Abdominal Radiology

Published online: 8 September 2018

Abdom Radiol (2019) 44:713–722
https://doi.org/10.1007/s00261-018-1757-z

MR imaging findings of the prostate gland following prostate artery embolization: results from a prospective phase 2 study

Rehan Ali,[1] Ahmed Gabr,[1] Samdeep K. Mouli,[1] Joseph Ralph Kallini,[1] Ahsun Riaz,[1] Ronald Mora,[1] Robert J. Lewandowski,[1,3] Elias Hohlastos,[1] David D. Casalino,[2] Matthias D. Hofer,[5] Nabeel Hamoui,[5] Frank H. Miller,[2] John Hairston,[5] and Riad Salem [1,3,4]

[1]Department of Radiology, Section of Interventional Radiology, Northwestern Memorial Hospital, Robert H. Lurie Comprehensive Cancer Center, 676 N. St. Clair, Suite 800, Chicago, IL 60611, USA
[2]Department of Radiology, Section of Body Imaging, Northwestern Memorial Hospital, Robert H. Lurie Comprehensive Cancer Center, Chicago, IL, USA
[3]Division of Transplantation, Department of Surgery, Comprehensive Transplant Center, Northwestern University, Chicago, IL, USA
[4]Division of Hematology and Oncology, Department of Medicine, Robert H. Lurie Comprehensive Cancer Center, Northwestern University, Chicago, IL, USA
[5]Department of Urology, Robert H. Lurie Comprehensive Cancer Center, Northwestern University, Chicago, IL, USA

Abstract

Purpose: To assess changes in imaging and volume characteristics of the prostate gland by magnetic resonance (MR) following prostatic artery embolization (PAE) for benign prostate hyperplasia.
Methods: With IRB approval, we analyzed prospectively acquired MR data of PAE patients at baseline and 6-month following treatment from 2015 to 2017. We reviewed prostate MRs looking for sequelae of embolization [changes in signal intensity and/or enhancement, infection/inflammation, infarction, edema, and change in intravesical prostatic protrusion (IPP)]. We calculated the total volume (TV) and central gland volumes (CGV) using DynaCAD® and measured change in volumes. Analyses were performed using SPSS with $p < 0.05$ considered significant.
Results: Forty-three patients ($n = 43$) met our inclusion criteria. 93% (30/43) and 100% (43/43) showed a decrease in TV and CGV at 6-months respectively. At baseline, median TV was 86 cc (range 29.4–232) and median CGV was 54.4 cc (range 12.9–165.5). Median decrease in TV was 18.2% (CI 13.3–27.2) ($p = 0.0001$) and median decrease in CGV was 26.7% (CI 20.4–35.9) ($p = 0.0001$). Thirty-seven percent (16/43) of patients had IPP at baseline; 100% showed a decrease in size of median lobe at follow-up. At 6-month follow-up, 33% (14/43) showed imaging features of infarction, 79% (34/43) had decrease in T2-signal intensity, and 51% (22/43) showed a decrease in enhancement. None had edema, peri-prostatic fat changes or infection/inflammation.
Conclusion: PAE causes a statistically significant reduction in the TV and CGV. There is also a reduction of the degree of IPP. Non-specific findings of infarction, decrease in T2-signal, and enhancement were also seen.

Key words: Prostate artery embolization—MRI prostate—Volumetric assessment—Prospective data

Benign prostatic hyperplasia (BPH) results in prostatic enlargement and compression of the urethra. Urethral compression results in lower urinary tract symptoms (LUTS) including urinary frequency, weak stream, and nocturia [1]. Bladder outlet obstruction (BOO) from BPH may lead to urinary retention, incontinence, hematuria, bladder calculi, urinary tract infection, and renal insufficiency. Additionally, there is often a significant reduction in quality-of-life (QoL). The prevalence of histological BPH increases from 50% of 50-year-old

Correspondence to: Riad Salem; *email:* r-salem@northwestern.edu

males to > 80% in those aged > 80. Moderate to severe LUTS occurs in 43%, 41%, and 46% of men in their 50, 60, and 70 s, respectively [2]. BPH-related symptoms and subsequent deterioration of QoL results in a significant impact on the health care system, accounting for 4.5 million visits to physician in the year 2000 [3]. Treatments for BPH include pharmacological therapies, minimally invasive thermotherapy [transurethral needle ablation of the prostate (TUNA), transurethral microwave therapy (TUMT)], prostatic urethral lift (PUL), transurethral resection or ablation of prostate [transurethral resection of prostate (TURP), laser enucleation, vaporization], and prostatectomy [4–8]. While the efficacy of most non-pharmacological interventions for BPH/LUTS has been established, attendant procedural morbidity and associated effects of recovery, failure to relieve symptoms, and/or patient discomfort represent limitations of current standards of care [9].

Innovative technologies are needed to improve outcomes of LUTS secondary to BPH and to overcome the shortcomings of current procedures (e.g., bleeding, ejaculatory dysfunction, and post-operative irritative urinary symptoms). Prostatic arterial embolization (PAE) is an innovative new method to treat BPH that shares the same mechanism of action as uterine artery embolization for leiomyomas [10]. In an early study, DeMeritt reported successful BPH treatment using 300–500 micron microspheres [11]. More recently, there have been reports of PAE in animal models demonstrating promising outcomes, with a decrease in prostate perfusion, volume, and necrosis at histopathology [12–14]. Carnevale published short-term outcomes of PAE in two patients, with symptom improvement, decrease in prostate volume, and intravesical prostate protrusion (IPP) [15]. Most recently, Carnevale reported a 30% reduction of prostate volume and marked improvement in urodynamic testing [16]. The largest series comes from Pisco, where 630 PAEs demonstrated excellent outcomes [17]. While clinical results have been published, there are no contemporary reports on the MR findings following PAE. Herein, we describe the imaging findings of the first, prospective study of PAE for BPH.

Prostate artery embolization (PAE) is a novel transarterial method of treating BPH [18]. Retrospective analyses have demonstrated that PAE results in decreased perfusion and a reduction in gland size with resultant improvement in BPH/LUTS [19–21]. Given these findings, we initiated a prospective, controlled phase 2 study of PAE in BPH/LUTS patients. Herein, we report on the imaging and volume characteristics of the prostate gland using magnetic resonance (MR) following prostatic artery embolization (PAE) for benign prostate hyperplasia (BPH).

