromuscular composition of the human prostate stroma [20].

Our data at 1-month imaging follow-up examinations showed inconsistent changes in prostate size. The prostate shrank remarkably in four dogs with mean reduction of 48.6% ± 17.4% compared with the findings immediately before transarterial prostatic embolization. In the other three dogs, the mean size increased 157.0% ± 18.1%. Thus there was no overall significant difference (p = 0.894) in change in prostate size. More important, the imaging examinations and histopathologic study revealed that the enlarged prostates in all three dogs consisted of a large intraprostatic cavity occupying almost all of the prostate. In the four dogs with prostates that shrank, only small or medium-sized intraprostatic cavities with partial residual glands were found.

It is generally believed that transarterial prostatic embolization can induce ischemic necrosis and subsequent liquefaction within the prostate and that the prostate shrinks as the liquefied necrotic tissue is absorbed. Our findings, however, suggest that the prostate may become enlarged 1 month after transarterial prostatic embolization. We speculate that the increase in prostate size after transarterial prostatic embolization resulted mainly from massive ischemic necrosis, liquefaction of necrotic tissue, and the associated inflammatory reaction. In addition, because transarterial prostatic embolization destroys almost all the gland and its vasculature inside the prostate, after a large intraprostatic cavity with a prostatic pseudocapsule forms, the large amount of liquid in the cavity cannot be absorbed in 1 month. Although the size of the prostate increased in three dogs 1 month after embolization, formation of a large cavity indicates embolization was more effective in the three dogs than in the four dogs in which the size of the prostate decreased. Accordingly, in addition to shrinkage of the prostate, formation of a large intraprostatic cavity after transarterial prostatic embolization may be another factor for evaluating the effectiveness of transarterial prostatic embolization.

The goal of therapy for BPH is to relieve lower urinary tract symptoms by decompressing the prostatic urethra. The results of our study showed that transarterial prostatic embolization can induce prostatic infarction and secondary shrinkage of the prostate gland and intraprostatic cavitary necrosis around the prostatic urethra. These findings suggest that transarterial prostatic embolization may relieve lower urinary tract symptoms in patients with BPH. The effectiveness of transarterial prostatic embolization in the management of BPH has been proved in clinical trials. DeMeritt and colleagues [7], in a clinical case report, described performance of transarterial prostatic embolization on a patient with BPH and severe gross hematuria that required blood transfusion. The patient stopped bleeding immediately after embolization, lower urinary tract symptoms were greatly relieved, and the prostate had shrunk to 62% of its most enlarged volume during 12 months of follow-up. Carnevale and colleagues [26] reported use of transarterial prostatic embolization as primary treatment of BPH in two patients. Both patients experienced acute urinary retention due to BPH and underwent transurethral catheter drainage. The two patients urinated spontaneously when the urethral catheter was removed 10 and 15 days after transarterial prostatic embolization. They continued to void normally as of the 6-month follow-up evaluation.

A major limitation of this study was that we did not address the technical reasons for the inconsistent pathologic response to transarterial prostatic embolization, that is, the findings of large intraprostatic cavities in three dogs and of small or medium-size cavities in four dogs. Further studies are needed with more dogs and a focus on technical issues, such as the ideal embolic agent, proper size of embolic agent, and endpoints of injection of embolic agent to define specific transarterial prostatic embolization techniques in the management of BPH. The other limitation of the study was the short follow-up period (1 month) after embolization. To evaluate chronic pathologic reactions of the prostate to transarterial prostatic embolization, especially changes in the size of the intraprostatic cavities, a long-term follow-up study is necessary.

Regarding concern whether the large intraprostatic cavities with increased overall prostate size might accentuate outflow symptoms, we were not able to address this issue using the canine model, as described earlier [15, 16]. A clinical trial of the management of BPH with high-energy transurethral microwave therapy [22] showed that intraprostatic cavities were found in 35 of 83 patients (42%) and that the presence of a cavity correlated positively with improvement in urinary performance and relief of outlet obstruction. At 3-month follow-up evaluation, the mean improvement in peak urine flow rate was 8.5 ± 7.3 mL/s in patients with a cavity and 4.8 ± 5.4 mL/s in patients without a cavity. We believe, however, that large intraprostatic cavities with increased overall prostate size can be easily drained when necessary under the guidance of transrectal sonography in patients with BPH after transarterial prostatic embolization.

Conclusion

Our study showed that transarterial prostatic embolization can be successfully performed in a canine model of hormone-induced prostatic hyperplasia. The technique

is safe and has no major complications. Two typical responses to transarterial prostatic embolization after 1 month of follow-up were shrinkage of the prostate with small or medium-size intraprostatic cavities and increased prostate size with large intraprostatic cavities. These findings suggest a possible application in the care of patients with BPH.

Acknowledgments

We thank Virginio Garcia-Martinez, Laura Luis, and Juan R. Lima for comments on the manuscript and technical support in the animal experiments.

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