Method

Patients cohort/selection

This study was compliant with Health Insurance Portability and Accountability Act (HIPAA) and approved by local Institutional Review Board (IRB). All patients included in the study provided written informed consent for treatment. The patient cohort was derived from our 45 patients, FDA-approved, prospective, open-label investigational device exemption study performed from August 2014—July 2017 for patients with BPH/LUTS refractory to pharmacotherapy treated with prostate artery embolization (PAE) [22]. Inclusion criteria were: age > 45, BPH-related LUTS refractory to/contraindicated for medical treatment, prostate volumes of > 40 grams, IPSS > 13 and QoL score > 2, peak flow rate (Qmax) ≤ 12 with voided volume ≥ 125 cc with completed MR scans at baseline and 6-months follow-up (Fig. 1). Exclusion criteria included: prostate/bladder cancer, stones, urethral strictures, neurogenic/hypotonic bladder, prior treatment for urinary incontinence, penile prosthesis, artificial urinary sphincter, prostatitis, pelvic radiation, and prostate specific antigen (PSA) > 4.0(unless malignancy ruled out). Forty-five patients (n = 45) underwent PAE at our institution during this time period. 4% (2/45) of patients were excluded from the imaging analysis due to non-compliance with the 6 months imaging follow-up. As a result, 96% (43/45) of patients were analyzed.

Baseline characteristics

Age, baseline serum prostate specific antigen (PSA), and free PSA were evaluated. At baseline, patients underwent magnetic resonance imaging (MRI). A multi-parametric 3.0 Tesla MRI (AchievaTX; Philips) was performed using a torso phased-array coil and including multiplanar T2-weighted images, axial T1-weighted images, axial diffusion-weighted images, and dynamic contrast-enhanced (DCE) images of the prostate.

Prostate artery embolization

Embolization was performed under local anesthesia and conscious sedation via the right common femoral artery. Initially, pelvic angiography was performed to evaluate the iliac and prostatic arteries. Subsequently, the left prostatic artery was catheterized off the anterior division of the left hypogastric artery. Perfusion of the prostate was confirmed by cone-beam CT. Embolization of the left prostatic artery was performed using Embospheres® 300–500 um particles (Merit Medical, Utah). This was repeated for the right prostatic artery. The end-point chosen for embolization was arterial stasis.

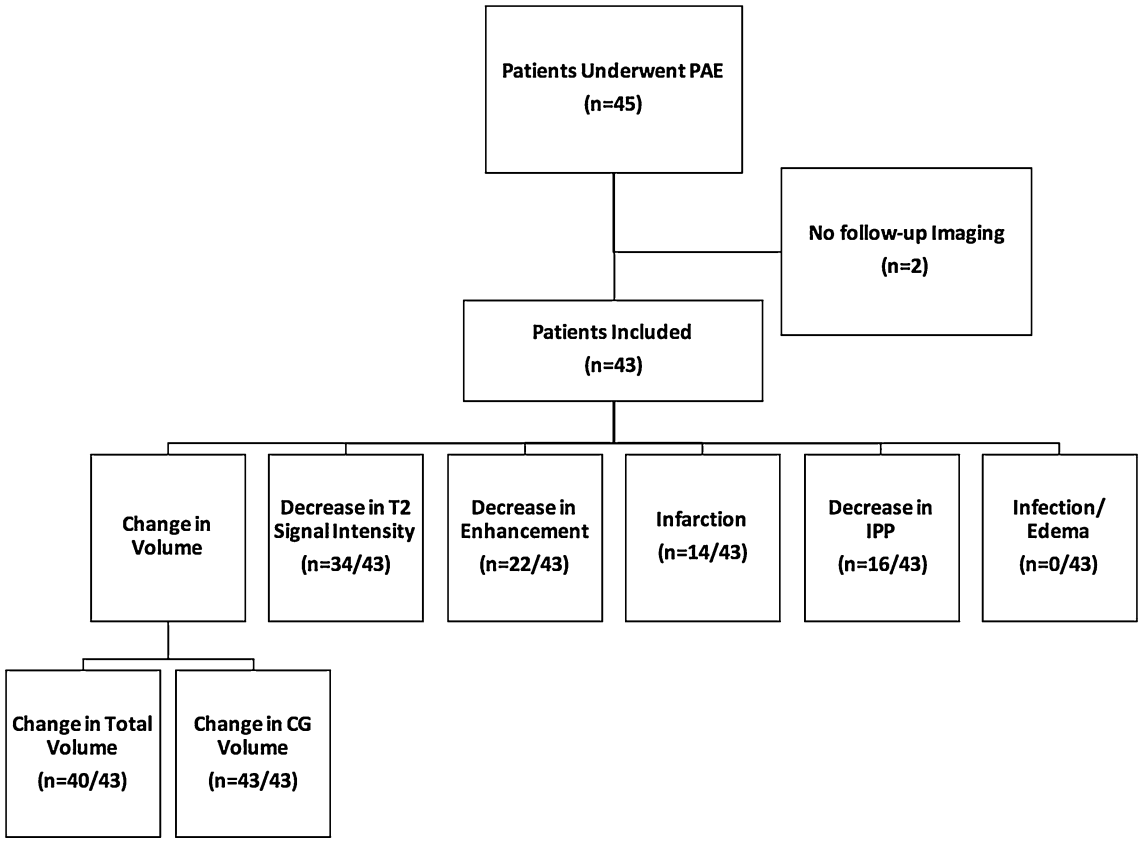

Fig. 1. Study flow chart. PAE, prostate artery embolization; CG, central glad; IPP, intravesical prostatic protrusion.

Imaging characteristics

By protocol, patients had MR imaging at baseline and at 6 months post-PAE. Baseline and follow-up MRI were performed using the same field strength and imaging sequences to counter substantial impact on signal intensity and imaging appearance to avoid bias in signal intensity evaluation. For IPP assessments, mainly sagittal views were used. All MRI examinations were interpreted by seven abdominal radiologists with 4–24 years of experience with prostate MRI. Findings were catalogd by an abdominal radiologist and researcher with 24 and 3 years of experience with prostate MRI, respectively. MRIs were analyzed for changes in prostate size; changes in T2 signal intensity (normal to decreased relative to the adjacent prostate and more similar in signal intensity to muscle); diminished enhancement compared to pre-treatment exams relative to the unenhanced images; sites of infection/inflammation (as indicated by new site of decrease T2 signal intensity and increased and early enhancement compared to normal prostate tissue); stranding of the periprostatic fat that might reflect post-treatment infection/inflammation or edema; abscess (as indicated by new fluid collection with restricted diffusion and perhaps peripheral enhancement); and infarction (as indicated by new areas that show no evidence of enhancement after contrast administration).

Volumetric analyses

Baseline and 6-month MR prostate volumes were measured through DynaCAD Prostate® (Invivo Diagnostic Imaging, Philips) including the total gland volume (TV) and central gland volume (CGV). Percentage change in TV and CGV was calculated. Change in size of intravesical prostatic protrusion (IPP) was also documented. IPP is an anatomical configuration measured as vertical distance from the tip of the protruding prostate to the base of the bladder at the base of the prostate gland [23]. Substratification analyses by baseline prostate volume < 80 and ≥ 80 cc were performed. TV and CGV changes were compared using independent t test among patients who received unilateral PAE treatment vs bilateral PAE treatments. All analyses were performed using IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp; $p < 0.05$ was considered significant.

Results

Patient Cohort

Forty-three patients met the inclusion criteria and were included in this study. Median age was 68 years (range 47–87). At baseline, median total PSA was 4.1 ng/dL (range 0.11–13.9) and free PSA was 0.78 ng/dL (range 0.04–4.2). Eighty-eight percent of the patients (38/43) had a single treatment session of PAE while 12% (5/43) had two treatment sessions because of either failure of first treatment ($n = 1$), separate sessions for left and right side of prostate ($n = 3$), and/or aborting procedure due to increased radiation dose during first treatment session (large body habitus, $n = 1$) (Table 1). There was no statistically significant TV change ($p = 0.69$) or CGV change ($p = 0.44$) in patients who received unilateral ($n = 4$) vs bilateral ($n = 39$) PAE treatments for BPH. Median total prostate and central gland volumes were 86 cc (range 29.4–232) and 54.4 cc (range 12.9–165.5), respectively.

Volumetric analyses

Ninety-three percent (40/43) of the patients had a decrease in the total prostate volume and 100% (43/43) had central gland volume reduction. Median decrease in total prostate volume was 18.2% (CI 13.3–27.2) and median decrease in central gland volume was 26.7% (CI 20.4–35.9) (Table 2). For the overall cohort, observed median decrease in total and central gland (CG) volume was 21.4 cc (CI 14.9–28.1) ($p < 0.0001$) and 18.6 cc (CI 13.8–23.4) ($p < 0.0001$) at 6-months (Fig. 2A, B). When substratified by baseline prostate volume (PV) ≤ 80 cc ($n = 18$), median decrease of 8.7 cc (CI 4.7–12.7) and 9.1 cc (CI 6.7–11.6) was observed in the TV ($p = 0.0003$) and CGV ($p < 0.0001$). For baseline PV > 80 cc ($n = 25$), median decrease of 30.6 cc (CI 20.9–40.2) and

Table 1. Baseline characteristics

Variable		Total cohort ($n = 43$)
Age (years)	Mean (range)	68 (47–87)
PSA (ng/mL)		
Total	Median (range)	4.1 (0.11–13.9)
Free	Median (range)	0.78 (0.04–4.2)
Number of treatment sessions	One	38 (88%)
	Two	5 (12%)
Prostate arteries embolized	Unilateral	4 (9%)
	Bilateral	39 (91%)
Baseline prostate volume (cc)		
TV	Median (range)	86 (29.4–232)
CGV	Median (range)	54.4 (12.9–165.5)

PSA, prostate specific antigen; TV, total prostate volume; CGV, central gland volume

Table 2. Prostate volumes pre and post embolization

Volume	Total cohort ($N = 43$) (cc)				Patients with baseline total prostate volume ≤ 80 cc ($n = 18$) (cc)				Patients with baseline total prostate volume > 80 cc ($n = 25$) (cc)			
	Pre PAE	Post PAE	% Δ	*p* value	Pre PAE	Post PAE	% Δ	*p* value	Pre PAE	Post PAE	% Δ	*p* value
Total prostate volume												
Median	86.4	74.4	18.2	< 0.0001	51.6	43.2	14.4	0.0002	135.0	107.5	21.6	< 0.0001
Mean	101.5	80.1	19.2		53.3	44.7	16.6		136.2	105.6	21.4	
Range	29.4–232	25.3–158.2	10.4–44.4		29.4–79.6	25.2–81.3	8.2–39.4		82.3–232	59.7–158.2	10.4–44.3	
Central gland volume												
Median	54.4	44.0	26.7	< 0.0001	30.3	20.9	31.3	< 0.0001	91.9	73.1	26.7	< 0.0001
Mean	67.0	48.4	27.9		31.1	21.9	30.2		92.9	67.5	26.5	
Range	12.8–165.5	7.1–131.7	3.5–47.0		12.9–56.4	7.1–44.1	12.1–45.3		41.0–165.5	21.9–131.6	3.5–47.0	

Nota bene: percentage reduction in mean/median is NOT the mathematical reduction of the mean/median itself
PAE, prostate artery embolization; % Δ, percentage reduction

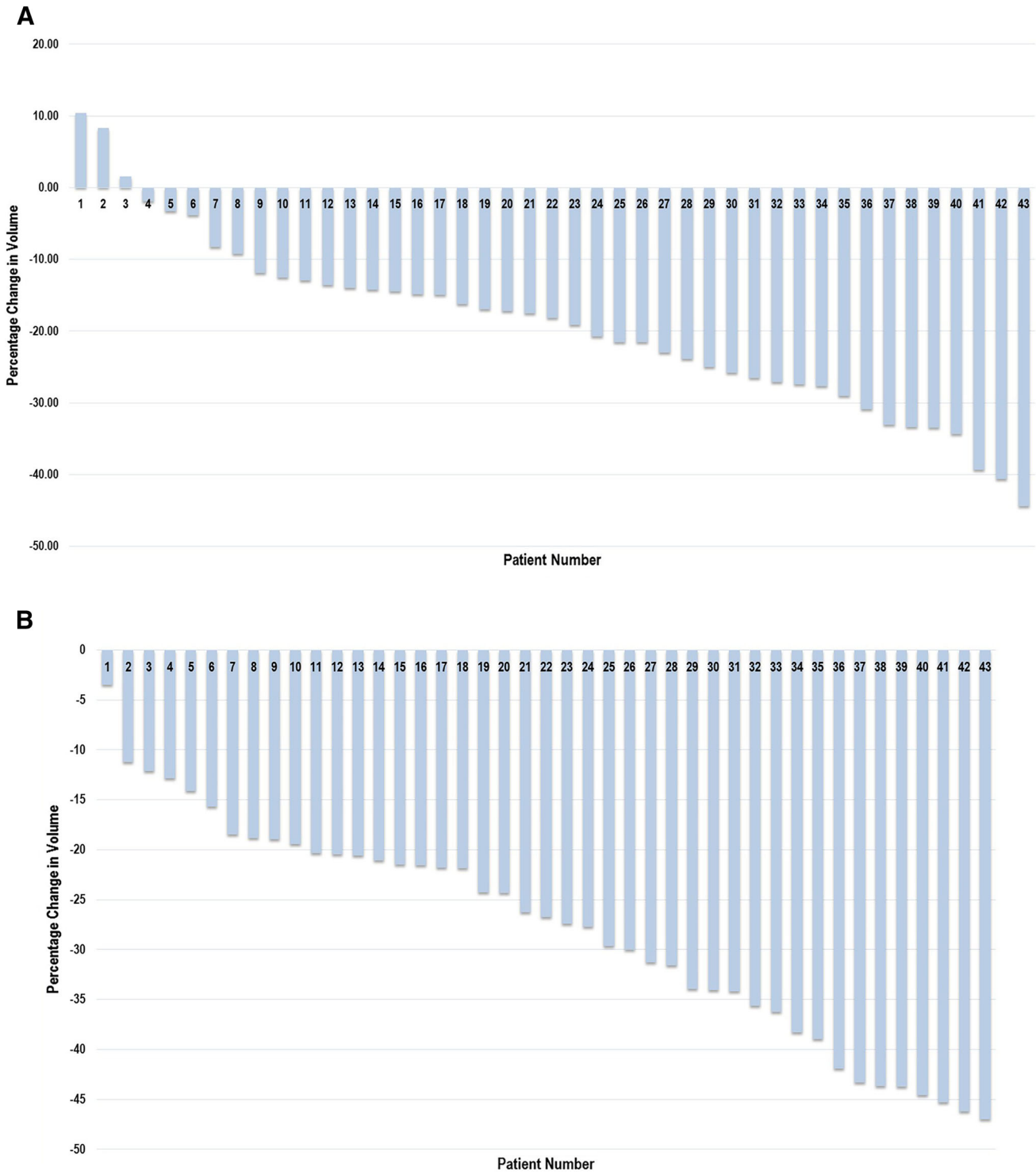

Fig. 2. **A** Percentage change in total volume of prostate. **B** Percentage change in central gland volume of prostate.

25.5 cc (CI 18.4–32.5) was observed in TV ($p < 0.0001$) and CGV ($p < 0.0001$) (Fig. 3A, B). Three patients had 1.5%, 7.6% and 9.4% increase in total prostate volume following treatment while all of them had decreased CGV (Figs. 4, 5).

Changes in imaging characteristics

No patient demonstrated findings of infection/inflammation, abscess, or changes in the periprostatic fat. Seventy-nine percent (34/43) of patients had qualitative

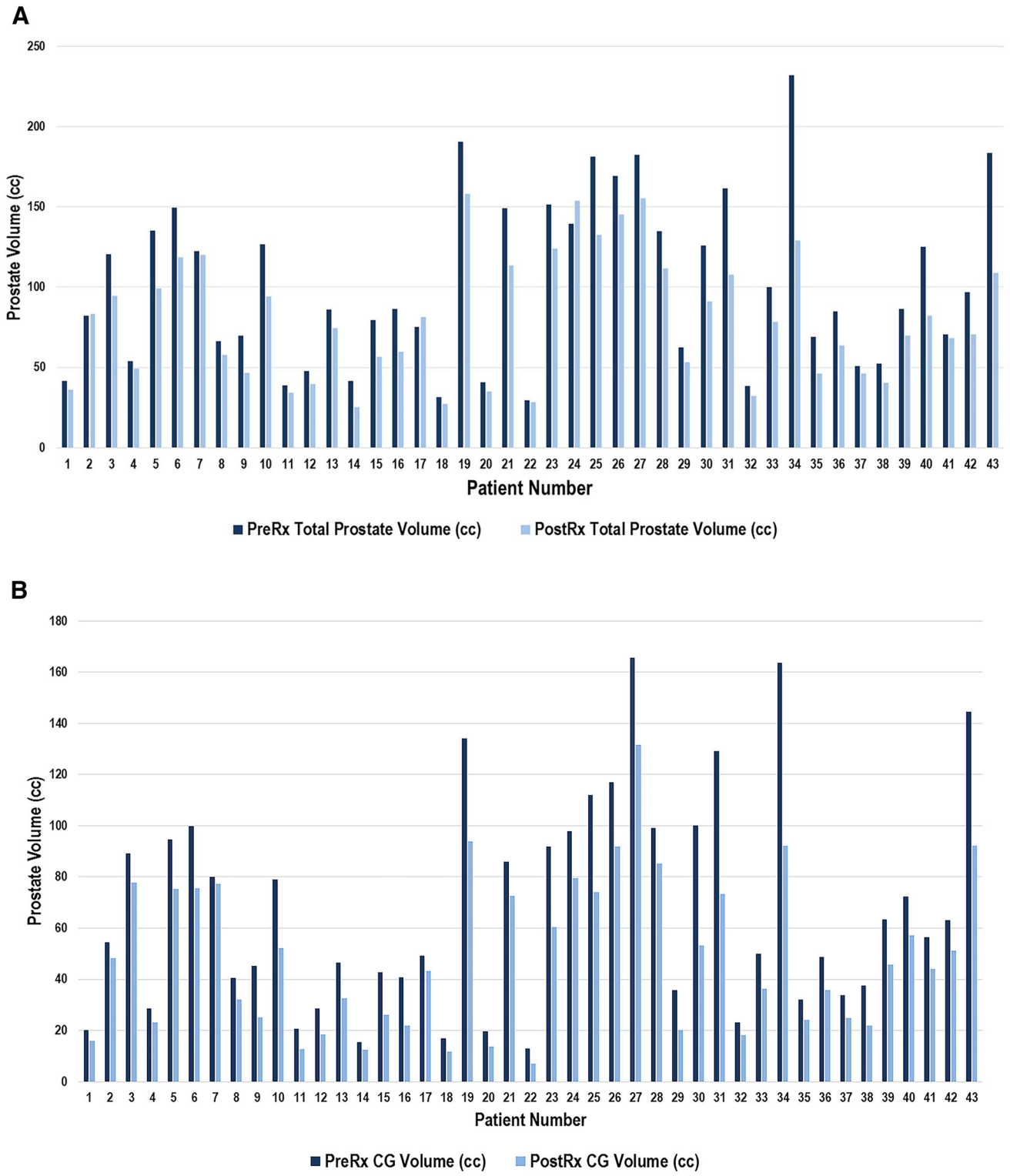

Fig. 3. **A** Pre-treatment and post-treatment total volume of prostate. **B** Pre-treatment and post-treatment volume of central gland of prostate.

decrease in T2-signal intensity relative to the surrounding prostate generally more similar to muscle signal intensity, while 21% (9/43) showed no change in signal intensity (Figs. 4, 5). Fifty-one percent (22/43) showed subjective decrease in enhancement at follow-up post-treatment compared to the pre-treatment exam while 49% (21/43) had no change in enhancement. Of 22 patients with decreased enhancement on follow-up, 73% (16/22) patients

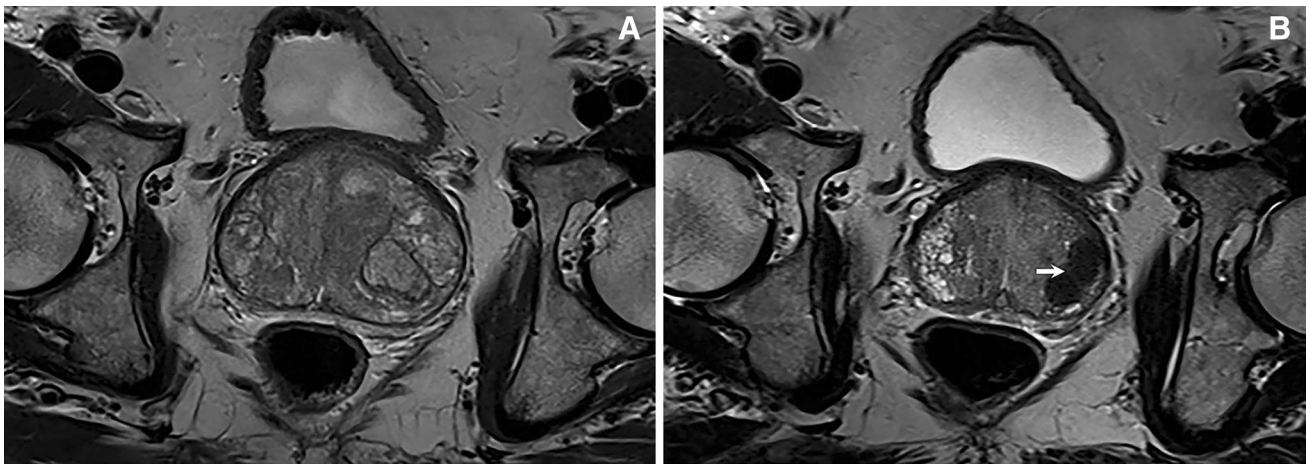

Fig. 4. 70-year-old male with LUTS/BPH. **A** Pre-treatment axial T2-weighted MR image shows enlarged prostate with BPH. **B** Post-treatment axial T2-weighted MR image shows reduction in total volume of the prostate and central gland and infarct (arrow) seen as low signal intensity involving the left transition zone.

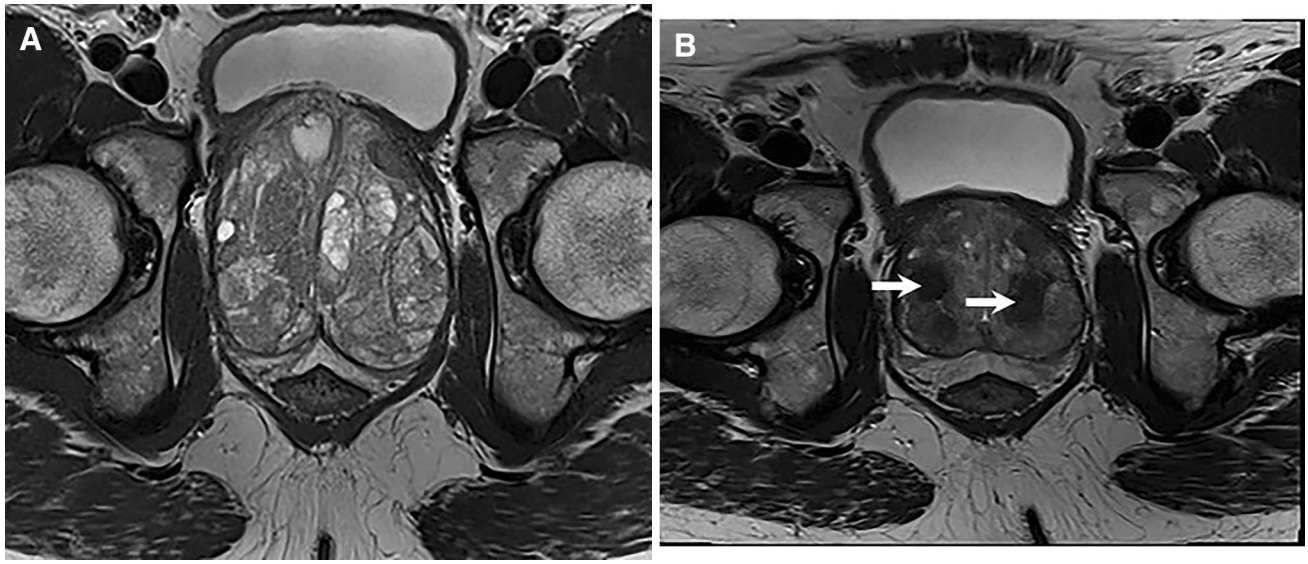

Fig. 5. 67-year-old male with bladder outlet symptoms. **A** Pre-treatment axial T2-weighted MR image shows enlarged prostate with BPH. **B** Post-treatment axial T2-weighted MR image shows reduction in total volume and central gland volume, decrease in signal intensity and infarcts (arrows) of the treated prostate.

had > 80 cc prostate volume at baseline. Thirty-three percent (14/43) exhibited small areas of infarction following treatment while 67% (29/43) had no infarction (Figs. 4, 5). Median TV reduction of 10 cc was observed in patients who did not developed infarction in opposite to median TV reduction of 33.6 cc who developed infarction. All patients that demonstrated an IPP at baseline exhibited a decrease at follow-up. Median IPP at baseline was 28 mm (range 15–47) while post-PAE median IPP was 21 mm (range 9–42) (Fig. 6).

Discussion

PAE is typically performed by an interventional radiologist under local anesthesia via transfemoral approach where the prostatic artery is catheterized to deliver embolization particles until stasis, leading to ischemic necrosis and subsequent prostate volume reduction [20]. In observational studies, PAE was associated with improvements from baseline in International Prostate Symptom Score (IPSS), maximal urinary flow (Qmax), prostate volume, post-void residual volume (PVR), quality-of-life (QoL), and International Index of Erectile Function (IIEF) [24]. Leading benefits of PAE include minimally invasive nature, applicability for poor surgical

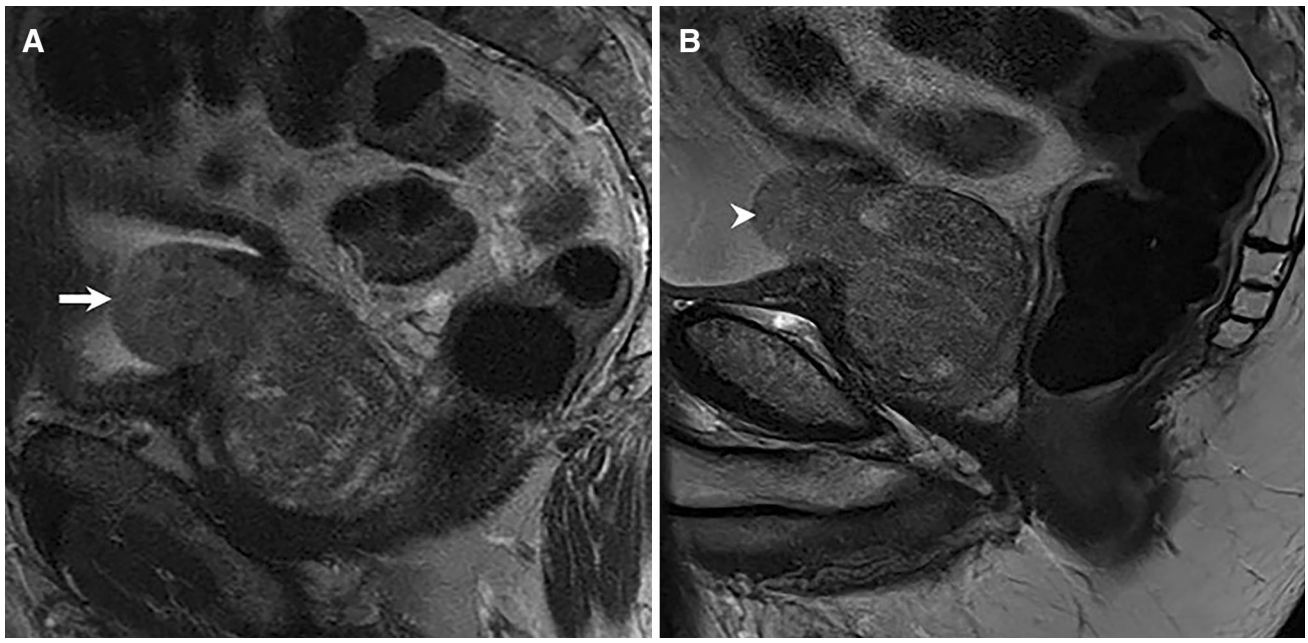

Fig. 6. 70-year-old male with BOO/BPH. **A** Pre-treatment sagittal T2-weighted MR image shows intravesical prostatic protrusion (arrow) of 31 mm. **B** Post-treatment sagittal T2-weighted MR image shows decreased intravesical prostatic protrusion (arrowhead) of 23 mm.

candidates, or patients with large volume prostates where transurethral procedures (> 80–100 cc) are suboptimal [20]. PAE has been endorsed by the Society of Interventional Radiology (SIR) for the treatment of BPH [13].

We assessed TV and CGV following PAE, which, as part of normal BPH pathophysiology, are both increased. In our cohort, forty patients (40/43) had decrease in TV and all (43/43) had CGV reduction. CGV decrease was more pronounced than TV decrease (− 27% vs. − 18%), supporting the concept that PAE can be effective for BPH from an anatomic volume reduction standpoint. Volume reduction was more noticeable with patients who had larger prostates at baseline (− 8.7 cc for ≤80 cc vs. − 30.6 cc for > 80 cc). Kisilevzky et al. has reported reduced prostate volume following PAE [25].

While BPH most commonly occurs with enlarged glands, in some cases, it may be associated with normal to moderate increase in prostatic volume. In particular, hypertrophied prostatic median lobes causing bladder neck protrusion and an IPP exceeding 5.5 mm has been associated with BOO [23, 26]. The median lobe can be challenging to treat with other minimally invasive therapies due to possible injury of the bladder neck or ureteral orifices. Moreover, some procedures (e.g., urethral lift) are contra-indicated in patients with median lobes. Patients with IPP exhibit lower reduction in International Prostate Symptom Score after TURP, compared to patients that do not have protrusion. This suggests that TURP in patients with an elevated IPP may have reduced efficacy compared to those without elevated IPP [27]. In our cohort, sixteen patients had bladder protrusion, and all demonstrated a decreased IPP with improvement in LUTS and results were published elsewhere. The potential mechanism of PAE would suggest that IPP can be treated without the mechanical reduction of this tissue that is required with other minimally invasive therapies.

Other than objective changes in volumes, several imaging features were noted following PAE. 79% of patients demonstrated decreased in T2-weighted signal intensity, and 51% showed a decrease in enhancement (48%). A signal loss on MR is generally a sign of degeneration or dilapidation [28]. In the case of BPH, PAE causes ischemia of feeding vessels resulting in shrinkage of nodules or prostate stroma by loss of water, causing signal intensity loss [29]. Decreased enhancement was more obvious among patients who developed significant prostate volume reduction. Moreover, PAE may imitate the signal alteration observed in cases with underlying chronic prostatitis and vice versa.

Among other findings, small areas of infarcts were observed in 33% of patients. These occurred exclusively in the CG, were characterized by initial hypointensity on contrast-enhanced T1 and T2-weighted images, and have been reported to become smaller and isointense to the remaining CG over time [30]. We are unable to corroborate this finding since we have only single time-point imaging follow-up. Altough 100% cohort showed reduction in CGV, but it was more pronounced in patients with infarction on imaging. Previously, Frenk et al. published MR changes following PAE, where they re-

ported infarction in 70.6% ($n = 12/17$) of patients. This finding is not consistent with our 33% observation, likely due to our single 6-month follow-up rather than 1 or 3 months, and infarcts progressively decrease in size or disappear with time. One explanation for this inconsistency may be the higher number of patients with larger baseline prostate volume (> 80 cc) in our cohort (58% vs 18%) compared with the Frenk study [31]. Unlike in fibroid embolization, infarction on MR is not common following PAE. The mechanism of action differs from uterine artery embolization (UAE) in which the successful MR imaging findings of UAE include lack of fibroid enhancement from infarction. As a result, other objective features such as prostate total volume and central gland volume become more relevant.

The strengths of our study include: specific correlation and attention of volumetric imaging parameters including the central gland, prospective design, homogenous interventional technique, and complete follow-up of the cohort. There are no published prospective studies reporting on the MR imaging findings of PAE for BPH. This study is the first to report on the reduction of the CGV and IPP following PAE [22]. Limitations include the single-center nature, lack of a control arm, use of single type of particle, low power, and lack of imaging correlation to clinical outcome.

Conclusion

PAE results in a significant reduction in total and central gland volumes and a decrease in the IPP. Other findings include infarction, decrease in T2 signal intensity, and decreased enhancement.

Author contributions Study concept and design: Rehan Ali, Ahmed Gabr, Samdeep K. Mouli, Joseph Ralph Kallini, Ahsun Riaz, Ronald Mora, Elias Hohlastos, Robert J. Lewandowski, Matthias D Hofer, Nabeel Hamoui, John Hairston, David D. Casalino, Frank H. Miller, Riad Salem. Acquisition of data: Rehan Ali, Ahmed Gabr, Samdeep K. Mouli, Joseph Ralph Kallini, Ahsun Riaz, Robert J. Lewandowski, Nabeel Hamoui, David D. Casalino, Frank H. Miller, Riad Salem. Analysis and interpretation of data: Rehan Ali, Ahmed Gabr, Samdeep K. Mouli, Joseph Ralph Kallini, Ahsun Riaz, Ronald Mora, Elias Hohlastos, Nabeel Hamoui, John Hairston, Frank H. Miller, Riad Salem. Drafting of the manuscript: Rehan Ali, Ahmed Gabr, Samdeep K. Mouli, Ahsun Riaz, Robert J. Lewandowski, Nabeel Hamoui, Matthias D Hofer, John Hairston, David D. Casalino, Frank H. Miller, Riad Salem. Critical revision of the manuscript for important intellectual content: All Authors. Statistical analysis: Rehan Ali, Ahmed Gabr, Samdeep K. Mouli, Joseph Ralph Kallini, Ahsun Riaz, Ronald Mora, Robert J. Lewandowski, Nabeel Hamoui, Frank H. Miller, Riad Salem. Administrative, technical, or material support: All Authors. Study supervision: Rehan Ali, Ahmed Gabr, Samdeep K. Mouli, Ahsun Riaz, Ronald Mora, Robert J. Lewandowski, Matthias D Hofer, Nabeel Hamoui, Frank H. Miller, Riad Salem.

Compliance with ethical standards

Funding This research was funded by Northwestern Medicine.

Conflict of interest RS serves on a DSMB for Merit Medical. None of the other co-authors report a conflict of interest.

References

1. Medina JJ, Parra RO, Moore RG (1999) Benign prostatic hyperplasia (the aging prostate). Med Clin N Am 83(5):1213–1229
2. Barry MJ, Fowler FJ Jr, O'Leary MP, et al. (2017) The American urological association symptom index for benign prostatic hyperplasia. J Urol 197(2s):S189–S197. https://doi.org/10.1016/j.juro.2016.10.071
3. Kaplan SA (2007) Identification of the patient with enlarged prostate: diagnosis and guidelines for management. Osteopath Med Primary Care 1:11. https://doi.org/10.1186/1750-4732-1-11
4. Alemu MH (2009) Transurethral resection of the prostate (TURP)–in the treatment of benign prostatic hypertrophy (BPH) in Mekelle, Ethiopia. Ethiop Med J 47(1):65–69
5. Zlotta AR, Giannakopoulos X, Maehlum O, Ostrem T, Schulman CC (2003) Long-term evaluation of transurethral needle ablation of the prostate (TUNA) for treatment of symptomatic benign prostatic hyperplasia: clinical outcome up to five years from three centers. Eur Urol 44(1):89–93
6. Lee KT, Tan HH, Li MK, et al. (1995) Transurethral microwave thermotherapy (TUMT) for benign prostatic hyperplasia (BPH)–our first 100 cases. Singap Med J 36(2):181–184
7. Issa MM (2005) The evolution of laser therapy in the treatment of benign prostatic hyperplasia. Rev Urol 7(Suppl 9):S15–22
8. Mearini E, Marzi M, Mearini L, Zucchi A, Porena M (1998) Open prostatectomy in benign prostatic hyperplasia: 10-year experience in Italy. Eur Urol 34(6):480–485
9. Shrivastava A, Gupta VB (2012) Various treatment options for benign prostatic hyperplasia: a current update. J Mid-Life Health 3(1):10–19. https://doi.org/10.4103/0976-7800.98811
10. Pisco J, Bilhim T, Pinheiro LC, et al. (2016) Prostate embolization as an alternative to open surgery in patients with large prostate and moderate to severe lower urinary tract symptoms. J Vasc Interv Radiol 27(5):700–708. https://doi.org/10.1016/j.jvir.2016.01.138
11. DeMeritt JS, Elmasri FF, Esposito MP, Rosenberg GS (2000) Relief of benign prostatic hyperplasia-related bladder outlet obstruction after transarterial polyvinyl alcohol prostate embolization. J Vasc Interv Radiol 11(6):767–770
12. Faintuch S, Mostafa EM, Carnevale FC, et al. (2008) Abstract No. 14: prostatic artery embolization as a primary treatment for benign prostatic hyperplasia in a canine model. J Vasc Interv Radiol 19(2):S7. https://doi.org/10.1016/j.jvir.2007.12.016
13. McWilliams JP, Kuo MD, Rose SC, et al. (2014) Society of Interventional Radiology position statement: prostate artery embolization for treatment of benign disease of the prostate. J Vasc Interv Radiol 25(9):1349–1351. https://doi.org/10.1016/j.jvir.2014.05.005
14. Sun F, Sanchez FM, Crisostomo V, et al. (2008) Benign prostatic hyperplasia: transcatheter arterial embolization as potential treatment–preliminary study in pigs. Radiology 246(3):783–789. https://doi.org/10.1148/radiol.2463070647
15. Carnevale FC, Antunes AA, da Motta Leal Filho JM, et al. (2010) Prostatic artery embolization as a primary treatment for benign prostatic hyperplasia: preliminary results in two patients. Cardiovasc Interv Radiol 33(2):355–361
16. Carnevale FC, da Motta-Leal-Filho JM, Antunes AA, et al. (2013) Quality of life and clinical symptom improvement support prostatic artery embolization for patients with acute urinary retention caused by benign prostatic hyperplasia. J Vasc Interv Radiol 24(4):535–542. https://doi.org/10.1016/j.jvir.2012.12.019
17. Pisco JM, Bilhim T, Pinheiro LC, et al. (2016) Medium- and long-term outcome of prostate artery embolization for patients with benign prostatic hyperplasia: results in 630 patients. J Vasc Interv Radiol 27(8):1115–1122. https://doi.org/10.1016/j.jvir.2016.04.001
18. Somani BK, Hacking N, Bryant T, et al. (2014) Prostate artery embolization (PAE) for benign prostatic hyperplasia (BPH). BJU Int 114(5):639–640. https://doi.org/10.1111/bju.12672
19. Uflacker A, Haskal ZJ, Bilhim T, et al. (2016) Meta-analysis of prostatic artery embolization for benign prostatic hyperplasia. J Vasc Interv Radiol 27(11):1686–1697. https://doi.org/10.1016/j.jvir.2016.08.004
20. Noor A, Fischman AM (2016) Prostate artery embolization as a new treatment for benign prostate hyperplasia: contemporary status in 2016. Curr Urol Rep 17(7):51. https://doi.org/10.1007/s11934-016-0608-0

21. Sun F, Crisostomo V, Baez-Diaz C, Sanchez FM (2016) Prostatic artery embolization (PAE) for symptomatic benign prostatic hyperplasia (BPH): Part 2, insights into the technical rationale. Cardiovasc Interv Radiol 39(2):161–169. https://doi.org/10.1007/s00270-015-1238-5
22. Salem R, Hairston J, Hohlastos E, et al. (2018) Prostate artery embolization for lower urinary tract symptoms secondary to benign prostatic hyperplasia: results from a prospective FDA-approved investigational device exemption study. Urology . https://doi.org/10.1016/j.urology.2018.07.012
23. Shin SH, Kim JW, Kim JW, Oh MM, du Moon G (2013) Defining the degree of intravesical prostatic protrusion in association with bladder outlet obstruction. Korean J Urol 54(6):369–372. https://doi.org/10.4111/kju.2013.54.6.369
24. Shim SR, Kanhai KJ, Ko YM, Kim JH (2017) Efficacy and safety of prostatic arterial embolization: systematic review with meta-analysis and meta-regression. J Urol 197(2):465–479. https://doi.org/10.1016/j.juro.2016.08.100
25. Kisilevzky N, Faintuch S (2016) MRI assessment of prostatic ischaemia: best predictor of clinical success after prostatic artery embolisation for benign prostatic hyperplasia. Clin Radiol 71(9):876–882. https://doi.org/10.1016/j.crad.2016.05.003
26. Lee LS, Sim HG, Lim KB, Wang D, Foo KT (2010) Intravesical prostatic protrusion predicts clinical progression of benign prostatic enlargement in patients receiving medical treatment. Int J Urol 17(1):69–74. https://doi.org/10.1111/j.1442-2042.2009.02409.x
27. Lee HS, Kim SJ, Song JM, Kim KJ, Chung HC (2010) Comparative analysis of outcomes after transurethral resection of the prostate according to prostate shape shown by transrectal ultrasonography. Korean J Urol 51(7):483–487. https://doi.org/10.4111/kju.2010.51.7.483
28. Jara H, Yu BC, Caruthers SD, Melhem ER, Yucel EK (1999) Voxel sensitivity function description of flow-induced signal loss in MR imaging: implications for black-blood MR angiography with turbo spin-echo sequences. Magn Reson Med 41(3):575–590
29. Noworolski SM, Henry RG, Vigneron DB, Kurhanewicz J (2005) Dynamic contrast-enhanced MRI in normal and abnormal prostate tissues as defined by biopsy, MRI, and 3D MRSI. Magn Reson Med 53(2):249–255. https://doi.org/10.1002/mrm.20374
30. van Walderveen MA, Kamphorst W, Scheltens P, et al. (1998) Histopathologic correlate of hypointense lesions on T1-weighted spin-echo MRI in multiple sclerosis. Neurology 50(5):1282–1288
31. Frenk NE, Baroni RH, Carnevale FC, et al. (2014) MRI findings after prostatic artery embolization for treatment of benign hyperplasia. AJR 203(4):813–821. https://doi.org/10.2214/ajr.13.11692